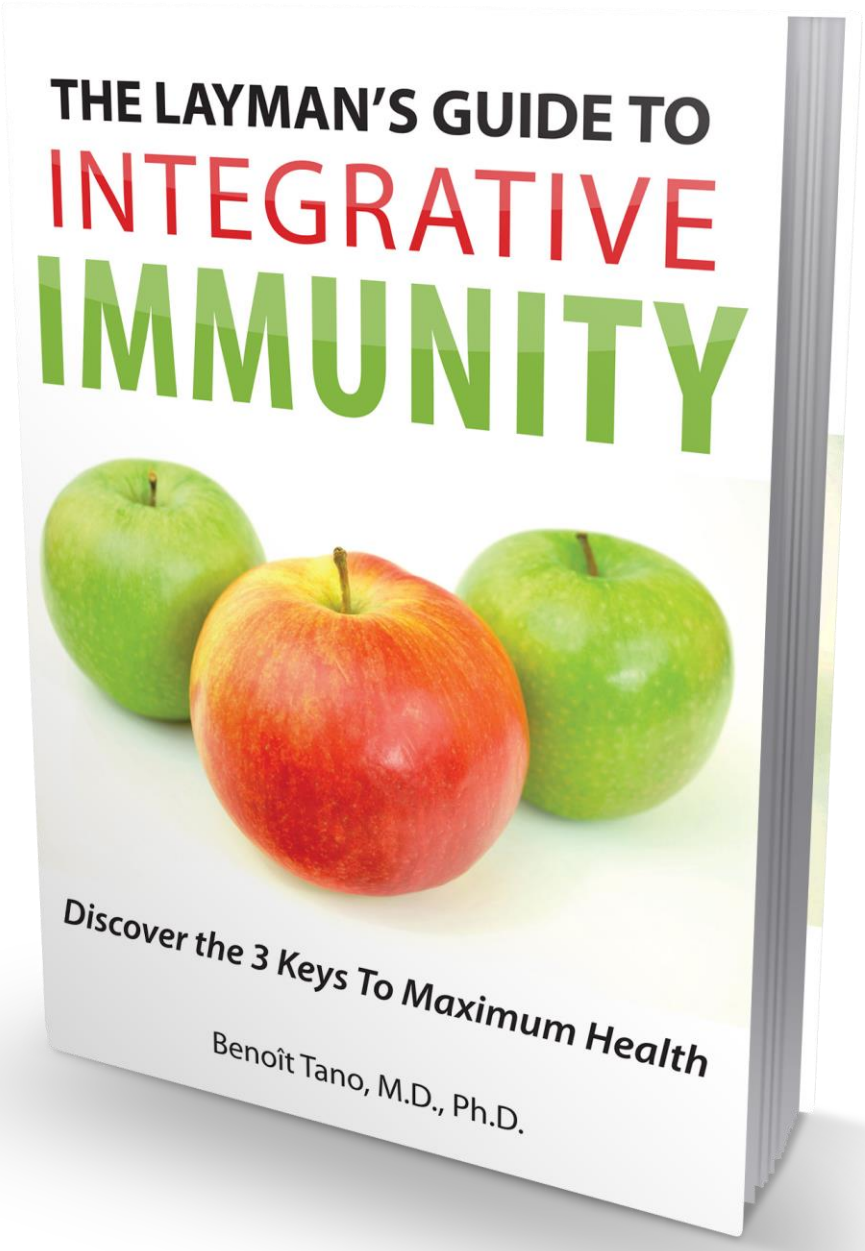
THE LAYMAN'S GUIDE TO INTEGRATIVE IMMUNITY

Discover the 3 Keys To Maximum Health

Benoît Tano, M.D., Ph.D.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INTEGRATIVE MEDICAL PRESS (IMP)

Layman's Guide to Integrative Immunity: Discover the 3 Keys to Maximum Health

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In Memory of my 5th and 6th grade teacher,

MR. BERNARD NIANHOULOU

a great visionary who taught me learning secrets that I still use today.

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INTRODUCTION

DEFINITION OF INTEGRATIVE IMMUNITY

Integrative Immunity is the study of the interrelationships and communication among the body systems that nurture, sustain, defend, and keep the body alive. When the optimal communication breaks down, diseases emerge.

Two major systems—the endocrine and immune systems—drive the majority of all nontraumatic pathologies. The effect of environmental pesticides and common household chemicals on these two systems is the focus of this book. In particular, I will discuss the role of pesticides in the explosion of obesity and allergies on our planet, and offer strategies in Integrative Immunity to address these problems.

THE ROLE OF PESTICIDES IN THE OBESITY AND ALLERGY EPIDEMICS

In 1985, the Centers for Disease Control and Prevention (CDC) decided to begin tracking obesity in the U.S. Since that time, obesity has become epidemic in America and has increased dramatically worldwide. In the 1990s, it was also discovered that allergies (environmental and food allergies) were increasing. These are currently at epidemic proportions.

What has changed in our environment that is causing these health problems? Obesity is the number one killer in the U. S. and, increasingly, the world. It kills in silence and indirectly, standing as the root cause of a great many of the diseases treated by healthcare providers. These diseases include hyperlipidemia, heart disease, diabetes, hypertension, and malignant tumors, to name only a few.

If any other epidemic had caused as many deaths as obesity and its comorbidities, the whole world would be diligently searching for a cure; yet, most healthcare providers still take a surprisingly casual attitude toward obesity. In most cases, it is treated as a more or less benign condition easily controlled if patients would only put forth some effort. This is to say that obesity is often treated as if it were the patient's fault, with healthcare providers frequently assuming that a patient's obesity is the result of poor dietary choices and a sedentary lifestyle. The root cause of the obesity epidemic is not explored, and serious weight-loss treatment plans are not offered.

Allergies caused by environmental pollutants and toxins, as well as by various foods, is another area that is largely unexplored. Healthcare practitioners treat symptoms, but rarely look for causes in patients' environments and in the food they eat.

PESTICIDES AS ENDOCRINE DISRUPTORS

Many healthcare practitioners are at a loss when faced with environmental allergies (asthma, rhinitis, sinusitis, atopic dermatitis, urticaria, angioedema, etc.), food allergies, and autoimmune diseases. Is the cause of the growing obesity epidemic also the cause of the growing allergy epidemic? Are pesticides, herbicides, and other chemicals—especially endocrine-disrupting chemicals used as pesticides and common household chemicals including cosmetics and cosmeceuticals—to blame?

On the spectrum of diseases, patients are showing up in clinics around the world with more obesity, diabetes, hypertension, hyperlipidemia, anxiety, depression, inflammatory thyroid disease (Hashimoto's thyroiditis), inflammatory bowel diseases, foggy thinking, and decreased libido. Women are experiencing hormone-related symptoms and conditions, such as precocious puberty (in young girls), painful menstrual cramps, heavy menstrual bleeding, irregular menses, ovarian cysts, endometriosis, fibroid tumors, fibrocystic breast disease, and cervical dysplasia. Breast cancer, uterine cancer, ovarian cancer, cervical cancer, vaginal cancer, and colon cancer are also increasing in prevalence. Men are experiencing enlarged prostates, decreased libido due to low testosterone, and prostate and colon cancer. Many other cancers, such as liver, bladder, and kidney cancers, and lymphomas and leukemia are all increasing. Even Third World countries are seeing an increase in these cancers and pathologies.

This book will dig up the bones and set readers on the course of optimal lifestyle choices that will bring them maximum health—the only wealth of nations—and happiness. It is often said that we are products of our environment, and we are, indeed!

THE WIDESPREAD PREVALENCE OF ENDOCRINE DISRUPTORS

Endocrine-disrupting compounds comprise a number of chemical categories including drugs, pesticides, substances used in the plastics industry and in consumer products, industrial by-products, pollutants, and agrochemicals. Some of these chemicals are pervasive and widely dispersed in the environment and may bioaccumulate. Some are termed persistent organic pollutants (POPs). These toxins can be carried great distances and have been found virtually everywhere in the world.

Food is a major vehicle by which people are exposed to endocrine disruptors. Diet is believed to account for up to 90% of an individual's burden of organochlorine insecticides such as polychlorinated biphenyls (PCBs), which were commonly used in the manufacturing industry until the late 1970s; and dichlorodiphenyltrichloroethane (DDT), a pesticide widely used in the U.S. from the 1940s until it was banned in 1972. These compounds are fat soluble and, therefore, are likely absorbed from the environment and stored in the fatty tissue of animals that we eat as well as in our own fat cells.

With the increase in household products containing pollutants and the decrease in the quality of building ventilation, indoor air has also become a significant source of endocrine-disrupting chemical exposure.

ENDOCRINE DISRUPTORS AND HUMAN DISEASES

In this book, we will explore the interface between the endocrine system and the immune system. Environmental toxins, their effects as endocrine disruptors (especially as the source of the estrogen epidemic), and their geographical distributions will be highlighted and correlated with the obesity epidemic and atopic diseases (diseases extrinsically caused) and so-called autoimmune diseases. In particular, we will look closely at the following:

- The connection among pesticides, common household chemicals, and what I call the Hormone Imbalance Syndrome (HIS): high estrogen, low thyroid function, and low male hormones, such as testosterone, and the impact of these factors on well-being.
- The impact of Hormone Imbalance Syndrome on obesity and its comorbidities, as well as on allergies.

- The reason southern and midwestern states in the U.S. have more obesity, more morbidity, and higher mortality than other regions.
- Pesticide-usage maps, obesity maps, and mortality maps of the U.S. will be correlated.
- The connection between pesticides and the growing allergy epidemic.
- The unified relationship between the endocrine system and the immune system.

HOW ENDOCRINE DISRUPTORS AFFECT HUMAN HORMONES

Endocrine disruptors comprise chemicals that, at certain doses, can interfere with the endocrine (hormone) system in mammals. Specifically, endocrine disruptors may be associated with learning disabilities and cognitive (brain) disorders, birth defects, various kinds of cancer (as noted earlier), and sexual development problems such as feminization in males and masculinization in females. Although fetuses are at the greatest risk of harm from the effects of endocrine disruptors, these chemicals affect all of us.

One of the first people to speak about endocrine disruptors was Theo Colborn (1927-2014). In 1993, she presented a paper in which she stated that environmental chemicals disrupt the development of the endocrine system, and the effects of exposure during fetal development are often permanent.

STEPS TO EFFECTIVELY PREVENT AND TREAT OBESITY AND ALLERGIES

In this book, we will discuss preventive and therapeutic strategies for obesity and allergies. Among the most useful protocols in preventing these conditions is the Estrogen-Free Lifestyle (EFL). This change will counteract the effects of Hormone Imbalance Syndrome. Other Integrative Immunity strategies will also be presented.

PART I: KNOW YOUR BODY

THE PROBLEM WITH DIVIDING HEALTHCARE TREATMENT INTO SPECIALTIES

Medicine divides the functions of the human body into the following twelve categories or systems:

1. Reproductive System
2. Endocrine System
3. Digestive System
4. Immune System
5. Cardiovascular System
6. Respiratory System
7. Lymphatic System
8. Nervous System
9. Muscular System
10. Skeletal System
11. Integumentary System
12. Urinary System

One more, which I believe is as important and influential as the other twelve, may be added:

13. The "Psychic" or Mind-Body-Soul System

Each body system is equally important. They are interrelated and work together to sustain health and life. Each system is complex and difficult to study; therefore, modern medicine has divided the body systems into specialties and subspecialties.

Although such specialization is necessary because no one physician or healthcare practitioner can know all the body systems in detail, this division of medical care has resulted in a failure to track the interrelationships among the various body systems.

What we see today are multispecialty referrals, different physicians prescribing different drugs that interact with each other to cause more symptoms, increased medical costs, and poor health outcomes. Integrative Immunity tries to address many of these healthcare problems by studying symptoms and diseases holistically.

REVIEW OF SYSTEMS

The secret to a successful visit with your practitioner starts with knowing your body well enough to detect changes. You can greatly enhance your own well-being by paying attention to subtle changes in your body. Do this by reviewing your body systems regularly. Especially before a visit with your practitioner, notice and write down changes in your body. Take the time to get in touch with what's going on with your body, from head to toe. This will help your practitioner to determine the causes of your problems and prescribe appropriate treatments.

Create a routine in which you ask yourself daily or weekly if you have any of the following symptoms:

Head: hair loss, headaches (migraine or sinus-pressure headaches)?

Eyes: blurry vision, double vision, itchy eyes, watery eyes, redness of the eyes, light sensitivity?

Ears: ear discharge, ear pain, fullness in the ears, hearing loss, ringing in the ears?

Nose: runny nose, stuffy nose, sneezing, itchy nose, nose bleeds?

Mouth: lip swelling, tongue swelling, mouth sores, dry mouth, bad breath (halitosis)?

Throat: sore throat, trouble swallowing, postnasal drainage, itchy throat, voice change?

Neck: neck pain, neck stiffness, lumps in the neck?

Chest: sleep apnea, chest tightness, choking, coughing, shortness of breath, wheezing, chest pain, heart palpitations?

Abdomen: abdominal distention, bloating, pain or cramps in the abdomen, bloody stool, constipation, diarrhea, nausea, vomiting?

Urinary: painful urination, blood in the urine, increased frequency of urination, genital sores? For men, add: penile discharges, scrotal swelling, and testicular pain? For women, add: frequent urinary tract infections (UTIs), any menstrual disturbances, painful menstrual cramps, irregular menses, heavy bleeding, ovarian cysts, fibroid tumors, fibrocystic breast disease, endometriosis, cervical dysplasia found by abnormal Pap smear?

Musculoskeletal: joint pain, back pain, gait problems, joint swelling, muscle aches (fibromyalgia)?

Skin: skin color change, pallor, rash, sores, wounds?

Neurological: lightheadedness, dizziness, numbness, seizures, speech difficulty, passing out (syncope), tremors, weakness?

Hematological: lumps in the neck, bruise easily, bleed easily?

Psychiatric: agitation, behavioral problems, confusion, decreased concentration, depression, anxiety, nervousness, panic attacks, mood swings, sleep disturbance?

General: activity change, appetite change, fevers or chills, excessive sweating, night sweating, hot flashes, fatigue ("tired all of the time" syndrome), abnormal weight gain?

As you notice, I have gone from head to toe and covered most of the body systems. If you have any of these symptoms, mention them to your practitioner as soon as possible. There could be many causes, so that is the reason it is best to consult with an expert, and not use the internet or the experiences of family and friends to determine the cause.

One question that you and your practitioner might ask is, is there a common denominator among the symptoms you are experiencing? There are two major factors that cause a multitude of diseases today—extrinsic and intrinsic. Extrinsic causes are environmental factors such as pollution, infections, and accidents. Intrinsic factors include genetic tendencies or disorders and the effects of the environment on our genes, which is known as epigenetics. There is not a lot we can do presently to change the genes we were born with (intrinsic factors), but there is much we can do to improve our environment and reduce our exposure to toxins (extrinsic factors) that cause disease.

PART II: KNOW YOUR ENVIRONMENT

INTRODUCTION: THE RISE OF HOMO ECONOMICUS AND THE DEMISE OF WORLD HEALTH

It may seem like a digression, but I refer now to the work of Adam Smith (1723-1790), a Scottish economist who published the book, *Wealth of Nations*, in 1776. In his book, he voiced his thoughts: You do your things (your self-interest) and I will do mine (my self-interest). These things are guided by an invisible hand that brings them together to make a wonderful world for all. This is the creed of *Homo economicus*, the economic man. The economic man is in business for his own self-interest and does not necessarily care about what happens to the environment or to others. Many individuals are focused on amassing wealth, regardless of the environmental consequences. The invisible hand has led to worldwide income inequality. Fulfilling our own self-interests has created a powerful few who control all of the world resources.

Another thinker, Reverend Thomas Robert Malthus (1766-1834), was an English cleric and scholar who influenced political economy and demography. He is perhaps most well-known for his book, *An Essay on the Principle of Population,* which he published under the pseudonym Joseph Johnson. According to Malthus' book, the world population grows at a geometric rate, but the substances to feed that population grow at an arithmetic rate. The discrepancy between these two growth patterns has led to a major gap between the supply of and demand for food. Where there is demand, someone will find a way to make money from it. *Homo economicus* is always ready to exploit such situations for his self-interest.

The behavior of *Homo economicus* has led to pollution of the earth and the rise of chronic diseases. *Homo economicus* has, therefore, led to the demise of world health. This process can be summed up as the pursuit of self-interest (greed) at the cost of everything else.

Historically, population pressures led to an increased demand for food, which, in turn, stimulated mass production of foods through the synthesis and use of agrochemicals (pesticides) and industrial chemicals. Agrochemicals have caused a boomerang effect—producing food to feed the world (with the assumption that preventing starvation makes the population healthier), which has resulted in environmental pollution and the demise of world health.

OUR LOVE AFFAIR WITH PESTICIDES: HISTORY OF PESTICIDE USE IN THE U.S.

Organochlorine Insecticides

Although organochlorine pesticides such as dichlorodiphenyltrichloroethane (DDT) and its metabolites were banned in the 1970s, they are still persistent in the environment. The activity of organochlorines takes place through the sodium channels of the body and affects various cells. For example, when these chemicals bind the sodium channels on mast cells and basophils, they cause degranulation of these cells, and mediators such as histamine and leukotrienes are released. These substances are responsible for allergic reactions. When organochlorines attach to the sodium channels on the pancreatic islet cells, they cause a cascade that leads to insulin release. Insulin causes a proliferation of mast cells and basophils that leads to further allergic reactions.

Organochlorines are also estrogenic, and when they attach to the estrogen receptor-alpha on mast cells and basophils, they cause histamine and leukotriene release via another pathway.

Organophosphate Insecticides

Organophosphate and carbamate insecticides largely replaced the organochlorine insecticides that were banned in the 1970s. Organophosphate and carbamate insecticides are acetylcholine-esterase inhibitors. Acute exposure to these insecticides can lead to acetylcholine poisoning.

The classic constellation of symptoms of cholinergic excess caused by acetylcholine poisoning includes copious respiratory and oral secretions, diarrhea and vomiting, sweating, altered mental status, autonomic instability, and generalized weakness that can progress to paralysis and respiratory arrest. These can be summarized in the acronym, MUDDLES: Miosis (constriction of the pupil), Urination, Diarrhea, Diaphoresis (sweating), Lacrimation (watery eyes), Excitation of the central nervous system (anxiety), and Salivation.

Frequent acetylcholine exposure and acetylcholinemia (too much acetylcholine in the blood) may cause symptoms in children such as the agitation and behavioral problems seen in attention-deficit hyperactivity disorder (ADHD), attention-deficit disorder (ADD), and even autism.

Long-term exposure to these insecticides leads to conditions such as chronic nasal and food allergies, obesity, diabetes, and hypertension.

Mast cells and basophils have receptors for acetylcholine, and when this chemical attaches to these receptors, the effect is the same as that seen with organochlorines: mast cell and basophil degranulation and the release of mediators such as histamine and leukotrienes. These chemicals, in turn, cause allergic reactions such as rhinitis, conjunctivitis, asthma, atopic dermatitis, urticaria, and angioedema.

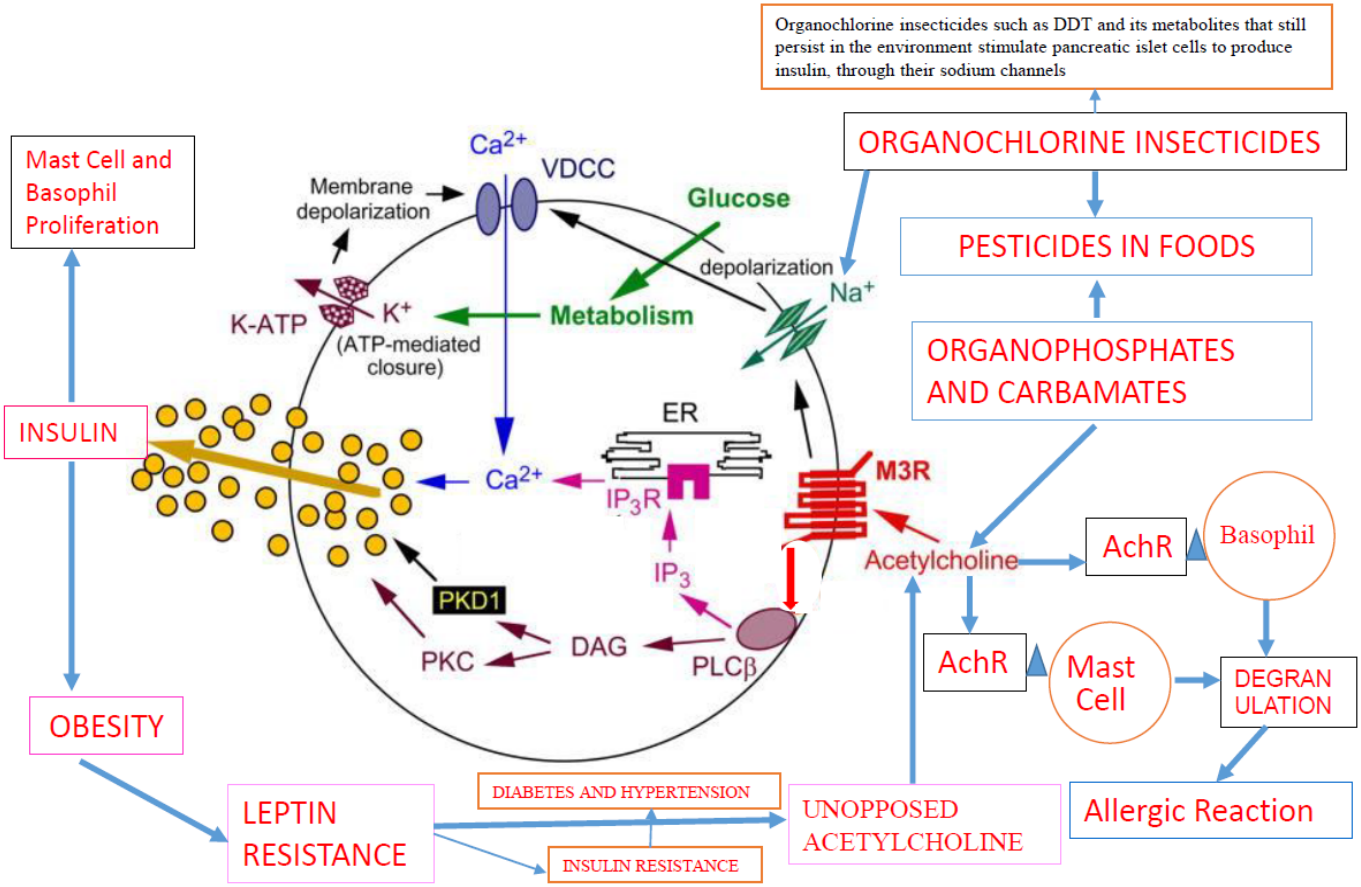
Acetylcholine also binds to the muscarinic receptor 3 on pancreatic islet cells and causes a cascade that leads to insulin production. Insulin causes mast cell and basophil proliferation that leads to chronic allergic reactions.

Organochlorines, organophosphates, and carbamates induce insulin release. The presence of too much insulin in the body leads to a craving for sweets, leptin resistance, and insulin resistance, all of which are associated with a tendency toward obesity. Leptin resistance leads to unopposed acetylcholine action on pancreatic islet cells, which perpetuates chronic insulin resistance, hyperglycemia, diabetes, and hypertension. When the leptin level is normal, it dampens the effect of acetylcholine-induced insulin release from the pancreatic islet cells.

People with allergies (environmental and food allergies), obesity and weight gain, diabetes, and hypertension should avoid foods that are known to have a high content of these classes of pesticides.

The pathophysiology diagram below shows the different pathways through which acetylcholine increase induced by endogenous production and organophosphate-induced production leads to allergic reactions, insulin resistance, obesity, and comorbidities.

Figure 1. The Effects of Organochlorine and Organophosphate Pesticides on Allergic Reactions, Insulin Resistance, and Comorbidities.



Organophosphate insecticides and carbamates are Acetylcholine esterase inhibitors and, therefore, tend to increase Acetylcholine level in the blood. Acetylcholine attaches to the muscarinic receptor 3 (M3R) on the pancreatic islet cells and induce a cascade that leads to insulin production. Insulin causes Mast cell and Basophil proliferation, but causes obesity by disposing excess glucose into adipocytes (fat cells). Adipocytes produce leptin that eventually leads to leptin overload and leptin resistance. Leptin normally modulates the activities of Acetylcholine on the pancreatic islet cell and tend to reduce insulin production; however, leptin resistance leads to unopposed Acetylcholine activity that leads to more insulin production and insulin resistance. Too much insulin, in turn, leads to diabetes and allergic reactions and that is the reason obese women tend to have asthma and adult-onset allergies more often than the non-obese women.

Glyphosate

Glyphosate (brand name, Roundup) was introduced in the 1970s, but became widely used in the 1990s with the advent of GMOs (Genetically Modified Organisms). Glyphosate was the most commonly used active ingredient in agricultural pesticides in the U.S. in 2007 (most recent data available)—with 180 million to 185 million pounds used—and has been since 2001.

Glyphosate was used in cotton production before the advent of GMO species of cotton and continues to be used heavily in cotton and peanut production.

Other Endocrine-Disrupting Pesticides

Other commonly used endocrine-disrupting pesticides include Triazine compounds (such as atrazine), and chloracetamides.

PESTICIDES AS ENDOCRINE DISRUPTORS

Most of the chemicals used in agriculture worldwide are endocrine-disrupting chemicals (EDCs) that are generally classified as estrogen disruptors, thyroid disruptors, or androgen disruptors, which are the three main types of endocrine-disrupting chemicals implicated in obesity and allergies. Androgens are male hormones that are produced by both men and women.

THE ESTROGEN EPIDEMIC

What Is Estrogen?

Stated simply, estrogen is a hormone that makes certain things in the human body grow—the breasts and hips of pubertal females, and certain tumors and cancers later in life.

There are two primary sources of estrogen—endogenous (originating in the body) and exogenous (originating in the environment).

Endogenous Estrogens

Endogenous estrogens comprise the following: estradiol, estrone, estriol, estetrol, and estrogen metabolites.

Estradiol, or more precisely, 17β-estradiol, also known as E2, is a steroid and estrogen sex hormone, and the primary female sex hormone. It is named for and is important in the regulation of estrous and menstrual female reproductive cycles. Estradiol is essential for the development and maintenance of female reproductive tissues, but it also has important effects in many other tissues including bone. While estrogen levels in men are lower compared to those in women, estrogens have essential functions in men as well. Estradiol is found in most vertebrates as well as many crustaceans, insects, fish, and other animal species.

Estriol, also known as E3, is one of the three main estrogens produced by the body, although it is only produced in significant amounts during pregnancy. It is generated by the placenta from an androgen steroid created in the fetal liver and adrenal glands.

Estrone is an estrogenic hormone secreted by the ovary as well as by adipose tissue. Estrone is the least abundant of the three primary forms of estrogen; estradiol is present almost always in the reproductive female body, and estriol is abundant primarily during pregnancy.

Estetrol, or 15α-hydroxyestriol, contains four hydroxyl groups, which is the reason it is also known as (E4). Estetrol is an estrogen steroid produced only during pregnancy by the liver of the developing fetus. It is found in detectable levels in maternal serum at around week 20 of fetal development. Estetrol is synthesized from estradiol (E2) and estriol (E3) by two enzymes, 15α- and 16α-hydroxylase. It was first discovered in the urine of pregnant women by Egon Diczfalusy and coworkers in 1965. At birth, the liver of the newborn loses its capacity to synthesize E4 because E2 and E3 are no longer expressed.

Since 2001, E4 has been studied extensively. It's been discovered that E4 may be suitable for use in several indications: contraception, hormone replacement therapy for menopausal symptoms, and as a treatment for breast cancer and osteoporosis.

Estetrol is being developed as the estrogenic component in an oral contraceptive pill by Estetra (Belgium). Pantarhei Bioscience B.V. (The Netherlands) is developing estetrol for hormone replacement therapy and treatment of breast cancer and osteoporosis.

Estrogen Metabolites

The role of environmental pollutants such as bisphenol A or F and other xenoestrogens in human pathologies is becoming clearer. Hyperestrogenism, hypothyroidism, and hypo or hyperandrogenism are some of the devastating effects of environmental pollutants. Estrogen involvement in cancer is well studied, and it is known that estrogens may cause six different cancers or more in women. It is reported that breast, uterine, ovarian, vaginal, cervical, and colon cancer in women and prostate and colon cancer in men may be due to the estrogens. Many studies point to estrogen metabolism to catechol estrogen metabolites as the mechanism through which genotoxicity occurs.

Estrogen metabolites are derived from estrogen metabolism by the liver. When we ingest estrogens (estrogen pills, estrogenic foods, and estrogenic chemicals), they end up in the liver. The liver uses the cytochrome P-450 enzymes to break down these estrogens into components known as estrogen metabolites. The following are major endogenous estrogen metabolites that are either protective or damaging:

2-hydroxyestrone

In general, the 2-hydroxyestrogen metabolites are considered protective. If you have heard that broccoli protects you from cancer, this is the source of that information. High levels of 2-hydroxyestrone may protect you from estrogen-related cancers, such as breast, uterine, ovarian, vaginal, cervical, colon, and prostate cancers, and estrogen-related autoimmune diseases, such as lupus and rheumatoid arthritis. The 2-hydroxyestrone increases with the following:

Intake of cruciferous vegetables
Intake Omega-3 fatty acids
Exercise

2-methoxyestrogens

2-methoxyestrone
2-mthoxyestradiol

These two metabolites are considered protective only if methylated.

2-methoxyestrone:2-hydroxyestrone ratio

This ratio serves as a gauge for methylation efficiency in the body.

2-hdroxyestrogens are protective when converted to methylated forms.
Low ratios mean inadequate methylation of estrogen.
Low ratios may be due to stress.
Stress causes catecholamines (adrenaline, norepinephrine, and dopamine) production using an enzyme called COMT (catechol-O-methyltransferase).
If the ratio is low, then nutritional support for methylation, such as trimethylglycine (Betaine), methylcobalamin (B12), SAMe, and methylfolate will be optimal.

4-hydroxyestrogens

4-hydroxyestrone
4-hydroxyestradiol

These metabolites are considered procarcinogenic, and free-radical inducing.

4-methoxyestrogens

The following metabolites are considered to be protective:

- 4-methoxyestrone
- 4-methoxyestradiol

They are considered to be protective if they are methylated.
Methylation may improve with methyl donors such as:

- TMG (Trimethylglycine)-Betaine
- Folate (Metafolin)
- methylcobalamin (B12)
- B6
- Magnesium

This is the reason I recommend nutritional supplements such as Nutrient 950 with NAC, a well-conceived multivitamin that offers the methylated forms of the B-vitamins and folate. I also recommend Betaine (TriMethylGlycine) for that purpose.

4-methoxyestrone:4-hydroxyestrone ratio

This ratio is also a gauge for methylation efficiency in the body.
4-Hydroxyestrogens are protective when they are converted to methylated forms. Low ratios mean inadequate methylation of estrogen. Low ratios may be due to stress. Stress causes catecholamines production using COMT. You should take nutritional support for methylation if the 4-methoxyestrone:4-hydroxyestrone ratio is low.

16-alpha-hydroxyestrone

Increases with obesity, pesticides exposure, and low thyroid.
High levels are non-beneficial and may be associated with the following:

- Estrogen-induced cancers (breast, uterine, ovarian, vaginal, cervical, colon, prostate).
- Autoimmune diseases (lupus, rheumatoid arthritis, multiple sclerosis).

2-hydroxyestrone:16-alpha-hydroxyestrone ratio

This ratio is useful to gauge estrogen metabolism. Low ratio may be associated with likelihood of estrogen-induced diseases (breast, uterine, ovarian, vaginal, cervical, colon cancers), and autoimmune diseases. The ratio can be modified by consumption of plenty of cruciferous vegetables (see table 23) and/or cruciferous vegetables extract: DIM (Diindolylmethane, a metabolite of I3C). I3C (indole–3–carbinol) is the primary cruciferous vegetables extract. Calcium D-glucarate can also improve the 2:16 ratio. Combinations of these three supplements exist (CDG, by Ortho Molecular, and DIM Detox by Pure Encapsulations).

Genova Diagnostics Hormonal Health Kit for women, reports the estrogen metabolites (2-hydroxyestrone, 16-alpha-hydroxyestrone, and the 2:16 ratio) in addition to basic hormones such as progesterone, estradiol, estrone, Estriol, DHEA, testosterone, and free androgen index. I have used this test to evaluate the hormone status of women. The results and follow-up results have been consistent and reproducible. If a patient has low DHEA by her initial hormonal health test, and she fails to take DHEA, the repeat test will show low DHEA. This consistency and the report of the estrogen metabolites are the factors that have led me to continue using this serum test. I have tried both the saliva test and the Genova diagnostics urine test without much success. The lack of success with the saliva test and urine test may stem from using topical BHRT instead of extended release oral BHRT.

Endogenous Estrogens

Most endogenous estrogens in women are produced during the menstrual cycle. These hormones are also produced in women and men by the adrenal glands and by conversion of testosterone to estrogens. Aromatase is the enzyme that facilitates the conversion of testosterone to estrogen.

Obesity increases the activity of aromatase; hence, obese men and women tend to have more estrogen in their bodies than lean individuals. Excess estrogen in men is responsible for the gynecomastia (the appearance of breast tissue) in men. This phenomenon is also associated with age, obesity, and alcohol abuse.

Estrogens increase production of insulin, which can lead to obesity. Obesity, in turn, leads to leptin resistance and insulin resistance. Insulin resistance is a precursor of diabetes and hypertension. [Swislocki, Arthur, M.D.,"Insulin Resistance and Hypertension," *American Journal of Medical Sciences,* August 1990, vol. 300 (2):104 - 15].

Insulin also leads to mast cell and basophil proliferation, which increase allergic reactions.

Xenoestrogens

"Xeno" means strange or foreign. Xenoestrogens are a type of hormone that imitates estrogen. These can be either synthetic or natural chemical compounds. Synthetic xenoestrogens are widely used, industrial compounds that have estrogenic effects on living organisms even though they differ chemically from the estrogenic substances produced internally by the endocrine system of these organisms. Natural

xenoestrogens include phytoestrogens, which are plant-derived xenoestrogens. Because the primary route of exposure to these compounds is through consumption of phytoestrogen plants, they are sometimes called "dietary estrogens." Mycoestrogens, which are estrogenic substances found in fungi such as mushrooms, are another type of xenoestrogens. Metalloestrogens from heavy metals (aluminum, antimony, arsenite, barium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, selenite, tin, and vanadate) have been described. Xenoestrogens are clinically significant because they can mimic the effects of endogenous estrogen and, thus, have been implicated in precocious puberty and other disorders of the reproductive system.

Many herbicides and pesticides are xenoestrogens, as are a number of household chemicals, including beauty products (lotions, deodorants, shampoos, hair conditioners, perfumes, and colognes) that contain parabens, methyl parabens, propyl parabens, phthalates—often in the form of fragrance, mineral oils, and petroleum.

Phytoestrogens: Plant-Based Estrogens

Plant-based estrogens include the following: barley, rye, wheat, spelt, kamut, millet, GMO corn, and many other estrogenic plants not listed here. The first five are commonly known as gluten-containing grains. Since they are phytoestrogens, they affect women more than men, and this is the reason more women than men have gluten sensitivity and celiac disease.

Corn is estrogenic by association: it has been genetically modified to tolerate Roundup (main ingredient, glyphosate), which is known to be estrogenic. Several other chemicals are also used in the production of corn. Atrazine is an aromatase booster and converts testosterone to estrogen. Alachlor, acetochlor, metolachlor and metolachlor-S are toxic to the thyroid, cause nasal turbinate tumors in animals, and, perhaps, cause nasal turbinate enlargement and allergies in humans.

Symptoms Associated with Estrogen Dominance

When estrogen is normal or high and progesterone is low, this is known as estrogen dominance. The list of symptoms associated with estrogen dominance follows:

Symptoms Associated with Estrogen Dominance

Acceleration of the aging process (increased wrinkles that are not consistent with chronological age)

Adrenal exhaustion

Allergy symptoms (asthma, hives, rashes, sinus congestion)

Autoimmune disorders (lupus erythematosus, thyroiditis, possibly Sjögren's disease)

Breast cancer

Breast tenderness

Cervical dysplasia

Cold hands and feet as a symptom of thyroid dysfunction

Decreased sex drive

Depression with anxiety or agitation

Dry eyes

Early onset of menstruation

Endometrial (uterine) cancer

Endometriosis

Weight gain, especially around the abdomen, hips, and thighs

Fatigue

Fertility problems

Fibrocystic breasts

Fibromyalgia

Foggy thinking

Restless legs (particularly at night time in bed)

Itchy, burning sore ears

Sensation of foreign object in ear (such as bees or insects, tinnitus)

Vertigo, particularly around ovulation time onwards (more profound when lying down in bed)

Palpitations

Heartburn

Low resistance to infection

Sinusitis, head congestion, flu-like headaches

Premenstrual asthma

Painful, throbbing face, one side more than the other often reported

Aching teeth (dental checkup inconclusive)

Aching joints present in the form of rheumatism or arthritis; joint and muscle stiffness; nerve endings feeling very fragmented, fragile, and tender to touch; imitative of fibromyalgia syndrome

Pins and needles, sciatica, hip pain down one side predominantly quite common although bone mineral densities and hip x-ray tests are normal

Painful ovaries upon ovulation

Painful ovaries in the absence of ovulation, confusing women that they have ovulated

Obsessive, irrational thought and behavior patterns: not finding a lost item: trying to think of someone's name; being aware on one level, but unable to stop yourself on another.

Lack of lateral thinking and ability to multitask

Gallbladder disease Hair loss Headaches Hypoglycemia Hysterectomy (ovaries removed) Increased blood clotting (increasing risk of strokes) Infertility Irregular menstrual periods Irritability Insomnia Magnesium deficiency Memory loss Mood swings Osteoporosis PMS PCOS Premenopausal bone loss Premature births Prostate cancer (men) Sluggish metabolism Thyroid dysfunction mimicking hypothyroidism Uterine cancer Uterine fibroids Water retention, bloating	Feeling fragmented physically, emotionally, and spiritually Headaches and migraines: sharp pains through top of head Overwhelming panic attacks and unfounded fear Social phobia, sense of loss of social skills, withdrawal Unrelated grief and sadness Sluggish liver (aggravated by hormonal overload, overuse of synthetic HRT medications, xenoestrogens) Vocabulary/speech difficulty: "tongue tied," verbal stammer Cyclic throat problems: too many sore throats around ovulation time, throats that do not clear, consistent sore throats every month, tonsillitis, asthma, upper respiratory problems Acne or pimples, particularly just prior to menses, also in older women Premature wrinkling Chronic recurrence of thrush, cystitis, vaginitis Reports of acne on the vulva that flares at menses Chronic candida, bouts of diarrhea prior to the menses, some alternating constipation, especially with women who have cysts and endometriosis. Inability to focus Inability to concentrate Loss of short-term memory Alienation and loss of confidence Androgen side effects: facial hair, increased body hair Increased thickening and blackening of limb hair

Zinc deficiency Gritty, dry eyes Blurred vision and/or watery eyes, difficulty focusing Tender heels and/or feet, from sensitive to burning Leaky gut syndrome Inflamed bowel problems: colitis, irritable bowel syndrome, leaky gut syndrome Inability to lose weight and shift fluid Loss of control over bladder (stress incontinence), inability to empty, tender and sensitive (absence of bladder infection), fluctuation /variation of bladder paralysis Extreme dream agitation, anxiety, panic attacks	Atypical periods alternating from shorter or extending to longer, cycles become erratic Alternate from heavier to lighter periods, or a combination of both, fibroid tumors

Hormonal imbalances masquerading as any one of the above conditions may take many years to accumulate and can be insidious in their physical presentation.

Just remember, it took time to arrive at this point in your life, and it may take time to improve, reverse, and eliminate many of these problems.

Estrogen Dominance and Disease

The majority of cases of breast cancer, uterine cancer, and fibroid tumors are attributed to the effects of estrogen. Menstrual cramps, midcycle pain, and heavy periods are due to estrogen dominance. Postmenopausal bleeding is often attributed to estrogen dominance, and candida overgrowth in the gut and female genitalia is linked to estrogen.

In addition to causing cancer, too much estrogen and low progesterone in women are associated with benign tumors such as fibroid tumors, ovarian cysts, fibrocystic breast disease, endometriosis, and cervical dysplasia with abnormal Pap smear.

In men, the highly prevalent condition known as benign prostatic hypertrophy (BPH) is attributed to estrogen dominance. Symptoms of BPH include a frequent urge to

urinate due to pressure on the urethra caused by the overgrown prostate. Complete urethral obstruction can sometimes result. Prostate cancer, surprisingly, is also attributable to estrogen dominance.

A more complete list of pathologies that are potentially caused by endocrine-disrupting chemicals includes the following:

Environmental allergies
Food allergies and food sensitivities
Chronic sinusitis
Asthma
Atopic dermatitis
Migraine headaches
Hair loss (alopecia)
Hyperlipidemia
Hypertension
Diabetes
Cardiovascular diseases
Abdominal issues (bloating, cramps, constipation)
Diarrhea (irritable bowel syndrome (IBS) ymptoms)
Candida (vulvovaginitis)
Recurrent urinary tract infections (UTIs)
Dysmenorrhea, menorrhagia, irregular menses
Ovarian cysts, polycystic ovarian syndrome (PCOS)
Fibroid tumors
Fibrocystic breast disease
Endometriosis
Cervical dysplasia with abnormal Pap smear
Benign prostatic hypertrophy (BPH) in men with increased frequency of urination
Nocturia
Arthritis
Fibromyalgia
Urticaria
Angioedema
Hypoglycemic episodes and hypotension leading to lightheadedness and dizziness
Agitation
Anger
Behavioral problems
Grumpiness
Decreased concentration
Depression
Anxiety
Sleep disturbance
Hyperphagia (overeating)
Obesity
Hypothyroidism
Hashimoto's Thyroiditis

Graves' Disease
Autoimmune diseases (lupus, rheumatoid arthritis, multiple sclerosis)
Hot flashes
Night sweats
Foggy thinking
Fatigue
Cancers

Table 1 identifies symptoms attributed to estrogen dominance or progesterone deficiency.

Table 1. Differential Diagnosis of Symptoms Caused by Estrogen Dominance or Progesterone Deficiency

Estrogen Excess Problems	**Progesterone Deficiency Problems**
Nasal allergies Asthma Multiple chemical sensitivity BCP-induced blood clot Abnormal weight gain Morbid obesity Food allergy (gluten sensitivity) Anxiety/depression/panic attacks Flushing of the face/chest with or without beer ingestion Abdominal bloating/cramping/diarrhea with gluten ingestion Premenstrual migraines Chronic migraines	Dysmenorrhea (painful menstrual cramps) Menorrhagia (heavy menstrual bleeding) Endometriosis Hyperemesis gravidarum (severe nausea and vomiting during pregnancy) Postpartum depression Fibromyalgia Infertility

Estrogen Dominance and Its Relationship to Obesity, Diabetes, and Hypertension

Estrogen dominance leads to an increase in insulin, which is often first noticed as hypoglycemic (low-blood sugar) episodes, especially in women. Around noon and before lunch, the blood glucose drops due to the high insulin level. As a result, the individual feels shaky, nervous, and easily irritable.

Overproduction of insulin causes the body to make fat cells. Insulin directs glucose into fat cells, and when fat cells can no longer accommodate the levels of glucose in the body, a condition called insulin resistance develops. Insulin resistance leads to diabetes.

Insulin resistance also plays a role in hypertension by causing salt retention, which elevates blood pressure. Approximately half of patients who have hypertension have insulin resistance. Although high blood pressure is often referred to as "essential" hypertension, there is nothing essential about it. It is the result of many factors, and a primary one is insulin resistance.

When progesterone decreases, estrogen increases, and that leads to insulin increase. Initially, increased insulin causes hypoglycemic episodes, which are more common in women than in men. Hospital discharges for hypoglycemia in 2008 were most common in women living in the South and in the Midwest, followed by the Northeast and by the West. This trend is the same for most estrogen-dominance-related conditions and for hypotension (low blood pressure). When progesterone decreases, aldosterone initially decreases, and that leads to hypotension. I have seen several patients, especially women, who complain of hypotension. This low blood pressure is associated with low progesterone.

Low blood pressure causes the kidneys to release renin, which converts angiotensinogen I to angiotensin I; angiotensin I converts angiotensinogen II to angiotensin II using angiotensin- converting enzyme (ACE). Angiotensin II causes the aldosterone level to increase. This increase in aldosterone often overshoots the mark, and the patient's blood pressure eventually increases and requires therapy. Often ACE inhibitors, such as lisinopril, captopril, enalapril, Monopril, and other -pril blood pressure medications, and angiotensin receptor blockers (ARBs), such as losartan, candesartan, valsartan, and other -sartan medications, are used as blood pressure lowering agents.

High insulin also causes salt retention in the renal tubules, which ultimately leads to high blood pressure. These two mechanisms are major reasons for an increase in blood pressure, which is often called essential hypertension, meaning the cause is unknown. Estrogens directly cause high blood pressure; therefore, women with chronic estrogen dominance have more hypertension emergency department and hospital visits than men. The South—with its plethora of pesticides and, as a consequence, high levels of xenoestrogens—has the highest rate of hypertension in the country, followed by the Midwest, the Northeast, and the West.

The Effect of Estrogen Dominance and Progesterone Deficiency on the Thyroid

Too much estrogen causes an increase in thyroid-binding globulin (TBG), which is the protein that circulates thyroid hormones in the body. When thyroid-binding globulin levels increase, this protein binds all thyroid hormones, preventing them from being released for their intended effects, which include metabolism regulation. An increase in TBG causes low metabolism and increased fat-cell production. Oral, but not transdermal, estradiol increases TBG, whereas testosterone lowers TBG. Testosterone increases T3/T4 ratios. Estradiol does not affect T3/T4 ratios, regardless of the route of administration; therefore, the T4 substitution (Synthroid or Levoxyl) dose in women with primary hypothyroidism (characterized by impaired endogenous T4 production) must be increased when oral estrogens are administered. When progesterone is low, the thyroid may malfunction because progesterone helps the thyroid tissue absorb minerals such as selenium, potassium, zinc, and iodine, which are needed to convert the thyroid prohormone T4 into the active form T3. If these minerals are not present in the thyroid, T4 converts into a stored form called reverse T3 (rT3). Many healthcare providers, when they measure the thyroid, tend to measure only TSH (thyroid-stimulating hormone) and, if it is within normal range (0.45-4.5 reported by most laboratories), they pronounce the thyroid normal. This is not the case if the patient is obese or overweight. If an individual has problems with weight, it is likely the thyroid is not working properly as a result of too much thyroid binding globulin. With the advent of the estrogen and obesity epidemics, many women have developed an inflammatory autoimmune attack on their thyroid gland known as Hashimoto's thyroiditis. This condition is diagnosed by the presence of thyroid antibodies such as antithyroglobulin antibody or thyroid peroxidase (TPO) antibody in the blood. Studies have shown that production of these thyroid antibodies goes hand-in-hand with gluten and dairy sensitivity. Remember, dairy products may be estrogenic because of the growth hormones and other chemicals administered to cows that produce the milk. Cows are fed GMO corn that contains about 25 chemicals and at least two—glyphosate and

atrazine—are well-known estrogenic compounds. Alachlor, acetochlor, metolachlor, and metolachlor-S are heavily used in corn production. These chemicals are thyroid disruptors; hence, the estrogenic chemicals coupled with these thyroid disruptors may explain the underactive thyroid epidemic seen in the U.S. In addition, gluten-containing grains (barley, rye, wheat, spelt, kamut) are weak, plant-based estrogens. Since women, in general, have more estrogen than men, this may be the reason women have a higher incidence of Hashimoto's thyroiditis than men. The gluten protein has been demonized in recent years, but humans have been eating gluten-containing grains for thousands of years without associated health problems until recently. In recent years, especially, since the 1990s, we have seen a surge of the so-called gluten sensitivity. The increased gluten sensitivity correlates with the increased estrogen load in our environment. If you have weight issues and/or thyroid issues, you may want to stop eating gluten-containing grains and dairy as well as avoiding other foods and chemicals that are estrogenic. In most cases, you will start losing weight and your thyroid antibodies may decrease.

Inflammatory Conditions Associated with Progesterone Deficiency

The following inflammatory conditions are some, but not all the conditions that have been associated with insufficient progesterone and too much estrogen:

- Arthritis
- Asthma
- Endometriosis
- Fibromyalgia
- Interstitial cystitis (IC). Interstitial cystitis is a chronic, inflammatory condition of the submucosal and muscular layers of the bladder. It is a mast-cell disease that is more prevalent in women than in men. Estrogen dominance in women may cause IC. When estrogen binds to its receptor on the mast cell, it causes mast cell degranulation in the detrusor muscles of the bladder, which leads to IC symptoms. This is the reason antihistamines, such as hydroxyzine, Zyrtec and Allegra help to alleviate IC symptoms. Hyperestrogenic problems are solved by balancing the estrogens; hence, giving adequate amounts of progesterone and encouraging patients to eat cruciferous vegetables and/or to take cruciferous vegetables extract-based nutritional

supplements such as DIM (diindolylmethane) and I3C (Indole-3-Carbinol) may help IC patients.

- Multiple sclerosis
- Nonallergic rhinitis, and seasonal/perennial allergic rhinitis
- Obesity and asthma
- Premenstrual asthma (PMA)
- Premenstrual migraines
- Premenstrual dermatitis

The Effects of Estrogen on Coagulation

Coagulation (also known as clotting) is the process by which blood changes from a liquid to a gel. It potentially results in hemostasis—the cessation of blood loss from a damaged vessel, followed by repair. The mechanism of coagulation involves activation, adhesion, and aggregation of platelets along with deposition and maturation of fibrin.

The most consistent effects of estrogens on coagulation proteins follow:

- Elevation of thrombin and fibrinogen, two components of the coagulation process.
- Elevation of factors II, VII, IX, X, XII, and kallikrein, all of which participate in the coagulation process.
- Elevation of protein C and plasminogen, regulators of coagulation.

Although these elevations have been attributed to the estrogenic component in oral contraceptives, the progestin concentration may also influence these increases. This is the reason some women develop blood clots when they go on estrogen pills or estrogen alone hormone replacement such as Premarin and an estrogen/progestin combination such as PremPro.

The Varieties of Hormone Imbalances

Based on my clinical evaluation of patients who present with hormone imbalance, the laboratory results show twelve different hormonal patterns. The first group has no deficiency; there are very few of these individuals. The second group only lacks progesterone; the third group lacks both progesterone and estradiol; the fourth group lacks progesterone, estradiol, and dehydroepiandrosterone (DHEA). The fifth group lacks progesterone, estradiol, DHEA, and testosterone. Those individuals also often lack pregnenolone. The remaining groups show variations in the presence and absence of these hormones.

Estrogen dominance leads to increased production of insulin. When insulin increases, we crave sweets. This is the reason a woman craves sweets in the late luteal phase of her menstrual cycle. When a woman is closer to her period, her progesterone drops tremendously, but the estrogen is noticeably higher than the progesterone, thereby becoming dominant. This craving occurs because insulin goes up when estrogen is dominant. The estrogen effect on insulin gets worse when women reach their midthirties. In the midthirties, women have many menstrual cycles without ovulation, known as anovulatory menstrual cycle. The progesterone declines without ovulation and that leads to estrogen dominance. It is, therefore, interesting to note that many women start gaining weight in their midthirties and beyond.

If starches or sugar-containing foods are consumed, blood sugar (glucose) levels increase. The insulin then takes the glucose the body does not need for energy and stores it in fat cells. If this process continues, an individual will start to gain weight. If your blood glucose level is low and your insulin level is high, as often happens in late morning before lunch, you may feel shaky and even sweaty because you are having a hypoglycemic episode. This causes your adrenaline to rise, and glucagon is released to try to send whatever sugar that is in your body to the brain. The rise in adrenaline may cause you to feel hyper and impatient.

Leptin and Leptin Resistance

If you gain weight because of ongoing high insulin levels, a point is reached when the fat cells have had enough of this weight-gain business. They send a messenger molecule called leptin to the hypothalamus, which is the center for satiety and appetite located in the brain, to tell the hypothalamus they are saturated. The hypothalamus initially receives this message and alerts the insulin to stop the craving and suppress the appetite so the fat cells can decompress; however, nobody

sees weight loss overnight, including the fat cells, which continue to send more messages to the hypothalamus.

Eventually the hypothalamus suffers message overload and stops responding. When leptin's message is ignored, this is known as leptin resistance. Leptin resistance leads to insulin resistance, which simply means that the insulin now has no capacity to continue to get fat cells to accept glucose. As a result, the glucose remains in the blood; at high levels, this is known as diabetes. [From "Estradiol Binds to Insulin and Insulin Receptor Decreasing Insulin Binding *in vitro*." Robert Root-Bernstein, Abigail Podufaly, and Patrick F. Dillon. *Frontiers in Endocrinology* (Lausanne). 2014; 5: 118. Published online July 21, 2014. doi: 10.3389/fendo.2014.00118.

The Effects of Leptin Resistance on Mental Health, Thyroid Function, Inflammation, and Weight Gain

When you become leptin resistant, the levels of a chemical called serotonin, which is the "feel-good" hormone, decline and you experience anxiety, depression, and panic attacks. To alleviate these symptoms, your primary care doctor may put you on an antidepressant, which, in turn, suppresses your thyroid function. Low thyroid function is linked with weight gain, more anxiety, and fatigue. Fat cells also produce TNF-alpha and IL-6, which are inflammatory chemicals (cytokines). These substances cause aches and pains such as joint pain, back pain, muscle pain, and the generalized muscle aches known as fibromyalgia. TNF-alpha and IL-6 are also implicated in asthma and allergy symptoms. The more fat cells you have, the greater the production of these substances. Fat cells stimulate skin cells and the adrenal glands to produce more estrogen. The fat cells have an enzyme (chemical) in them called aromatase that converts testosterone to estrogens. Therefore, if you have a weight problem, you most likely have estrogen dominance and you may also have low testosterone as a consequence.

Over the past eight years, I have measured testosterone levels in men and women and found that even young men and women can have low testosterone. Low testosterone leads to low sex drive (decreased libido). This can cause marital discord that often leads to divorce. By the way, the number one reason for divorce in America is marital infidelity. It's not money! So please, before you divorce, fix your hormones!

Low testosterone also causes foggy thinking, forgetfulness, and apathy; and, in men, often unyielding (anankastic personality disorder), territorial, and grumpy behavior. Unfortunately, men in charge of the world affairs are middle-aged men who are

experiencing the effects of low testosterone—known in the vernacular as "low T." These men make decisions in government, and often they do not agree. The consequences of their behavior can be disastrous and can be seen in such examples as waging wars that are meaningless. When we see the evil face of war in a person such as Hitler, we should wonder, "What was his hormone status?" I will bet you that he had low T. Playing golf on chemically contaminated golf courses may not help your testosterone level. Above all, do not clean your golf ball with your tongue.

PART IIA: OBESITY

THE OBESITY MODEL

When patients present with obesity, hypertension, diabetes, or hyperlipidemia, the recommendation is usually to make some lifestyle changes to lose weight and address associated problems. If, within three months, the patient has not made any progress, medication therapy is implemented.

This practice, which is standard in medical offices around the world, actually makes little sense: most patients cannot lose excess weight permanently on their own and almost inevitably end up with medication therapy that attempts to control comorbidities associated with their obesity. Medication therapy for hypertension and diabetes may compound the problem by causing even more weight gain. From the standpoint of common sense, this is an absurd situation, but the fact is that the orthodox medical community continues to fight obesity using the same assumptions that have proven to be inadequate in slowing the obesity rate. I think it is time we examine our premises and consider new possibilities when it comes to understanding the cause of obesity. My research and clinical practice have led me to question the assumption that obesity hinges solely on patients' wills to keep their weight in check. The evidence strongly suggests that something more sinister is causing the epidemic of overweight in the Western world, particularly in America.

I now believe that most of the world's obesity today is due to hormone imbalance. As the evidence presented throughout this book proves, hormone imbalance, such as estrogen dominance, is caused by pesticides and herbicides used in farmlands and by other chemicals used in our daily lives. The organic food movement took hold in large part because many people rightly suspected that synthetic chemical (nonorganic) farming leads to contamination of farm products and the water sources, and that this leads to diseases such as obesity, cancer, and the rest. Frighteningly, this is indeed the case.

We continue to hear the argument that fast food and other poor dietary choices are the villains responsible for the obesity epidemic. Healthcare providers who see, firsthand, the impact of the obesity epidemic on their patients' health have largely refused to recognize the other possible etiologies. Often they are so caught up in the treatment of the symptoms of obesity that they forget about the more fundamental issues. Also, the financial pressures imposed on them as employees of massive, organized, medical conglomerates largely stifle any motivation to ask the big questions about the causes of the crisis. In medical schools, moreover, there are no courses dedicated to discovering the underlying cause of obesity. The emphasis is on the comorbidities instead of obesity itself.

The hypothesis commonly postulated is that obesity is the result of excessive food consumption and a sedentary lifestyle. The quality of the foods is often questioned, and so-called "junk foods" are seen as the culprits of the obesity epidemic. Many people assume that low-income individuals have a greater tendency to consume these junk foods and, therefore, bear the burden of obesity in the U.S. This is simply not the case.

HISTORY OF THE OBESITY EPIDEMIC IN THE U.S. AND WORLDWIDE

Obesity trends show that obesity has markedly increased in both adults and children since 1996. The CDC started to track obesity and published the first obesity map in 1985. Obesity in the U.S. showed a marked spike in the 1996-1997 data.

What happened in our country prior to the mid-1990s that caused this increase? Why are women more prone to obesity than men? In this section, I will provide answers to these questions.

Obesity is the number-one killer in the U.S. and, increasingly, the rest of the world because it is the source of many major diseases that cause the highest death rates worldwide. The obesity comorbidities shown in the following diagram illustrate this statement very well.

Figure 2. Comorbidities Associated with Obesity

There are increasing rates of morbidity and mortality associated with obesity comorbidities. These include:

- Cardiovascular diseases
- Respiratory diseases
- Gastrointestinal diseases
- Hypertension
- Diabetes

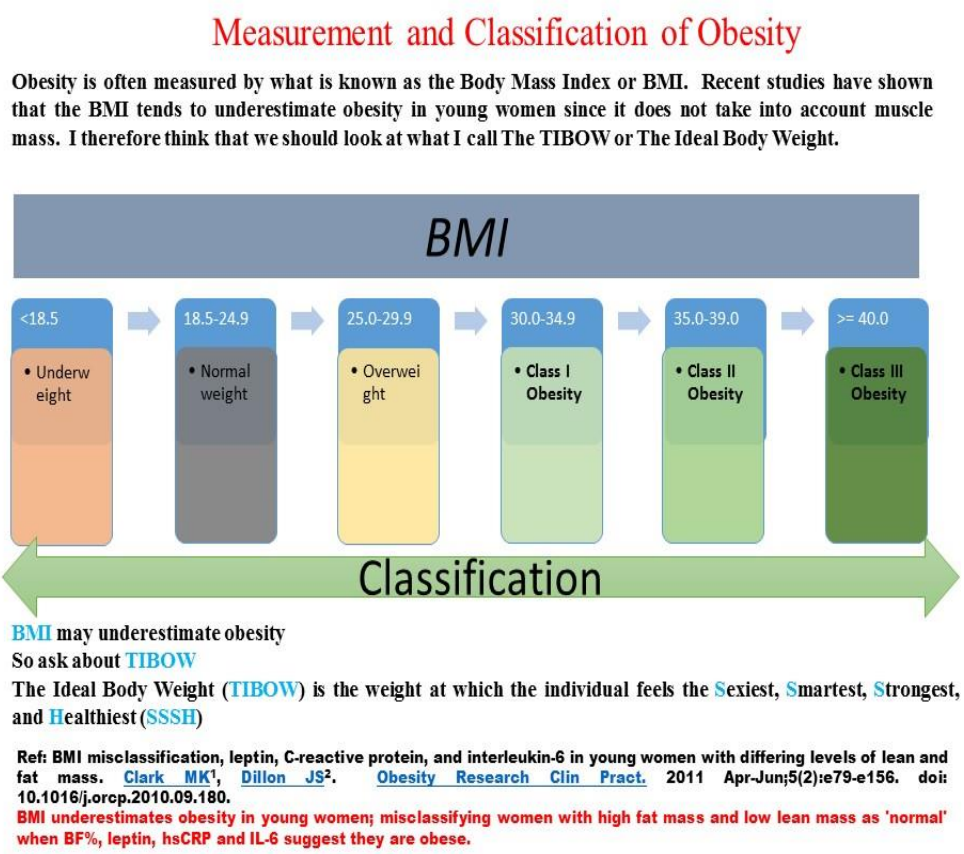
Hormone imbalances are also associated with obesity, and these have their own set of comorbidities:

- Hyperlipidemia and its consequences
- Cancer and other tumors
- Arthritis
- Allergic diseases

FORGET BMI, AND FOCUS ON TIBOW AND SSSH

Obesity, by definition, is present if an individual has a BMI (Body Mass Index) greater than or equal to 30. Overweight is defined as a BMI of 25–29.9. BMI is measured as weight in kilograms/height in meters squared. Recent studies have shown that BMI assessment tends to underestimate obesity in young women because it does not consider muscle mass. BMI underestimates obesity in young women by misclassifying women with high fat mass and low lean mass as 'normal' when body fat percentage, leptin level, hsCRP, and IL-6 suggest these women are obese.

Therefore, I think we should look at what I call TIBOW, The Ideal Body Weight. The Ideal Body Weight is the weight at which the individual feels the Sexiest, Smartest, Strongest, and Healthiest (SSSH). [Source: "BMI misclassification, leptin, C-reactive protein, and interleukin-6 in young women with differing levels of lean and fat mass." Clark MK, Dillon JS. *Obesity Research Clinical Practice* 2011 Apr-Jun; 5(2):e79-e156. doi: 10.1016/j.orcp. 2010.09.180].



OBESITY TYPES

There are three main morphotypes (body shapes) noted in obesity—abdominal obesity, hip area obesity, and a combination of the two.

The abdominal obesity group has a protruding abdomen due to insulin resistance and leptin resistance. Most of these individuals have diabetes or will have diabetes. Some of these future diabetics are often told by their primary care physicians that they have a pre-diabetic condition. The activity of insulin in this group may be related to stress that causes cortisol release. Cortisol, in turn, causes increases in insulin and blood glucose. Insulin directs glucose into the fat cells of the omentum (a sheet of fat that covers the intestines), which leads to ventral obesity.

In the second obesity group, fat forms primarily around the hips. This distribution is more prevalent in women and is often associated with estrogen dominance.

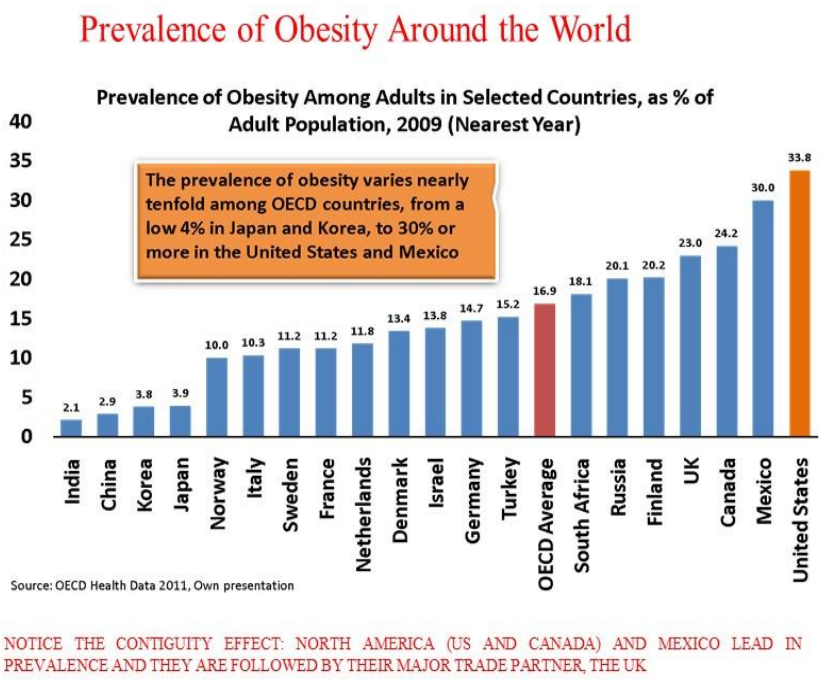
The third obesity group is the combination of the first two. This group exhibits fat around the hips and a protruding abdomen. These individuals have cortisol-insulin problems and estrogen dominance.

I have coined a new term—obesity à deux. Family members who live in the same household tend to have the same obesity pattern, even if they are not genetically related (such as husband and wife). This, again, indicates that environment may be the major culprit.

GLOBAL AND U.S. OBESITY STATISTICS

Here is a review of the prevalence of obesity among adults in selected countries around the world, as of 2009. As you can see from the chart below, the U.S. has the highest rate of adult obesity, with nearly 34% of adults classified as obese.

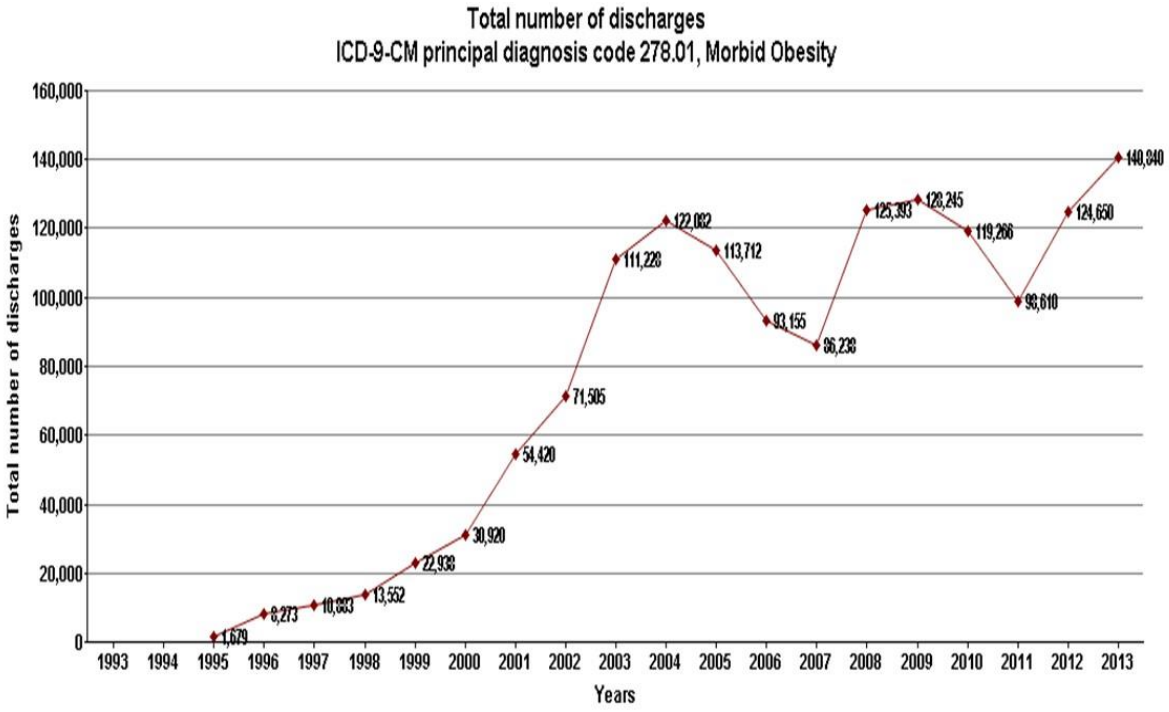
Figure 3. Prevalence of Obesity around the World



NOTICE THE CONTIGUITY EFFECT: NORTH AMERICA (US AND CANADA) AND MEXICO LEAD IN PREVALENCE AND THEY ARE FOLLOWED BY THEIR MAJOR TRADE PARTNER, THE UK

Figure 4 gives us another view of obesity in the U.S. This chart tracks the number of people discharged from hospitals between 1997 and 2013 who had the primary diagnosis of morbid obesity. You can see that this number increased 10-fold between 1997 and 2010, dropped significantly in 2011, then bounced back in 2013.

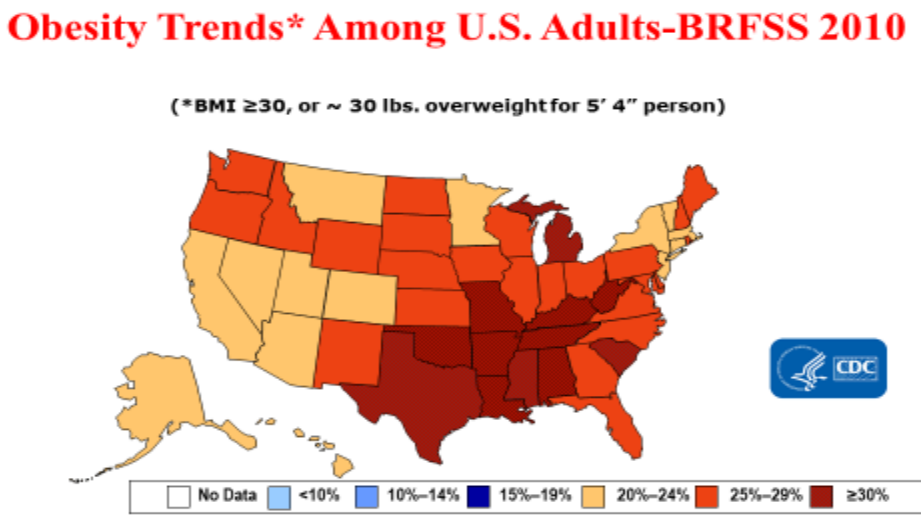
Figure 4. U.S. Obesity Trends: 1995-2013



Obesity Trends by State

Generally speaking, the South and Midwest are the areas of the U.S. with the highest rates of obesity, as shown on the map in Figure 5.

Figure 5. Obesity Trends in U.S. Adults



The states with the highest obesity rate in 2010 are shown in Figure 6. All but two of these states are located in the South.

Figure 6. U.S. States with Highest Obesity Rate

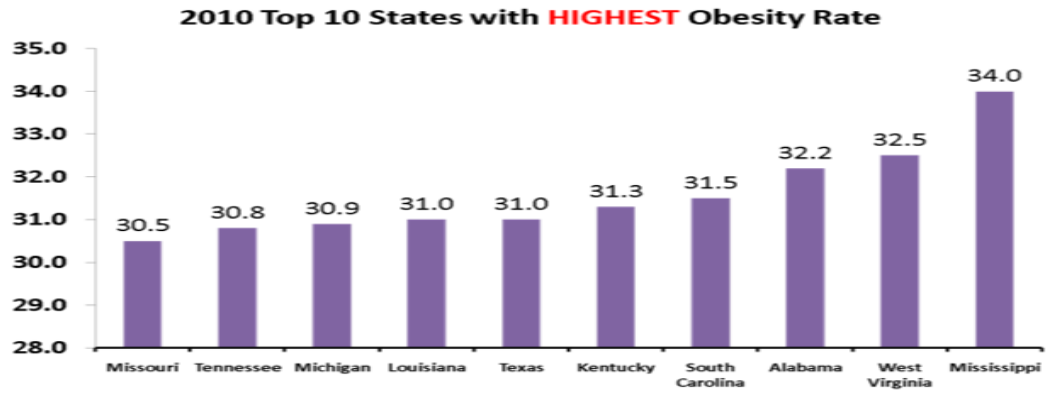


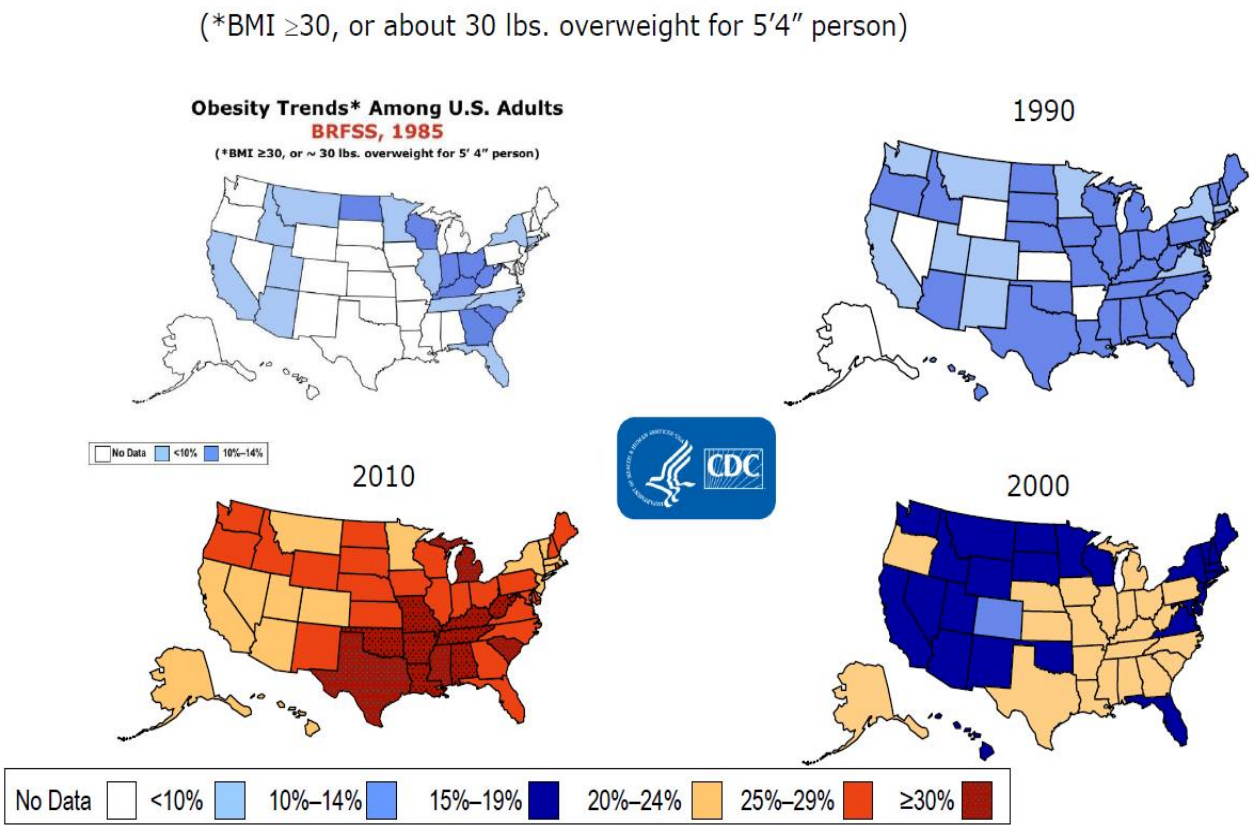
Figure 7. Adult Obesity Rates and Ranking by State, 2012

Rank	State	2012 obesity rate	95% confidence interval
1	Louisiana	34.7%	+/- 1.6 pts.
2	Mississippi	34.6%	+/- 1.6 pts.
3	Arkansas	34.5%	+/- 1.9 pts.
4	West Virginia	33.8%	+/- 1.6 pts.
5	Alabama	33.0%	+/- 1.5 pts.
6	Oklahoma	32.2%	+/- 1.4 pts.
7	South Carolina	31.6%	+/- 1.2 pts.
8	Indiana	31.4%	+/- 1.3 pts.
9	Kentucky	31.3%	+/- 1.4 pts.
10	Michigan	31.1%	+/- 1.3 pts.
10	Tennessee	31.1%	+/- 1.6 pts.
12	Iowa	30.4%	+/- 1.4 pts.
13	Ohio	30.1%	+/- 1.1 pts.
14	Kansas	29.9%	+/- 1.2 pts.
15	North Dakota	29.7%	+/- 1.8 pts.
15	Wisconsin	29.7%	+/- 1.9 pts.
17	Missouri	29.6%	+/- 1.6 pts.
17	North Carolina	29.6%	+/- 1.1 pts.
19	Texas	29.2%	+/- 1.4 pts.
20	Georgia	29.1%	+/- 1.7 pts.
20	Pennsylvania	29.1%	+/- 1.0 pts.
22	Nebraska	28.6%	+/- 0.9 pts.
23	Maine	28.4%	+/- 1.2 pts.
24	Illinois	28.1%	+/- 1.7 pts.
24	South Dakota	28.1%	+/- 1.6 pts.
26	Maryland	27.6%	+/- 1.3 pts.
27	Virginia	27.4%	+/- 1.4 pts.
28	New Hampshire	27.3%	+/- 1.5 pts.
28	Oregon	27.3%	+/- 1.7 pts.
30	New Mexico	27.1%	+/- 1.2 pts.
31	Delaware	26.9%	+/- 1.7 pts.
32	Idaho	26.8%	+/- 2.0 pts.
32	Washington	26.8%	+/- 1.0 pts.
34	Nevada	26.2%	+/- 1.9 pts.
35	Arizona	26.0%	+/- 1.8 pts.
36	Alaska	25.7%	+/- 1.8 pts.
36	Minnesota	25.7%	+/- 1.1 pts.
36	Rhode Island	25.7%	+/- 1.6 pts.
39	Connecticut	25.6%	+/- 1.3 pts.
40	Florida	25.2%	+/- 1.6 pts.
41	California	25.0%	+/- 1.1 pts.
42	New Jersey	24.6%	+/- 1.0 pts.
42	Wyoming	24.6%	+/- 1.8 pts.
44	Montana	24.3%	+/- 1.2 pts.
44	Utah	24.3%	+/- 1.0 pts.
46	Vermont	23.7%	+/- 1.4 pts.
47	Hawaii	23.6%	+/- 1.6 pts.
47	New York	23.6%	+/- 1.5 pts.
49	Massachusetts	22.9%	+/- 0.9 pts.
50	District of Columbia	21.9%	+/- 2.1 pts.
51	Colorado	20.5%	+/- 1.0 pts.

These rates show that in 2012, the South and the Midwest continue to lead in the obesity.

Below is a fascinating graphic showing the obesity rates among U.S. adults by state in 1985, 1990, 2000, and 2010. We have gone from a country in which less than 15% of adults were obese in 1985 to a nation in which more than 30% of adults in 12 states are obese in 2010.

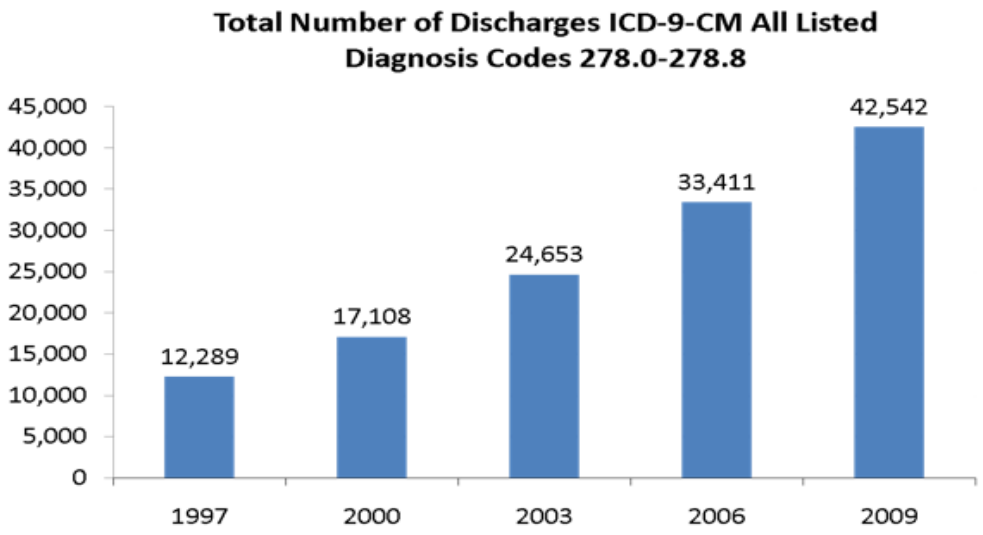
Figure 8. Obesity Trends by State and Year—1985, 1990, 2000, 2010



OBESITY AMONG AMERICAN CHILDREN

Obesity in children living in the U.S. more than tripled from 1997 to 2009, as shown in the graphic below. The ICD-9-CM diagnosis codes 278.0 to 278.8 refer to obesity, in this case, obesity in children.

Figure 9. Obesity Trends in Children, 1997-2009



The chart below shows obesity rates in children by age, gender, income level, and geographic area. Generally, older teenagers comprise the majority of obese children between the ages of 10 and 17. Post-pubertal girls have higher obesity rates than pre-pubertal girls or boys. Children in the West represent about 20% of obese children nationally. Also, higher income predicts higher obesity, which is not what we generally expect. It is often believed that obesity correlates with low-income populations.

Figure 10. Obesity Trends in Children by Age, Gender, Income Level, and Geographic Area.

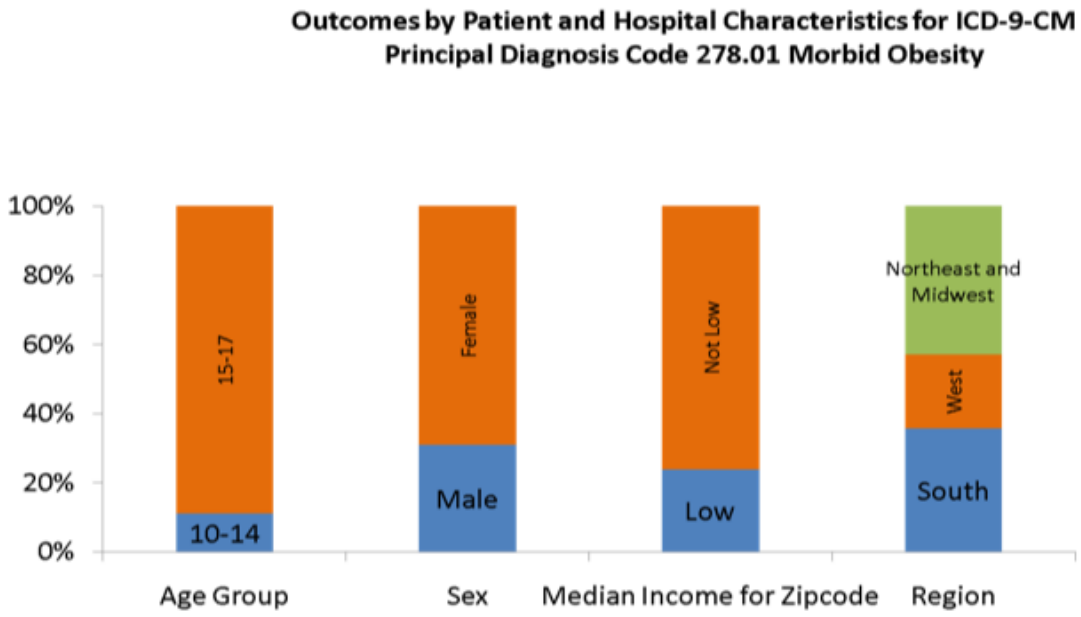
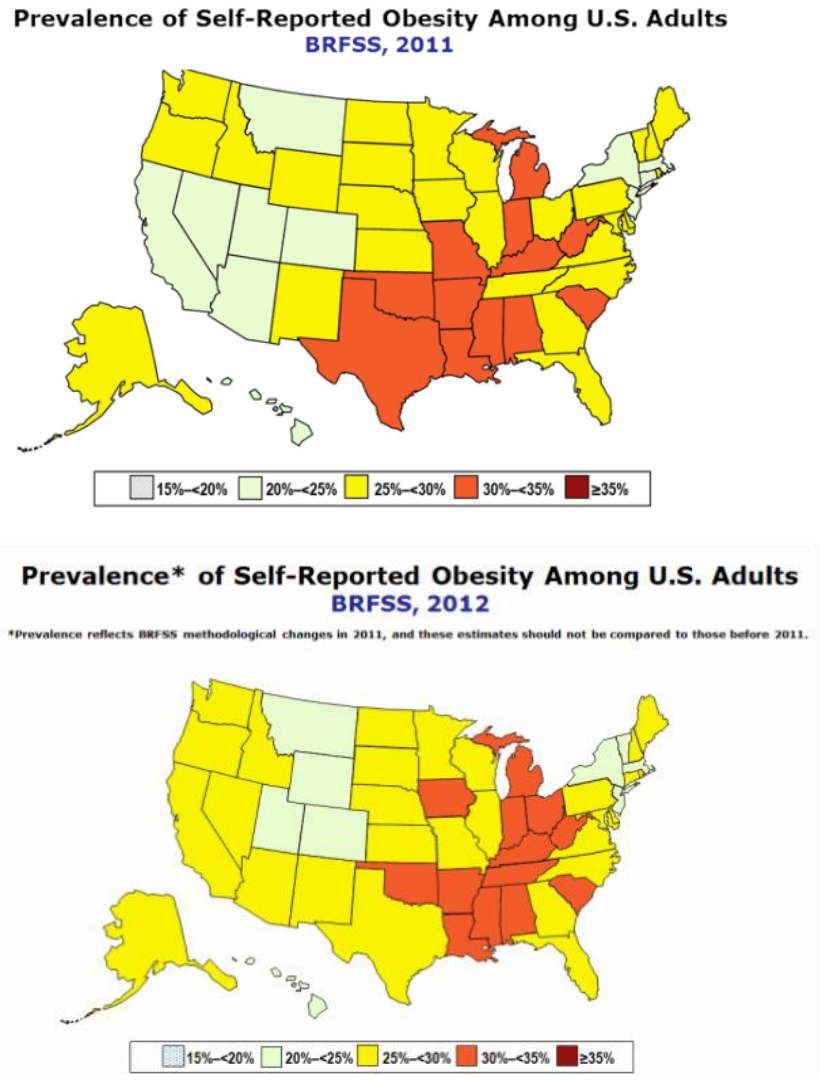


Figure 11. Prevalence of Self-Reported Obesity Rates among U.S. Adults, by State, in 2011 and 2012.



WHAT DO THE "FAT" STATES HAVE IN COMMON?

It is clear from the obesity maps that the southern states are at the forefront of a trend that affects all the other states, decreasing as it proceeds to the Midwest, the Northeast, and finally the West. States in the Mississippi Delta show the highest rates of obesity and mortality. This may be due to many rivers in the Midwest and South draining into the Mississippi River, carrying in them endocrine disrupting-pesticides and herbicides sprayed on farmlands upstream, as shown in the graphic below.

Figure 12. Major Rivers of the Contiguous U.S. that Drain into the Mississippi Embayment States

Major Rivers of the Contiguous US

This map shows that the most important rivers in the Midwest and the South drain into the Mississippi river that carries with it all the pesticides and herbicides from farm lands in the upper Midwest and Midwest into the Mississippi Embayment States (Mississippi, Louisiana, Arkansas, Missouri, Kentucky, Tennessee and contiguous neighbor states, Alabama, Texas, Oklahoma…)

River, Length in miles (flows into)

1. Missouri, 2,540 miles (flows into Mississippi River)
2. Mississippi, 2,340 miles (flows into Gulf of Mexico)
3. Yukon, 1,980 miles (flows into Bering Sea)
4. Rio Grande, 1,900 miles (flows into Gulf of Mexico)
4. St. Lawrence, 1,900 miles (flows into Gulf of St. Lawrence)
6. Arkansas, 1,460 miles (flows into Mississippi River)
7. Colorado, 1,450 miles (flows into Gulf of California)
8. Red, 1,290 miles (flows into Mississippi River)
9. Brazos, 1,280 miles (flows into Gulf of Mexico)
10. Columbia, 1,240 miles (flows into Pacific Ocean)
11. Snake, 1,040 miles (flows into Columbia River)
12. Platte, 990 miles (flows into Missouri River)
13. Ohio, 981 miles (flows into Mississippi River)
14. Pecos, 926 miles (flows into Gulf of Mexico)
15. Canadian, 906 miles (flows into Arkansas River)
16. Tennessee, 886 miles (flows into Ohio River)
17. Colorado, 862 miles (flows into Matagordo Bay)
18. North Canadian, 800 miles (flows into Canadian River)
19. Mobile, 774 miles (flows into Gulf of Mexico)
20. Kansas, 743 miles (flows into Missouri River)
21. Kuskokwim, 724 miles (flows into Bering Sea)
22. Green, 730 miles (flows into Colorado River)
23. James, 710 miles (flows into Missouri River)
24. Yellowstone, 692 miles (flows into Missouri River)
25. Tanana, 659 miles (flows into Yukon River)
26. Gila, 630 miles (flows into Colorado River)
27. Milk, 625 miles (flows into Missouri River)
28. Quachita, 605 miles (flows into Red River)

Refs. Encyclopedia Britannica and Kammerer, J.C., May, 1990, *Largest Rivers in the United States*, US Geological Survey Fact Sheet, Open File Report 87-242. Note: Measuring the exact length of a river is diffcult, and the length can change over time. Many references cite different lengths for the same river. Averages have been taken in these cases

Southern states are also the oldest cotton-producing states, as shown in the table below. Cotton requires three times the amount of pesticides as any other crop in the U.S.

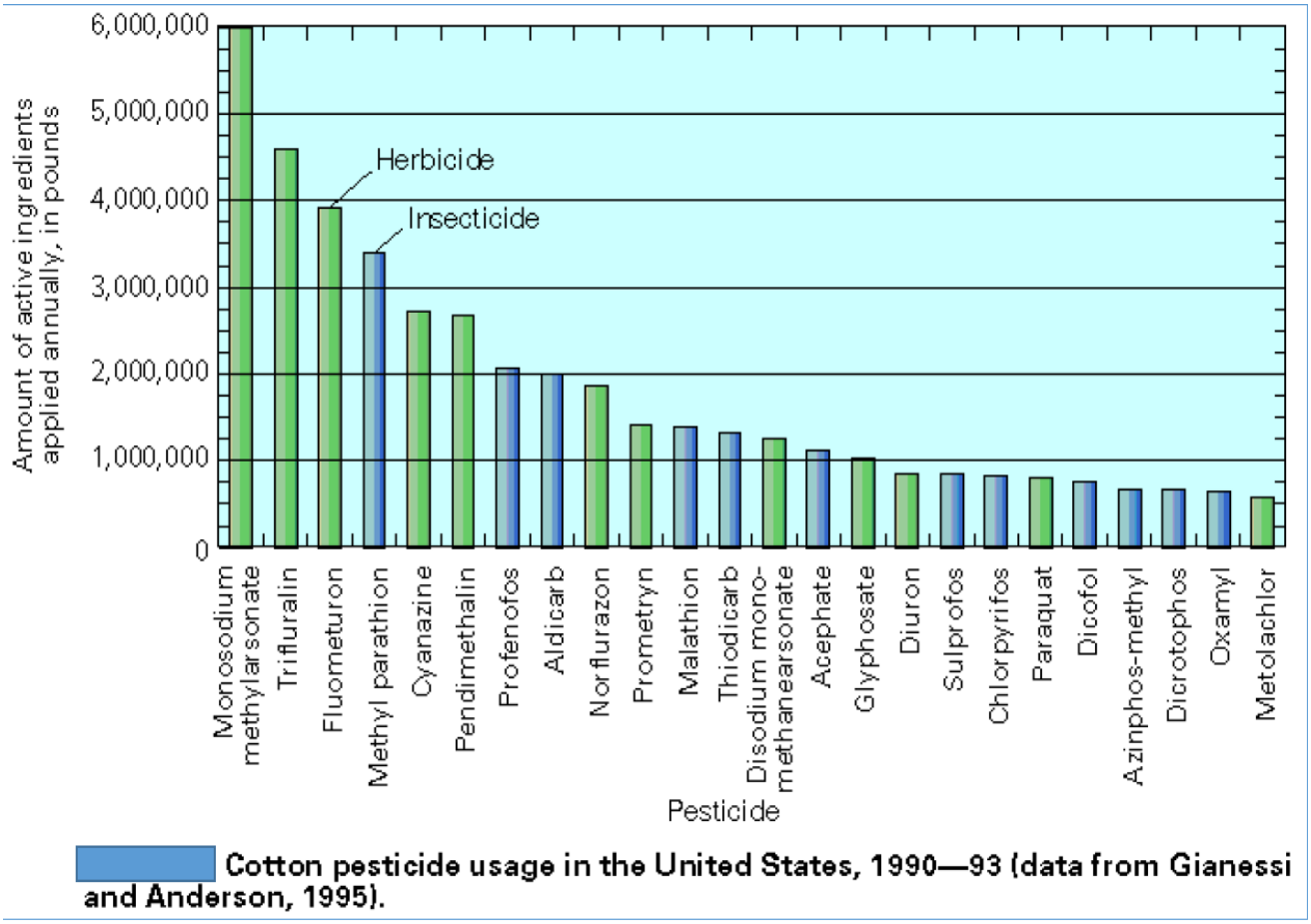
Table 2. U.S. Cotton Production by State, 2003-2004 to 2007-2008.

Upland	2003-2004*	2004-2005*	2005-2006*	2006-2007*	2007-2008*	5-year Average*+
Southeast	**4,529**	**4,631**	**5,153**	**5,048**	**3,237**	**4,528**
Alabama	820	814	848	675	416	745
Florida	117	109	135	166	116	125
Georgia	2,110	1,797	2,140	2,334	1,660	1,992
North Carolina	1,037	1,360	1,437	1,285	783	1,185
South Carolina	326	390	410	433	160	338
Virginia	119	161	183	155	102	143
Mid-South	**6,541**	**7,134**	**7,433**	**8,226**	**5,277**	**7,021**
Arkansas	**1,804**	**2,089**	**2,202**	**2,525**	**1,896**	**2,058**
Louisiana	**1,027**	**885**	**1,098**	**1,241**	**699**	**998**
Mississippi	**2,120**	**2,346**	**2,147**	**2,107**	**1,318**	**2,131**
Missouri	**700**	**830**	**864**	**985**	**764**	**798**
Tennessee	**890**	**984**	**1,112**	**1,368**	**600**	**1,036**
Southwest	**4,638**	**8,114**	**8,886**	**6,120**	**8,588**	**6,616**
Kansas	90	71	88	117	57	88
Oklahoma	218	303	358	203	281	258
Texas	4,330	7,740	8,440	5,800	8,250	6,270
West	**2,115**	**2,626**	**1,788**	**1,428**	**1,253**	**2,023**
Arizona	550	723	615	556	514	611
California	1,495	1,790	1,065	779	650	1,318
New Mexico	70	113	108	93	89	94
Total Upland	***17,823***	***22,505***	**23,260**	**20,822**	**18,355**	***20,188***
ELS	**2003-2004**	**2004-2005**	**2005-2006**	**2006-2007**	**2007-2008**	**5-year Average***
Arizona	5	6	7	13	5	10
California	371	683	558	687	793	580
New Mexico	13	19	22	20	8	18
Texas	44	38	44	45	46	43
Total ELS	***432***	***746***	***631***	**765**	**852**	***650***
All Cotton	**18,255**	**23,251**	**23,890**	**21,588**	**19,207**	**20,839**

Source: NASS, USDA. Note: Numbers may not add up due to rounding.
**Thousand Bales (480 lb. Bales) +5-year average is for Crop Year 2002-2006*

The States with largest cotton production are in red and they also have the highest obesity rates chronically. The contiguous States such as Alabama, Oklahoma and Texas also have high obesity rates

Figure 13. Pesticides and Herbicides Used in Cotton Production in the U.S., 1990-1993.



It is interesting to note that Glyphosate was used in cotton production before the advent of GMOs and continues to be used heavily in cotton and peanut production. This Glyphosate is coupled with many organophosphate and carbamate insecticides use in early 1990s.

THE REAL AND PURPORTED CAUSES OF OBESITY

It is often postulated that the fundamental cause of the current worldwide obesity epidemic is an energy imbalance between calories consumed versus calories expended. It is believed that, globally, there has been an increased intake of energy-dense foods that are high in fat, salt, and sugars and low in vitamins, minerals, and other micronutrients. Along with this increased consumption of high-calorie foods there has occurred a decrease in physical activity due to the increasingly sedentary nature of many forms of work. People simply move less. In addition, changing modes of transportation—driving rather than walking—along with increased urbanization have also lessened the calorie expenditures of most people worldwide.

Changes in dietary and physical activity patterns are often the result of environmental and societal changes associated with development combined with a lack of supportive policies in sectors such as health, agriculture, transport, urban planning, environment, food processing, distribution, marketing, and education.

From wrongful assumptions, wrong conclusions are drawn. It is often thought that overweight and obesity, as well as their related non-communicable diseases, are largely preventable at the community level. Supportive environments and communities are fundamental in shaping people's choices. By making the healthier selection of foods and regular physical activity the easiest choice, they will, therefore, prevent obesity. At the individual level, people can do the following:

- limit energy intake from total fats
- increase consumption of fruits and vegetables
- increase consumption of legumes, whole grains, and nuts
- limit the intake of sugars
- engage in regular physical activity
- achieve energy balance and a sustainable healthy weight

All of the above is true, to a certain extent, but it does not tell the whole story about the reason obesity has become epidemic. Endocrine-disrupting chemicals are the real backstory. As we have seen, regions in the U.S. that have the highest pesticide use correspond with a greater percentage of obese people in those areas. In Part IIC of this book, we will see how endocrine-disrupting pesticides cause obesity.

PART IIB: ALLERGIES

THE INCREASING PREVALENCE OF ENVIRONMENTAL AND FOOD ALLERGIES

Cases of environmental and food allergies are on the rise and are often frustrating to effectively treat because of a lack of understanding about their causes. Rhinitis, or inflammation of the nasal mucous membranes, can lead to postnasal drip, chronic or recurrent sinus infections, sinus pressure, headaches, and even asthma symptoms of chest tightness, shortness of breath, coughing, and wheezing. Food allergies in children and adults have reached epidemic proportions.

The bulk of environmental and food allergy cases are treated by primary healthcare providers or by patients who self-treat; however, many healthcare providers do not have a deep understanding of the biological mechanisms that cause allergic rhinitis or food allergy symptoms. Also, many patients who self-treat use over-the-counter (OTC) medications that can actually prolong nasal symptoms and can even pose further, more serious health risks.

In this part of the book, we will explore the origins of the childhood and adult-onset allergy epidemics and the most effective ways to correct these problems.

WHAT CAUSES YOUR NASAL ALLERGY SYMPTOMS?

A question you may have often asked yourself is "Why do I have these allergy symptoms (and other people do not)?"

For some individuals, allergies start in childhood. These individuals tend to inherit genes from one or both of their parents that make them susceptible to allergies. If one parent has allergies, the child has about a 40% chance of developing allergies. If both parents have allergies, the child has about a 70% to 90% chance of developing allergies. If neither parent has allergies, the child has about a 10% to 15% chance of developing allergies.

Genes that make one susceptible to allergies are not the only cause of allergic reactions. To have a reaction to allergens (substances in the environment that cause allergic reactions), the individual must carry the allergic gene *and* be in an environment in which the allergen is present. It is these gene-environment interactions that lead to allergic reactions.

The prevalence of allergic diseases in the developed world is partially explained by the hygiene hypothesis, which holds that allergies are due to too much cleanliness. In developing countries, people's immune systems are busy fighting widespread bacteria, viruses, protozoa, and parasites and have no time to concentrate on relatively innocuous substances such as pollens and other allergens. In the developed world, where individuals are not exposed to bacteria as much, the immune system, looking for work, turns to fighting innocuous allergens.

ATOPIC DISEASES

There are three diseases that are grouped together and known collectively as atopic (externally caused) diseases: atopic dermatitis (eczema), asthma, and allergic rhinitis (ear, nose, and throat symptoms). There is a phenomenon called the "atopic march," which is a progression of different atopic diseases. This typically begins with eczema in children at around age four or five months—when they are first exposed to solid foods. By age three to four months (sometimes sooner and, at other times, later), some children develop upper respiratory infections, such as respiratory syncytial virus (RSV) infection and other viral infections that lead to their first experiences of wheezing, coughing, shortness of breath, and chest tightness (asthma symptoms). These symptoms, often called reactive airway disease (RAD) by many primary care physicians, may continue until adulthood or may subside early in childhood. By age two, most children with atopy have developed allergic rhinitis symptoms.

Sometimes, the sequence is eczema-allergic rhinitis-asthma, and sometimes it is asthma-eczema -allergic rhinitis. If children have allergic rhinitis that is untreated or poorly treated, they may develop asthma symptoms as a consequence; this is known as allergic rhinitis and its impact on asthma (ARIA). Chronic sinusitis (sinusitis is a consequence of poorly treated rhinitis). When mucus does not drain from the sinuses, it becomes a breeding ground for bacteria, which may lead to sinusitis. Acute sinusitis may be caused by a viral infection. If that is the case, it will not respond to antibiotics (which are effective against bacteria); it may simply have to run its course. It is, therefore, important to observe acute sinusitis for at least seven days prior to initiating antibiotic therapy.

Food allergy is a fourth condition that may go along with these three atopic diseases. Food allergies often contribute to the exacerbation of atopic dermatitis, asthma, or rhinitis. Elimination of the offending food in the child's diet may alleviate the atopic dermatitis, asthma, or rhinitis symptoms.

The most common foods that cause allergies in children are peanuts, soy, milk, eggs, wheat, barley, rye, corn, potatoes, garlic and tree nuts.

THE ROLE OF THE IMMUNE SYSTEM IN ALLERGIC REACTIONS

The body has the most effective military power known to man. It is known as the immune system. The immune system comprises cells with specialized functions just like Special Forces in the military.

Antigen-Presenting Cells

The process of allergy sensitization starts with exposure of an allergen to an antigen-presenting cell (APC), such as macrophages, monocytes, dendritic cells, or B-cells, which I refer to as the "patrolmen" of the immune system. These cells live in the skin, the tissues, and the blood. These patrolmen "present" an allergen to the Th0 cell, which I call the "white blood cell super-hero or superman" because the Th0 cell can turn into Th1 cells or Th2 cells, depending on the level of threat posed by the allergen. The roles these cells play in the immune response are explained in the following paragraphs.

Action of the Th0 Cell and B-Cells (Bomb-Making Cells)

The Th0 cell, upon encountering endotoxins, bacteria, viruses, and other antigenic organisms, becomes a Th1 cell, which is specialized to stimulate B-cells (bomb-making specialists) to produce antibodies (smart bombs called IgM and IgG) for fighting bacteria, viruses, protozoa, and other nonallergic infectious pathogens.

The Th0 cell becomes a Th2 cell upon exposure to allergens. In the presence of allergens, the patrolmen (antigen-presenting cells) send a signal to the superman (Th0 cell), which transforms into a Th2 cell that multiplies.

The Th2 cells produce IL-4 and IL-13. IL stands for interleukin. Interleukins are chemicals used by Th2 cells to communicate with other white blood cells. Think of these chemicals as Th2's cell phone or walkie-talkie).

In addition to IL-4 and IL-13, the Th2 cells also produce IL-5, which generates a late-phase allergic response by stimulating eosinophils (white blood cells that specialize in killing parasites). Eosinophils contribute to allergic inflammation by producing toxic proteins—major basic protein (MBP), and eosinophilic cationic protein (ECP)—both of which are involved in chronic inflammation and tissue destruction. Th2 cells also produce cytokines, which are involved in allergic reactions; IL-3, which stimulates basophils to multiply; and IL-6, which is involved in chronic inflammation.

The Role of the IgE Bomb

Th2 cells use IL-4 and IL-13 to alert B-cells that allergens are present. The B-cell is so versatile that it can produce IgE against any allergen (tree, grass, and weed pollens; dust mites; molds; animal dander; cockroach; etc.). These bomb-making cells then manufacture a powerful bomb called IgE (a smart bomb for fighting

allergens). The IgE bomb circulates in the blood, searching for docking sites—known as IgE receptors—before it can detonate.

The Role of Mast Cells (Misery Cell no. 1) and Basophils (Misery Cell no. 2) in the Allergic Response

These docking sites are located on two kinds of white blood cells—mast cells (which I call Misery Cell no. 1, because they make people feel so miserable) and basophils (which I call Misery Cell no. 2). Mast cells are found in the skin and tissues, and basophils circulate in the blood. Basophils can also enter tissues just like mast cells do. When the circulating IgE bombs find the docking sites on the mast cells and basophils, they attach to them. The IgE attached to the receptors on these cells cause them to be armed, dangerous, and ready to explode.

When an individual is exposed to an allergen he or she has previously encountered, the allergen enters through the nose, eventually makes its way to the blood system, finds the specific IgE bombs designed for that particular allergen—which are already on the surface of the Misery Cells—and attaches to them.

To summarize, the bombs attach to the Misery Cells and allergens attach to the bombs. A cross-linking of all the IgE-bomb-allergen complexes occurs on the surface of the Misery Cells, and that is a signal for these Misery Cells to explode and pour out their preformed, bioactive granules in a process called degranulation. One of the chemical granules released is histamine. Histamine is a nerve-ending irritant and causes itching. Histamine triggers the allergy symptoms of itchy eyes, itchy nose, itchy throat, sneezing, runny nose, and, sometimes, itchy skin.

Other Chemicals Released by the Misery Cells

If histamine were the only chemical released by mast cells and basophils, the allergy solution would be simple: use an antihistamine and the symptoms disappear. However, Misery Cells release many more chemicals when they degranulate (a complete list of chemicals released by these cells can be found in my book, *Allergy Detective: Allergic Rhinitis Treatment Secrets Your Doctor May Not Tell You).*

Two of these chemicals, leukotrienes and prostaglandins, tend to cause late-phase allergic reactions such as stuffy nose, postnasal drip, coughing, and, in asthmatics, constriction of the airways resulting in wheezing, shortness of breath, and chest tightness. In most patients, these symptoms tend to occur at night because leukotrienes become activated in the evening and are active throughout the night.

THE CORRELATION BETWEEN PESTICIDE USE AND THE ALLERGY EPIDEMIC IN THE U.S.

Allergic Rhinitis

Seasonal or perennial allergic rhinitis (inflammation of the nose) may be caused by pollens, hay, dust mites, molds, or animal dander, including that of cats, dogs, horses, cattle, rabbits, gerbils, guinea pigs, and hamsters. Allergic rhinitis may also be triggered by exposure to foods, such as milk, eggs, peanuts, wheat, soy, some spicy foods, shellfish, and fish, among others.

Perennial nonallergic rhinitis is caused by any number of chemical sensitivities—cigarette smoke, wood smoke, perfumes, scented candles, air fresheners, household cleaning agents (Pinesol, Lysol, Clorox, and ammonia), car exhaust, diesel fuel, gasoline, or freshly sprayed pesticides. Perennial nonallergic rhinitis may also be triggered by environmental exposure to cold air, humid air, dry air, or high barometric pressure. Nonallergic rhinitis may follow an infection (acute and chronic sinusitis, Churg-Strauss Syndrome, influenza, RSV infection, or common cold) or be caused by use of certain medications (overuse of decongestant nasal sprays such as Oxymetazoline products, floral-scented steroid nasal sprays, such as fluticasone propionate, blood pressure medications such as beta-blockers, or cocaine abuse). Nonallergic rhinitis may occur in patients with benign tumors in the nose. Examples are nasal polyps (a growth in the nasal passages), angiofibroma (a growth comprising fibrous tissue and blood vessels), fibrous dysplasia (abnormal growth of bony tissue), hemangioma (an abnormal growth of blood vessels), inverted papilloma (a one-sided, wart-like growth), or osteoma (tumor of the bony tissue of the nose), and structure abnormalities (deviated septum, enlarged adenoids, congestion due to pregnancy, foreign body in the nose). Remember that herbicides such as alachlor, acetochlor, metolachlor, and metolachlor-S cause nasal turbinate tumors in animals. Lastly, and significantly, perennial nonallergic rhinitis may be caused by hormonal changes resulting from prolonged exposure to endogenous estrogens, phytoestrogens, xenoestrogens, or other endocrine-disrupting chemicals.

Miscellaneous medical causes of nasal congestion include diabetes (types 1 and 2), gastroesophageal reflux disease (GERD), AIDS and other disorders of the immune system, oral or intravenous steroid treatment, hypothyroidism (underactive thyroid gland), Kartagener syndrome (a genetic disorder that impairs function of cilia, which are the hair-like structures that normally move mucus through the respiratory tract), stress, Wegener's Granulomatosis (a rare and serious autoimmune disorder), and

cystic fibrosis (a genetic disorder in which the mucus is very thick and builds up). A note of caution: Children who have frequent sinus infections should be tested for cystic fibrosis.

Asthma

Asthma is a chronic inflammatory disease of the airways characterized by symptoms of wheezing, coughing, chest tightness, and shortness of breath caused by reversible airflow obstruction and bronchospasm. Asthma is believed to be triggered by a combination of genetic and environmental factors. Diagnosis is usually based on the pattern of symptoms, response to therapy over time, and a measurement of breath volume. It is clinically classified according to the frequency of symptoms, forced expiratory volume in one second (FEV1), and peak expiratory flow rate. Asthma may also be classified as atopic (extrinsic—caused by environmental factors) or non-atopic (intrinsic) where atopy refers to a predisposition toward developing type 1 hypersensitivity (an IgE-mediated allergic reaction provoked by re-exposure to a specific type of antigen) reactions.

Acute asthma symptoms are usually treated with an inhaled, short-acting, beta-2 agonist (such as albuterol, xopenex, or salbutamol) and oral corticosteroids. In very severe cases, intravenous corticosteroids, magnesium sulfate, and hospitalization may be required. Symptoms can be prevented by avoiding triggers such as allergens and irritants and by the use of inhaled corticosteroids. Long-acting beta agonists (LABA) or anti-leukotriene drugs may be used in addition to inhaled corticosteroids if asthma symptoms remain uncontrolled. The occurrence of asthma has increased significantly since the 1970s. In 2011, approximately 280 million people globally were diagnosed with asthma, and it caused 250,000 deaths.

Atopic Dermatitis

Atopic dermatitis is a skin rash that starts in children around age three to five months when solids are first introduced in their diet. Some children who have food allergies may develop eczema early on during nursing. A child may react to his or her mother's milk if the mother consumes foods that do not agree with the child. Sometimes, environmental allergies couple with food allergies to cause atopic dermatitis. At other times, early exposure to antibiotics for ear infections or other respiratory infections cause eczema in the children.

The treatment for eczema consists of the following:

1. Antihistamines to prevent the itch that leads to atopic dermatitis lesions. Eczema by definition is the itch that rashes. If the itching is prevented, then the rash will resolve. Hydroxyzine 10 mg/5 ml, 1-2-3 tsp at bedtime works very well in children in the short run.
2. Moisturizers for the skin such as Aquaphor or Vanicream. Use the moisturizer right after baths when the skin is still moist.
3. Topical steroids, and the most effective is Elocon 0.1% ointment. The ointment works better than the cream and other steroids such as Triamcinolone or hydrocortisone or even Clobetasol (favorite among dermatologists) are not as effective as Elocon (Mometasone). Since Elocon 0.1% is a potent steroid, applying just a thin layer to the affected areas is enough to clear the eczema lesions. Elocon should not be applied to the face. Use Derma-Smoothe, a milder steroid, for the face.
4. Probiotics to replenish the good gut bacteria will help in the treatment of eczema. If you use probiotics, make sure to feed them with prebiotics.
5. Vitamin D3, 35 IU/LB is important for the treatment of eczema and for the treatment of atopic diseases in children and adults.
6. SLIT (sublingual immunotherapy) for environmental allergens and for foods works well for long-term control of the eczema.

Food Allergies

Statistics

Recently, we have seen an increase in cases of food allergies in children and adults. In 2007, approximately 3 million children under the age of 18 were reported to have a food or digestive allergy in the previous 12 months. The prevalence of food allergies among children under the age of 18 increased 18% percent from 1997 to 2007. Kids who have a food allergy are two to four times more likely to have conditions such as asthma and other allergies.

Food allergies affect about 6% of children under the age of three.

Six and a half million Americans (or 2.3% of the general population) are allergic to seafood, and more than 3 million people in the U.S. report being allergic to peanuts, tree nuts, or both. More than 3% of adults have one or more food allergies.

Milk allergy is the most common childhood food allergy, affecting 2.5% of children younger than age 3. Approximately 80% of children outgrow their allergy to milk by age 16.

Egg allergy is the second most common food allergy in children, affecting 1.5%-3.2% of children. Approximately 68% of children outgrow their allergy to eggs by age 16.

Peanut allergy affects 1.2% of children. Approximately 20% of children outgrow it by age 6. Peanut allergy rates doubled in children from 1997-2002. The allergy is associated only with the consumption of peanuts; skin contact and inhalation exposure to peanut butter are unlikely to cause systemic allergic reactions or anaphylaxis.

Tree nut allergy (almonds, walnuts, etc.) affects 1.2% of the population. Approximately 9% of children outgrow tree nut allergy by age 6.

Most patients who are allergic to peanuts can safely eat other legumes such as soy or beans (95%), but they can have concurrent allergy to tree nuts such as walnuts or pecans (25% to 50%).

Anaphylaxis occurs in 20% of allergic reactions to peanuts and tree nuts.

It is estimated that the number of cases of anaphylaxis from foods in the U.S. increased from 21,000 per year in 1999 to 51,000 per year in 2008, based on long-term population studies of anaphylaxis from the Mayo Clinic in Minnesota.

From 2003 to 2006, food allergies resulted in approximately 317,000 visits to hospital emergency departments, outpatient clinics, and physicians' offices, according to data from multiple U.S. national surveys collected by the National Center for Health Statistics. Hospital admissions related to food allergies increased from 2,600 per year in 1998-2000 to 9,500 per year in 2004-2006, according to this same source.

It is estimated that food allergies cause approximately 150 to 200 fatalities per year, based on data from a five-year study of anaphylaxis in Minnesota conducted by the Mayo Clinic. Fatal food anaphylaxis is most often caused by peanuts (50%-62%) and tree nuts (15%-30%). Risk factors for fatal anaphylaxis include failure or delay in administration of epinephrine, history of asthma, and age range of 12 to 19 years.

The following food allergy trend diagrams show that 1996-1997 was a turning point (indicated by the blue triangle) in food allergy rates. What happened in 1996 that started an upward trend in food reactions?

In agricultural production, the major change in 1996 was the widespread usage of glyphosate (the major ingredient in Roundup) on genetically modified plants. Glyphosate is estrogenic; acts like an antibiotic to kill good gut bacteria; is a mineral chelator; and causes tumors in animals, according to several European studies.

For more information about glyphosate, visit GreenMedInfo.com, which follows these studies and reports them as they appear in medical journals. Other good sources are the Environmental Working Group at EWG.org and Dr. Mercola's various interviews with experts and journal articles at www.mercola.com.

Figure 14. The Rise in the Number of Cases of Anaphylactic Shock, 1993 to 2013

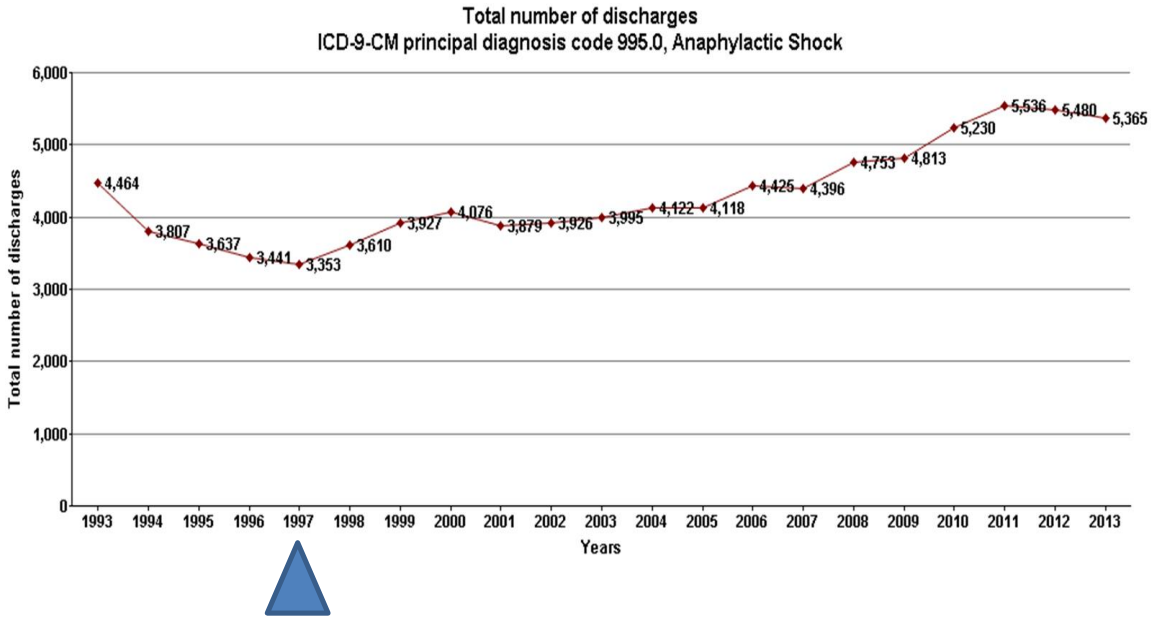


Figure 15. The Rise in the Number of Cases of Angioedema, 1993 to 2013

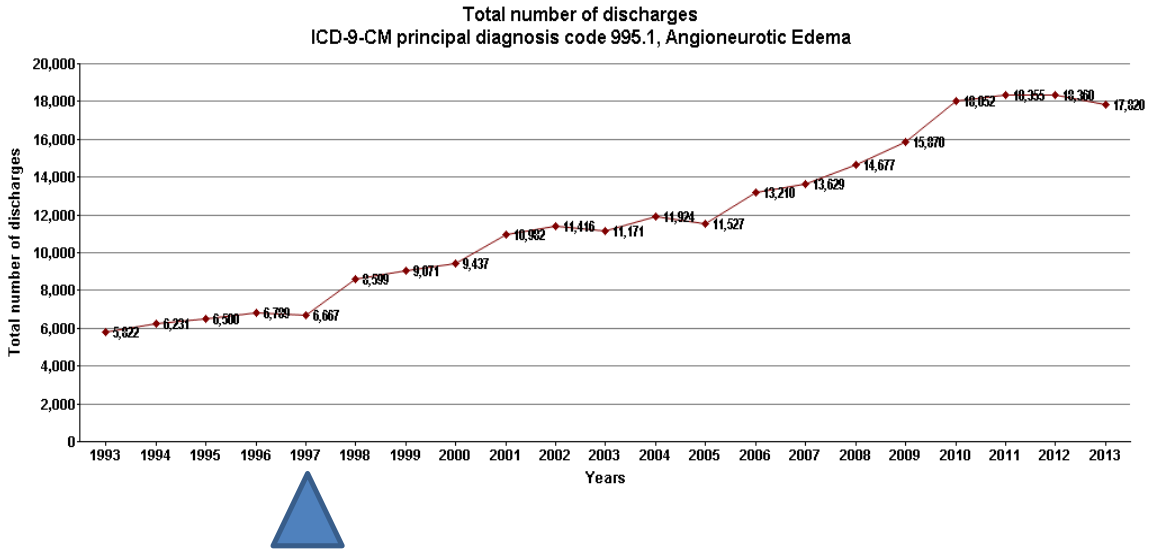
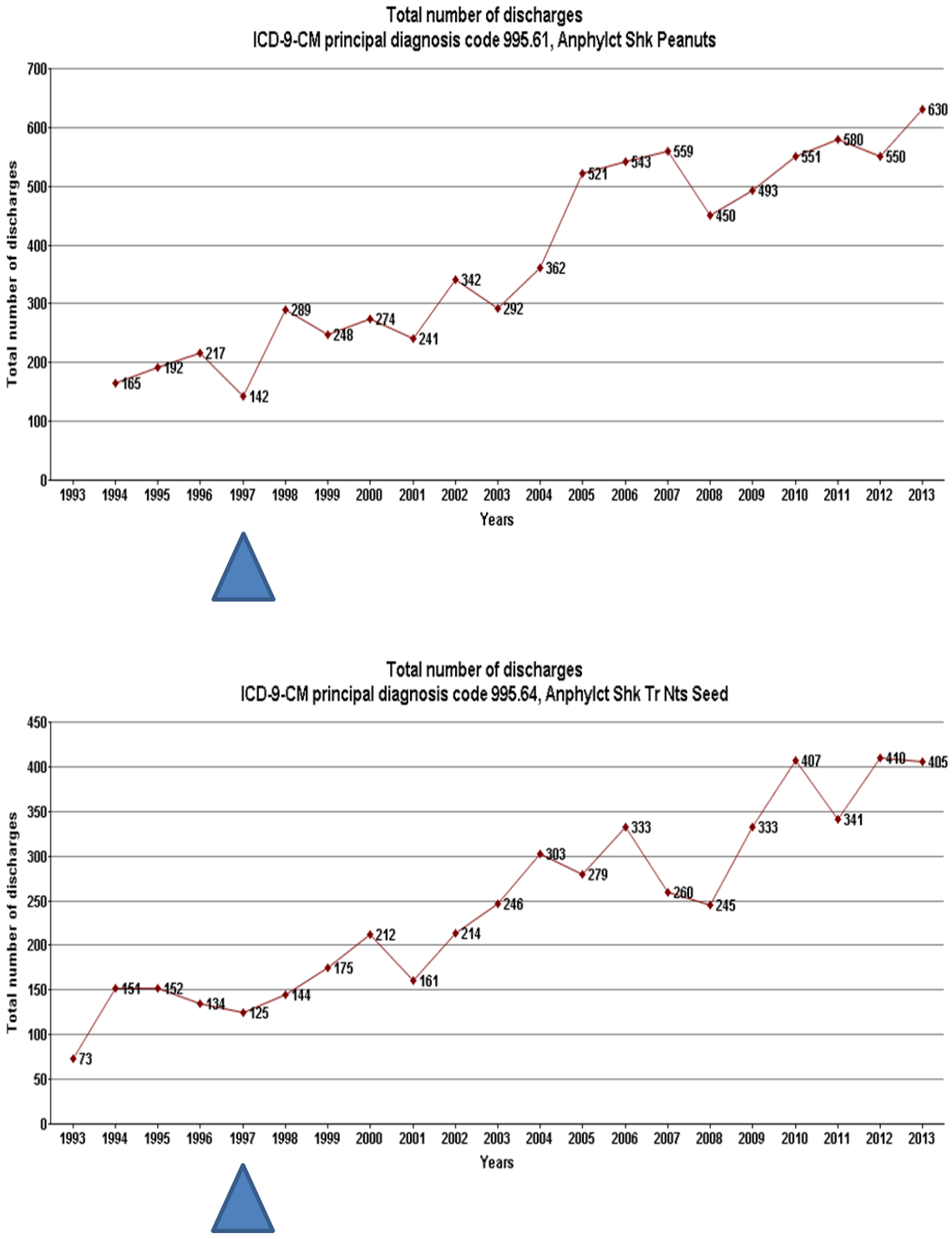
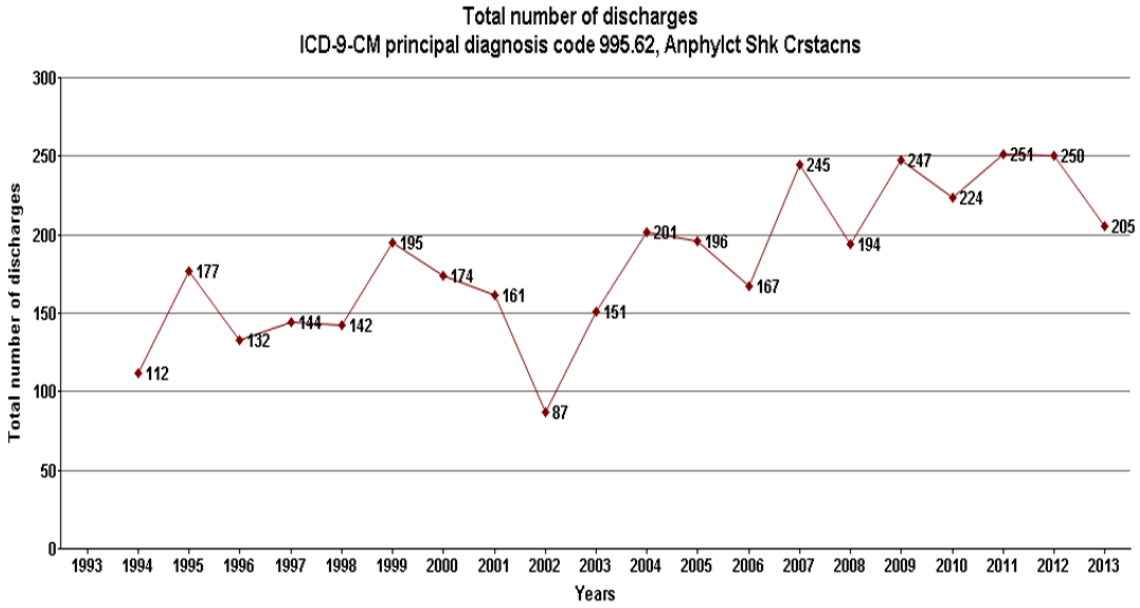


Figure 16. The Rise in the Number of Cases of Anaphylactic Shock Caused by Peanuts and Tree Nuts, 1993 to 2013



Figures 17 and 18. Rates of Anaphylactic Shock Caused by Crustaceans and Fish, 1993 to 2013



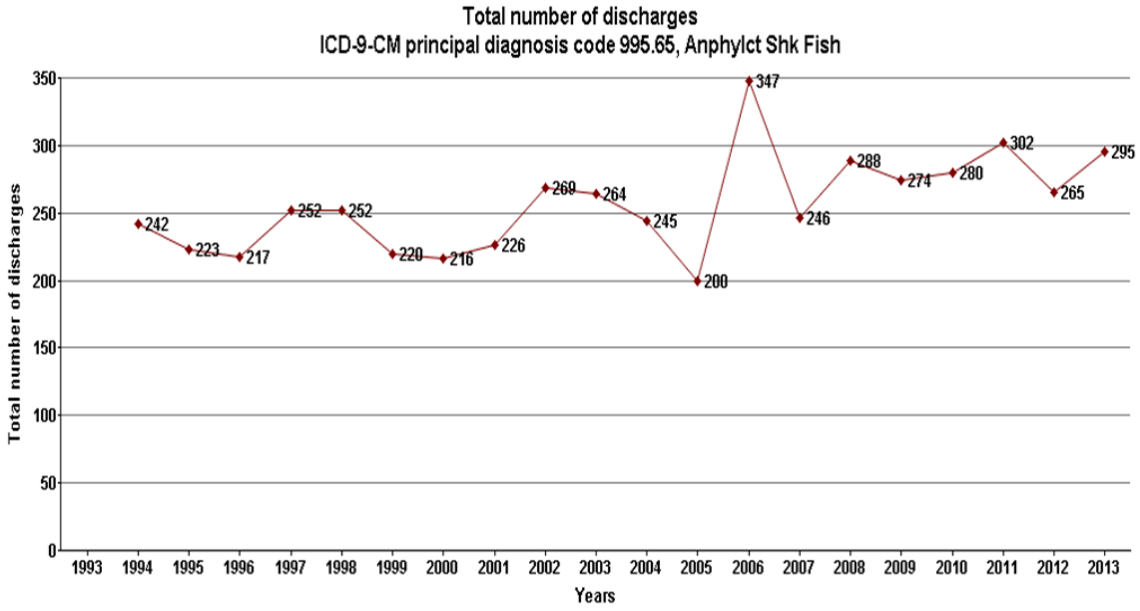
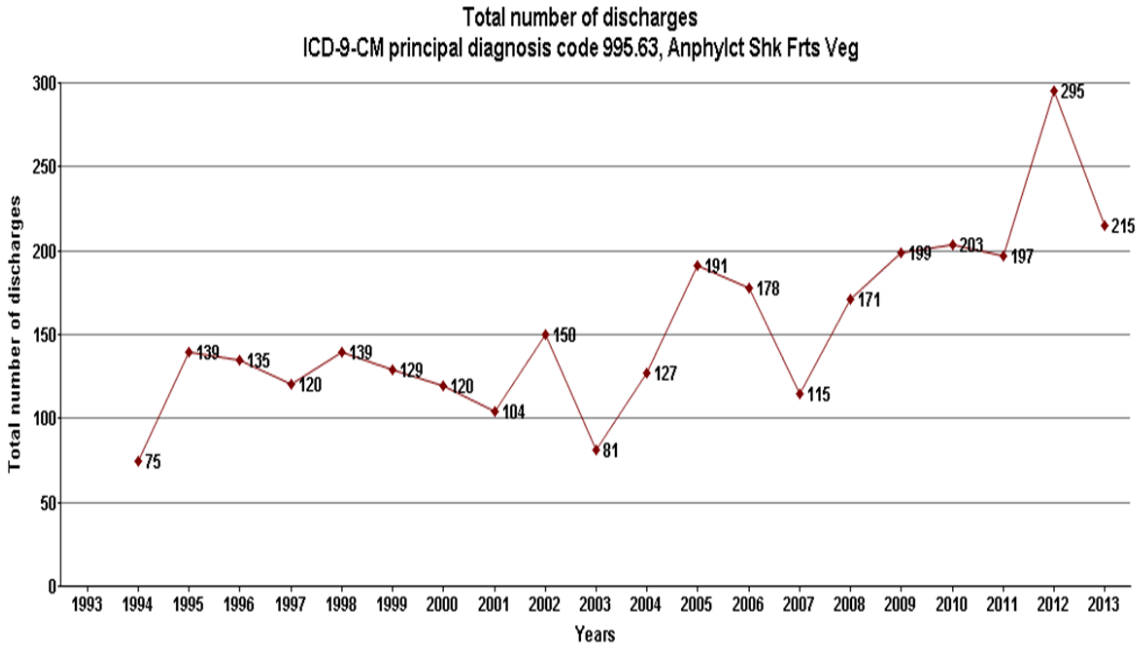
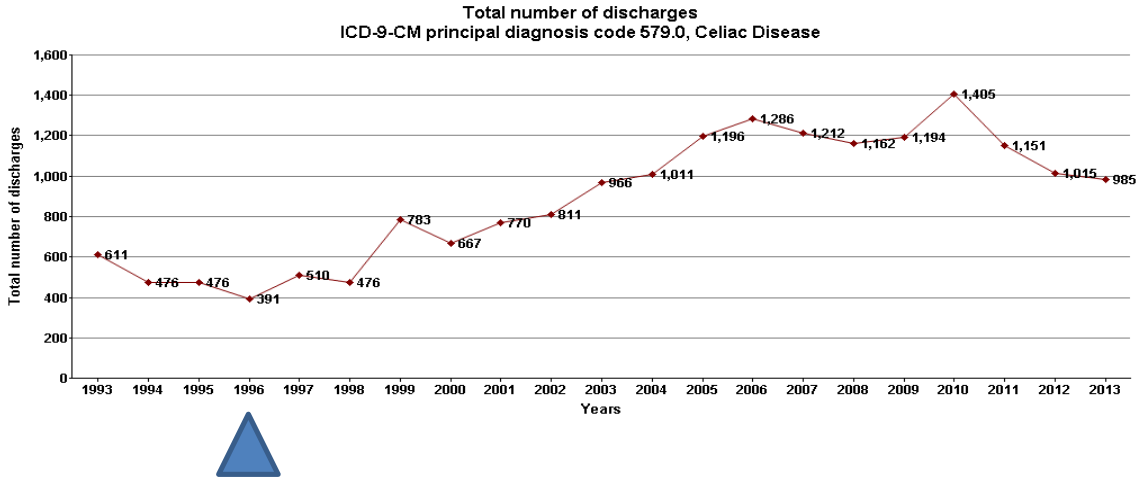


Figure 19. The Rise in the Number of Cases of Anaphylactic Shock Caused by Fruits and Vegetables, 1993 to 2013



Notice that allergic reactions to crustaceans and fish do not follow the same patterns as the reactions to agricultural products. There was no turning point in 1996-1997, because seafood was not exposed (or not as heavily exposed) to pesticides, unless farm-raised.

Figure 20. Increasing Rates of Celiac Disease, 1993 to 2013



In 2013 women had more hospital discharges and more ED visits for celiac disease than men as shown below

Outcomes for ICD-9-CM principal diagnosis code 579.0 Celiac Disease (2013)

		Total number of discharges	LOS (length of stay), days (median)	Charges, $ (median)	Costs, $ (median)	Aggregate costs
All discharges		985 (100.00%)	4.0	27,882	7,747	11,007,183
Age group	1-17	125 (12.69%)	2.0	22,605	4,898	1,386,244
	18-44	265 (26.90%)	4.0	26,293	6,320	2,319,200
	45-64	275 (27.92%)	4.0	29,341	8,621	3,926,059
	65-84	285 (28.93%)	5.0	33,891	8,915	3,160,186
	85+	*	*	*	*	*
Sex	Male	315 (31.98%)	4.0	29,341	8,274	3,342,287
	Female	670 (68.02%)	4.0	27,427	7,597	7,664,897
Median income for zipcode	Low	190 (19.29%)	4.0	26,907	6,320	1,867,203
	Not low	770 (78.17%)	4.0	27,453	7,811	8,585,432
	Missing	*	*	*	*	*
Region	Northeast	190 (19.29%)	5.0	34,176	9,553	2,150,031
	Midwest	255 (25.89%)	4.0	24,946	6,616	3,409,637
	South	330 (33.50%)	4.0	22,882	7,005	2,809,691
	West	210 (21.32%)	4.0	31,049	8,849	2,637,824

ICD-9-CM first-listed diagnosis code 579.0 Celiac Disease (2013) ED visits

		All ED visits	ED visits with admission to the same hospital	Discharged from the ED	Standard errors		
					All ED Visits	ED visits with admission to the same hospital	Discharged from the ED
All visits		1,499 (100.00%)	834 (100.00%)	665 (100.00%)	103	69	73
Age (mean)		44.96	52.13	35.98	1.64	1.64	1.64
Age group	<1	*	*		*	*	
	1-17	195 (12.99%)	*	92 (47.28%)	43	*	24 (9.28%)
	18-44	585 (39.04%)	203 (34.72%)	382 (65.28%)	64	34 (4.26%)	48 (4.26%)
	45-64	356 (23.72%)	226 (63.44%)	130 (36.56%)	49	34 (6.72%)	33 (6.72%)
	65-84	278 (18.58%)	224 (80.28%)	*	39	35 (5.40%)	*
	85+	80 (5.35%)	74 (92.31%)	*	21	20 (7.36%)	*
Sex	Male	426 (28.39%)	226 (53.09%)	200 (46.91%)	47	34 (5.39%)	32 (5.39%)
	Female	1,073 (71.61%)	608 (56.63%)	466 (43.37%)	91	57 (3.83%)	64 (3.83%)
Median income for zipcode	Low	396 (26.44%)	195 (49.18%)	201 (50.82%)	55	36 (5.56%)	35 (5.56%)
	Not low	1,058 (70.61%)	615 (58.10%)	443 (41.90%)	86	59 (4.07%)	62 (4.07%)
	Missing	*	*	*	*	*	*
Region	Northeast	319 (21.30%)	194 (60.66%)	126 (39.34%)	48	35 (8.14%)	35 (8.14%)
	Midwest	365 (24.33%)	169 (46.35%)	196 (53.65%)	55	29 (6.72%)	44 (6.72%)
	South	496 (33.06%)	276 (55.72%)	219 (44.28%)	57	42 (5.23%)	35 (5.23%)
	West	319 (21.30%)	195 (61.03%)	124 (38.97%)	45	29 (6.13%)	29 (6.13%)

The Pathophysiology of Food Allergies

Food reactions may be IgE-mediated, IgG-mediated, non-IgE-mediated or non-IgG-mediated (as occurs with reactions to pesticides), estrogen-mediated Xenoestrogens-mediated, or acetylcholine-mediated.

When food was produced naturally, food allergies were rare. Large-scale, commercial food production—in which chemicals are used as pesticides, preservatives, and to serve other functions—has led to increased reaction to foods. Chemicals bound to foods yield chemopeptides (toxpetides or xenopeptides), and estrogens bound to foods yield estropeptides. Chemical contamination of foods causes food molecules to be designated by the body as allergens, which are attacked by antigen-presenting cells.

The allergic process for food is exactly the same as that described earlier for respiratory allergies, beginning with the actions of antigen-presenting cells and ending with the effects on the body of the Misery Cells. Chemopeptides and estropeptides (antigenic peptides) are presented to Th0 cells. Th0 cells differentiate into Th1 (by action of IL-2, IFN-gamma) and Th2 (by action of IL-4, IL-13), which stimulate B-cells to convert into plasma cells that produce antibodies (IgM, IgG, IgA, IgE, and minute amounts of IgG4 insufficient for protection) against these new antigenic peptides. Antigen-presenting cells are stimulated to produce IL-1, IL-6 (which stimulates production of C-Reactive Protein—CRP—by hepatocytes), IL-12 (which activates NK cells to produce IFN-gamma), and TNF-alpha.

Estrogenic chemicals (organochlorines, glyphosate, bisphenol A [BPA], etc.) and endogenous estrogens bind directly to their receptors on mast cells and basophils to cause degranulation (allergic reactions). Intrinsic estrogens coupled with extrinsic estrogens cause more symptoms in women because women, in general, have more intrinsic estrogens than men.

As mentioned in the section on pesticides, organophosphate and carbamate insecticides cause accumulation of acetylcholine. When acetylcholine attaches to its receptors on mast cells and basophils, this leads to degranulation. Foods that are routinely contaminated by pesticides (peanuts, milk, soy, wheat, eggs, tree nuts, corn, etc.), therefore, tend to cause allergic reactions.

The bottom line: When food becomes antigenic due to contamination by pesticides, the allergic response is exactly the same as that which occurs in environmental allergies.

CHEMICALS USED IN FOOD PRODUCTION IN THE U.S.

The table below shows some of the pesticides used in food production and their effect on humans.

Table 3. Chemical Class of Pesticides, Mode of Action, and Effect on Humans

Year	Pesticide	Crop	PESTICIDES CLASS	PESTICIDES MODE OF ACTION	EFFECT ON HUMANS
2009	ATRAZINE	Spring wheat	TRIAZINE	AROMATASE BOOSTER	Testosterone/estrogen
2009	BROMOXYNIL	Spring wheat	HYDROXYBENZONITRILE		
2009	CARBOFURAN	Spring wheat	N-METHYL-CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	EPTC	Spring wheat	THIOCARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	GLYPHOSATE	Spring wheat	PHOSPHONOGLYCINE	EDC	Estrogenic/reproductive
2009	LINURON	Spring wheat	SUBSTITUTED UREA		
2009	METHOMYL	Spring wheat	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METHYL PARATHION	Spring wheat	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METOLACHLOR-S	Spring wheat	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	METRIBUZIN	Spring wheat	SELECTIVE TRIAZINE	EDC	Testosterone/estrogen
2009	PROPANIL	Spring wheat	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PROPARGITE	Spring wheat	UNCLASSIFIED	PAN BAD ACTOR	
2009	PROPICONAZOLE	Spring wheat	TRIAZOLE	PAN BAD ACTOR	
2009	TRIALLATE	Spring wheat	THIOCARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	TRIFLURALIN	Spring wheat	2,6-DINITROANILINE		

The rise in celiac disease and gluten-sensitivity may be directly related to the pesticides used in wheat production. Table 4 lists the many endocrine-disrupting chemicals used on wheat.

Table 4. Pesticides Found in Wheat

Chemicals in Wheat

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	ATRAZINE	Spring wheat	65,192,074	21,343	<1	2009	ATRAZINE	Spring wheat	67,005,515	66,707	<1
2009	BROMOXYNIL	Spring wheat	2,003,964	1,261,637	62.96	2009	BROMOXYNIL	Spring wheat	2,586,820	1,277,750	49.39
2008	CARBOFURAN	Spring wheat	302,673	750	<1	2008	CARBOFURAN	Spring wheat	1,123,405	2,393	<1
2009	EPTC	Spring wheat	1,829,047	569	<1	2009	EPTC	Spring wheat	3,500,796	569	<1
2009	GLYPHOSATE	Spring wheat	219,724,312	4,148,299	1.89	2009	GLYPHOSATE	Spring wheat	222,444,180	4,182,043	1.88
2008	LINURON	Spring wheat	212,786	27,982	13.15	2008	LINURON	Spring wheat	349,078	89,272	25.57
2009	METHOMYL	Spring wheat	941,971	56	<1	2009	METHOMYL	Spring wheat	1,477,625	56	<1
2009	METHYL PARATHION	Spring wheat	312,631	57,078	18.26	2009	METHYL PARATHION	Spring wheat	975,383	198,751	20.38
2009	METOLACHLOR-S	Spring wheat	29,215,535	122	<1	2009	METOLACHLOR-S	Spring wheat	33,534,830	122	<1
2008	METRIBUZIN	Spring wheat	1,125,519	69	<1	2008	METRIBUZIN	Spring wheat	2,055,536	69	<1
2009	PROPANIL	Spring wheat	4,847,819	192	<1	2009	PROPANIL	Spring wheat	4,871,388	192	<1
2008	PROPARGITE	Spring wheat	1,036,080	133	<1	2008	PROPARGITE	Spring wheat	2,755,932	133	<1
2009	PROPICONAZOLE	Spring wheat	1,125,968	246,237	21.87	2009	PROPICONAZOLE	Spring wheat	1,552,077	251,737	16.22
2009	TRIALLATE	Spring wheat	188,294	19,869	10.55	2009	TRIALLATE	Spring wheat	625,113	118,312	18.93
2009	TRIFLURALIN	Spring wheat	5,201,075	23,591	<1	2009	TRIFLURALIN	Spring wheat	8,222,225	208,184	2.53

There are even more chemicals used in producing wheat that are not included in the table above. Fusarium is a mold that grows in grains (corn, barley, rye, wheat, rice) and which produces mycoestrogens such as Zeralenone. Fungicides to control fusarium include mancozeb (which is a thyroid disruptor) and chlorothanil-daconil (which causes dermatitis and kidney damage).

Grain-related diseases and disorders that are on the rise include the gluten-sensitivity epidemic and celiac disease. Contamination of grains by endocrine disruptors may also contribute to obesity.

WHY IS PEANUT ALLERGY BECOMING MORE COMMON?

Here are few facts about peanuts:

- Peanuts are grown in the South.
- Peanut crops are rotated with cotton production.
- Peanuts require more than 40 pesticides (many of them are organophosphate and carbamates insecticides and now glyphosate is added).
- Cotton requires more than 100 pesticides.
- There is an increased pesticide load in the soil over time (POPs and new pesticides).
- As the pesticide load is increasing, so we see an increase in people with peanut allergies in the U.S. over time.
- Refer to the obesity and pesticides maps in Part II of this book and recall the findings in the southern states.
- Allergic reactions to peanuts have dramatically increased in the U.S. in recent years, especially since the mid-1990s.
- I suspect that the growing chemical load in the soil is driving this peanut allergy epidemic.

Pesticides Used in Peanut Production

Glyphosate and other estrogenic chemicals, as well as organophosphates and carbamates, are used in both cotton and peanut production. The latter two groups of chemicals cause an increase in acetylcholine. As noted in the previous section on nasal allergy pathophysiology, estrogens have receptors on mast cells and basophils that directly affect degranulation of these cells. These estrogens also enhance the effect of IgE. It is therefore possible that the increased estrogen load in our environment may be the reason for the growing number of people with peanut allergies.

Acetylcholine also has receptors on mast cells and basophils and can cause degranulation of these cells, resulting in allergic reactions. Acetylcholine also causes insulin release that leads to mast cell and basophil proliferation and, hence, an

increase in allergic reactions. The synergistic effect of these chemicals may explain not only the growing allergy epidemic, but also the growing obesity epidemic and increase in obesity comorbidities seen worldwide.

It is noteworthy that the two leading peanut-producing countries in the world do not have the same peanut allergy epidemic as is found in the U.S. and countries that consume American peanuts. Both China and India are leading peanut-producing and consuming countries, but there are no data indicating that children in these countries suffer the same allergic reactions to peanuts as do children in the U.S. and European countries that consume peanuts produced in America.

The reason for this discrepancy may likely be the production processes used. There is no crop rotation between peanut and cotton, for example, in China and India, and, therefore, the amount of pesticides used is less compared to that in the U.S. African countries also do not use many pesticides in their peanut production, and, therefore, there are fewer cases of allergic reactions to peanuts in Africa.

THE “DIRTY DOZEN” AND “CLEAN FIFTEEN” FOODS

There are some fruits and vegetables that are more allergy-producing (and dangerous) to consume than others, due to pesticide contamination, and these foods are commonly referred to as the “Dirty Dozen” by the Environmental Working Group. If possible, you should always purchase organic foods in this list:

1. Apples
2. Peaches
3. Nectarines
4. Strawberries
5. Grapes
6. Celery
7. Spinach
8. Sweet bell peppers
9. Cucumbers
10. Cherry tomatoes
11. Snap peas (imported)
12. Potatoes

Additionally, hot peppers, kale, and collard greens are frequently found to be contaminated with insecticides that are toxic to the human nervous system.

Take this list with a grain of salt because it is not exhaustive. It does not include peanuts, which are one of the most contaminated legumes in America today.

The EWG also issues a "Clean Fifteen" list of fruits and vegetables each year, which have the least amount of pesticides. In 2015 the following foods were on the list:

1. Avocados
2. Sweet corn (organic)
3. Pineapples
4. Cabbage
5. Sweet peas (frozen)
6. Onions
7. Asparagus
8. Mangoes
9. Papayas
10. Kiwi
11. Eggplant
12. Grapefruit
13. Cantaloupe
14. Cauliflower
15. Sweet potatoes

Key findings in 2015 from the EWG follow:

- Avocados are the cleanest of all produce: only 1% of avocado samples showed any detectable pesticides.
- Some 89% of pineapples, 82% of kiwi, 80% of papayas, 88% of mango, and 61% of cantaloupe had no residues.
- No single fruit sample from the Clean 15 tested positive for more than four types of pesticides.

PART IIC: THE RELATIONSHIP AMONG PESTICIDES, OBESITY, AND ALLERGIES

THE RELATIONSHIP BETWEEN FARMING PRACTICES AND OBESITY IN THE U.S.

The following information was derived from CDC obesity maps, U.S.G.S. pesticide/herbicide usage maps, and the Healthcare Cost and Utilization Project (HCUP) database. Analysis from 2002 and 2011 shows the highest annual use of active ingredients known to be thyroid-, estrogen-, and androgen-disrupting chemicals occurs in the South (Mississippi embayment states) and Midwest. Also, the incidence of hypothyroid disease is more prevalent (primarily in females) in these areas. It is reasonable to postulate that endocrine-disrupting chemicals should be scrutinized and regarded with the greatest suspicion for contributing to the American obesity epidemic.

It seems apparent that there is a connection among pesticides/herbicides, common household chemicals, and Hormone Imbalance Syndrome (HIS). There also seems to be a link between HIS and the growing food allergy and environmental allergy epidemics.

MEDICAL GEOGRAPHY: AN APPROACH TO UNDERSTANDING THE OBESITY EPIDEMIC

The common objection raised to using medical geography to correlate disease to an environmental factor is that correlation does not necessarily mean causation. Medical geography has been useful in the past, however, and continues to be useful in explaining important environmental disease phenomena.

John Snow (1813-1858) is regarded as the father of medical geography. He was born into a laborer's family on March 15, 1813 in York, Britain, and, at 14, was apprenticed to a surgeon. At age 23, he moved to London to start his formal medical education. Snow became a member of the Royal College of Surgeons in 1838, graduated from the University of London in 1844, and was admitted to the Royal College of Physicians in 1850.

At the time, it was assumed that cholera was airborne; however, Snow did not accept this “miasma” (bad air) theory, arguing that, in fact, cholera entered the body through the mouth. He published his ideas in 1849 in an essay titled, “On the Mode of Communication of Cholera.” A few years later, Snow was able to prove his theory in dramatic circumstances. In August 1854, a cholera outbreak occurred in the Soho

area of London. After careful investigation, including plotting cases of cholera on a map of the area, Snow was able to identify a water pump on Broad (now Broadwick) Street as the source of the disease. He had the handle of the pump removed, and cases of cholera immediately began to diminish; however, Snow's “germ” theory of disease was not widely accepted until the 1860s.

I will use medical geography methodology to demonstrate the relationship between pesticide use and the growing obesity and allergy epidemics in the U.S.

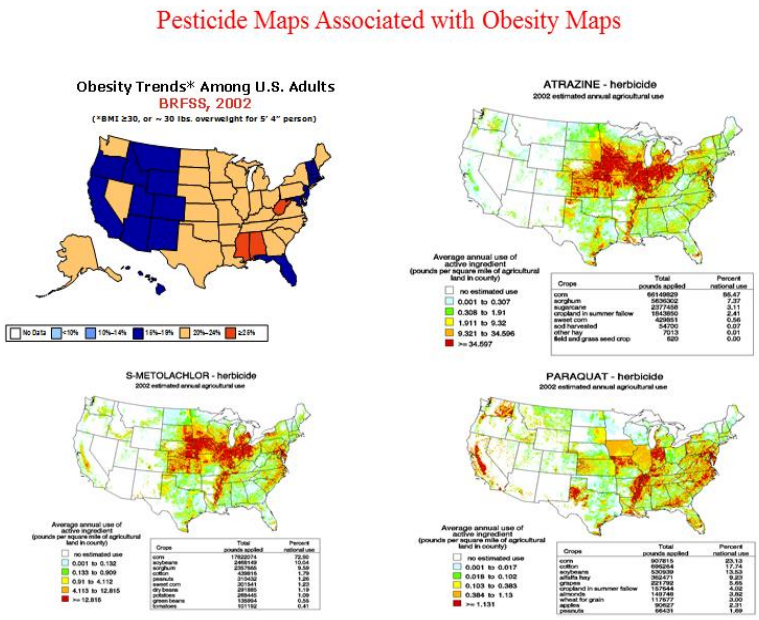
Why Is Obesity More Prevalent in the Mississippi Embayment States?

One of the primary crops grown in the Mississippi embayment states is cotton. Cotton requires three times more pesticides and herbicides than any other crop. The Mississippi River drains most of the upper Midwest and Midwest rivers that carry in them pesticides and herbicides sprayed on farmlands in these regions. The South is one of the oldest agriculture regions in the U.S., and designated agricultural land has received increasing amounts of pesticides load over the years. Some pesticides, such as organochlorines, organophosphates, and carbamates, newer ones, such as atrazine, glyphosate, and many other toxic pesticides are persistent in the environment, and may have cumulative and synergistic effects. Agricultural irrigation runoff represents a real danger for potable water. In addition, the Mississippi River floods frequently and carries pesticide toxins to many parts of the embayment states.

Bottom line: The more cotton production in the U.S., the more pollution, the more obesity and its comorbidities, and the more allergic reactions we see.

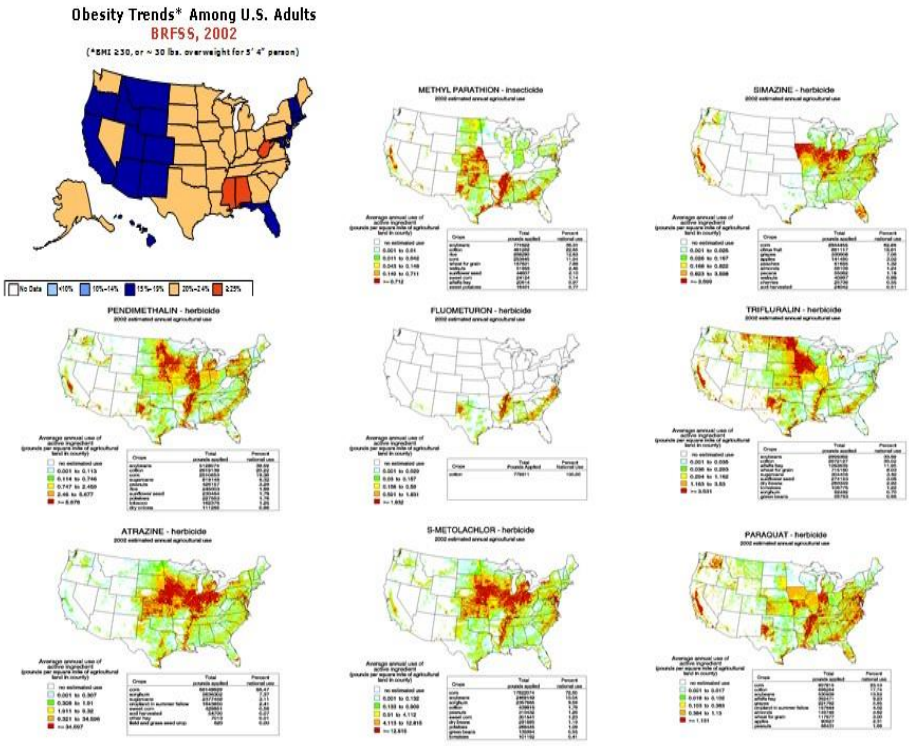
Figure 21 shows obesity rates and pesticide usage maps by state in the U.S.

Figure 21. A Comparison of Obesity and Pesticide Maps, U.S.



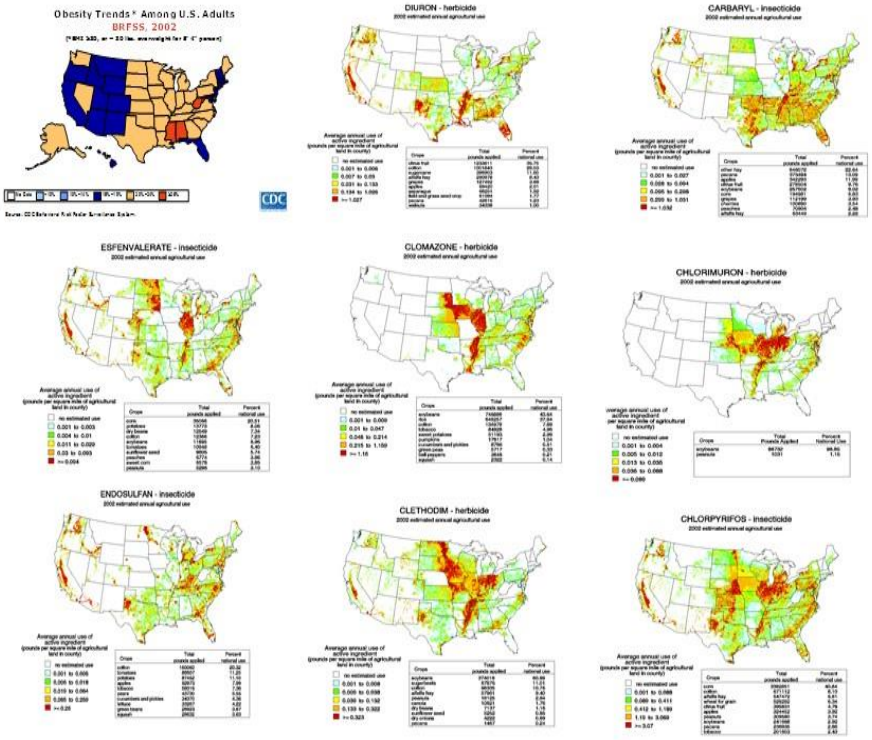
This shows the obesity map by the CDC and selected herbicide maps by USGS (US Geological Survey). Notice that the 2002 herbicide spray areas depicted in the USGS maps seem to coincide with the CDC 2002 obesity map area of high obesity rates. The areas of intense pesticide spray are depicted in red followed by yellow for the moderate spray and green mild spray, The right half of the US that receives the heaviest pesticides spray also has the highest obesity rates. You can even follow the pesticide trail to Mississippi and Louisiana, two of the most polluted states in 2002.

Pesticide Maps Associated with Obesity Maps



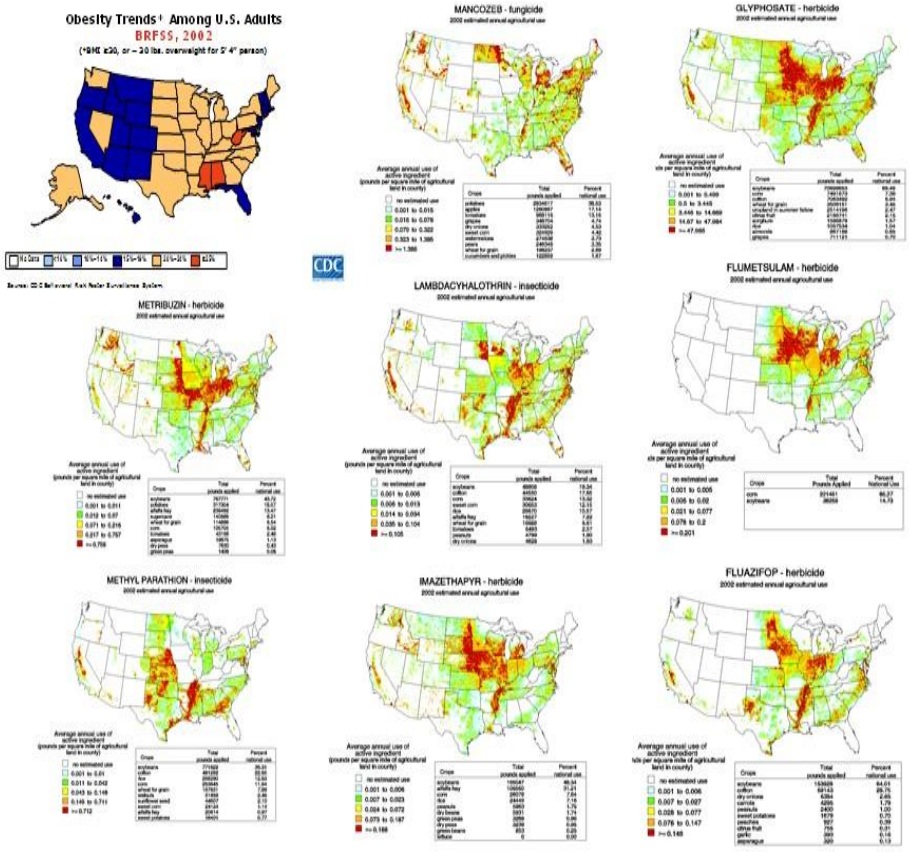
More 2002 herbicide spray areas compared with 2002 obesity rates in the same areas

Pesticide Maps Associated with Obesity Maps



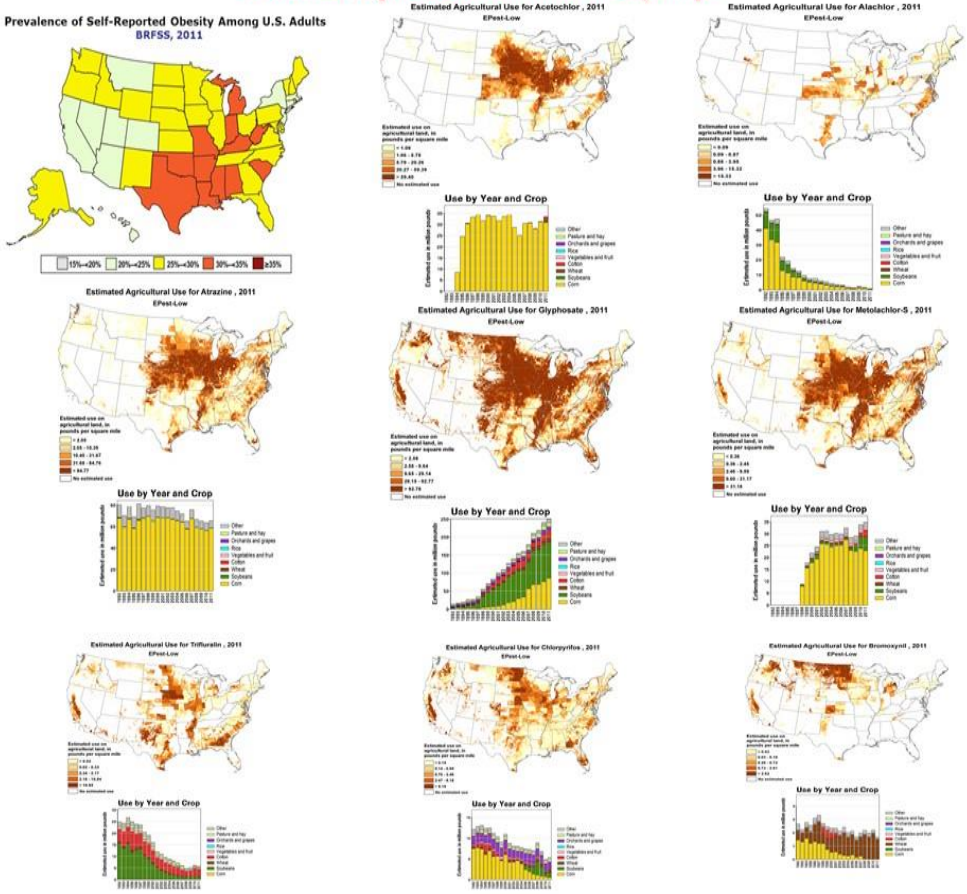
More 2002 herbicide spray areas compared with 2002 obesity rates in the same areas

Pesticide Maps Associated with Obesity Maps



More 2002 herbicide spray areas compared with 2002 obesity rates in the same areas

Pesticide Maps Associated with Obesity Maps



2011 Pesticides spray areas compared with 2011 obesity rates in the same areas

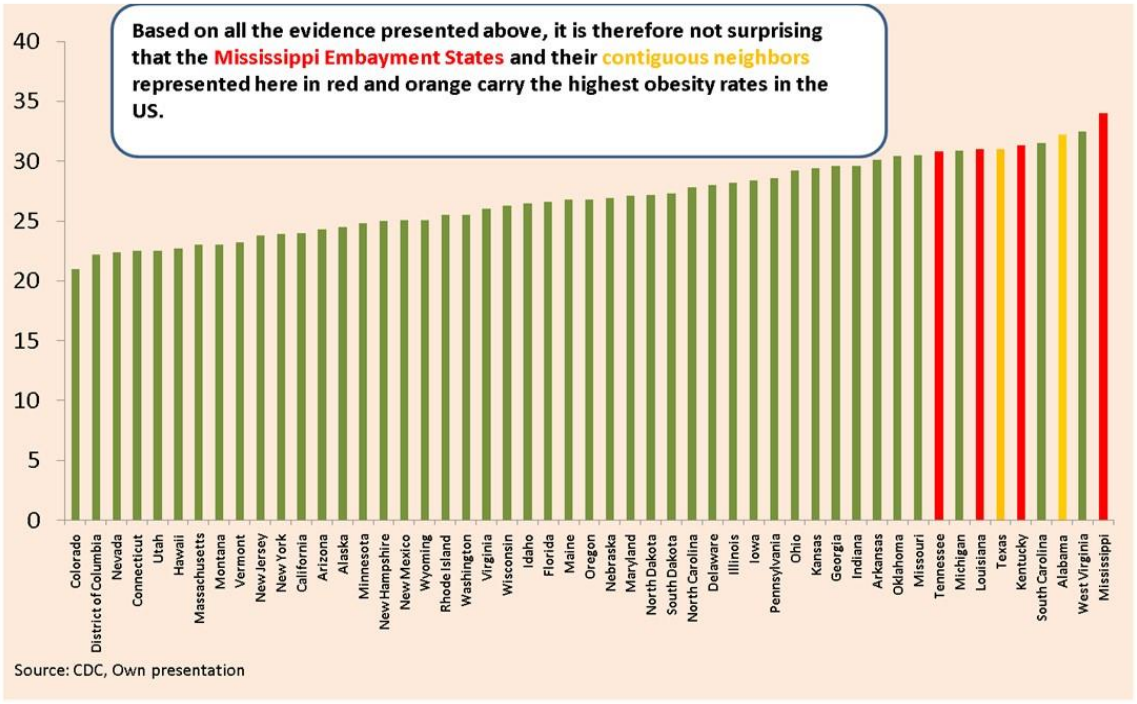
Table 5 and the chart following it show obesity rates by state, with the Mississippi River embayment states noted in red.

Table 5. Obesity Rates by State, 2009 (Mississippi Embayment States in Red).

State	%	State	%	State	%	State	%
Alabama	31.0	Illinois	26.5	Montana	23.2	Rhode Island	24.6
Alaska	24.8	Indiana	29.5	Nebraska	27.2	South Carolina	29.4
Arizona	25.5	Iowa	27.9	Nevada	25.8	South Dakota	29.6
Arkansas	30.5	Kansas	28.1	New Hampshire	25.7	Tennessee	32.3
California	24.8	Kentucky	31.5	New Jersey	23.3	Texas	28.7
Colorado	18.6	Louisiana	33.0	New Mexico	25.1	Utah	23.5
Connecticut	20.6	Maine	25.8	New York	24.2	Vermont	22.8
Delaware	27.0	Maryland	26.2	North Carolina	29.3	Virginia	25.0
Washington DC	19.7	Massachusetts	21.4	North Dakota	27.9	Washington	26.4
Florida	25.2	Michigan	29.6	Ohio	28.8	West Virginia	31.1
Georgia	27.2	Minnesota	24.6	Oklahoma	31.4	Wisconsin	28.7
Hawaii	22.3	Mississippi	34.4	Oregon	23.0	Wyoming	24.6
Idaho	24.5	Missouri	30.0	Pennsylvania	27.4		

Based on all the evidence presented above, it is therefore not surprising that the Mississippi Embayment States and their contiguous neighbors represented here in red and orange carry the highest obesity rates in the US.

2010 State Obesity Rate



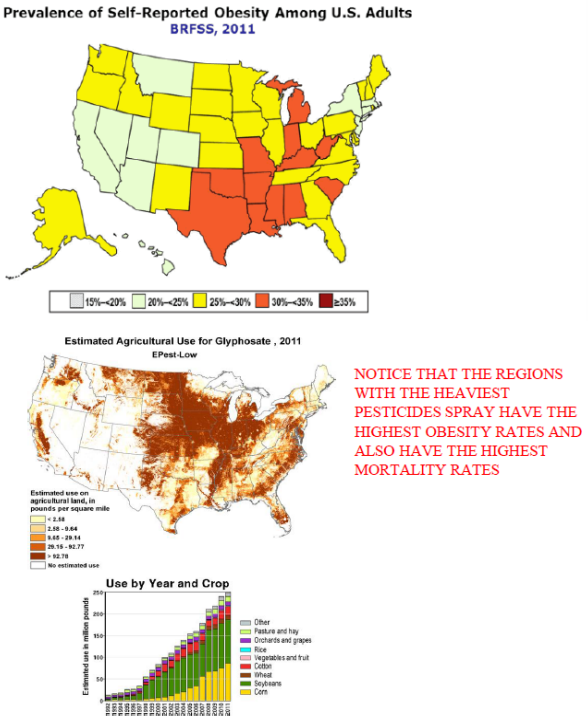
Source: CDC, Own presentation

OBESITY MAPS (2011-2012), MORTALITY MAP, AND PESTICIDES SPRAY MAP COMPARED

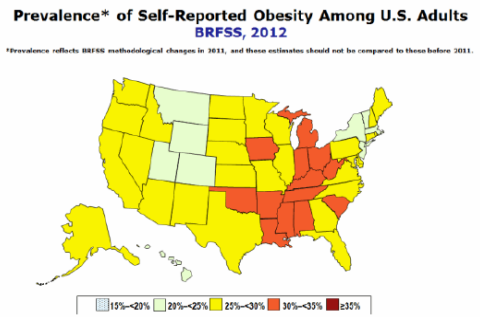
Figure 22 is a mortality map of the U.S., by state. Note the correlation among high mortality rates, obesity rates, and pesticide usage among the four maps presented here.

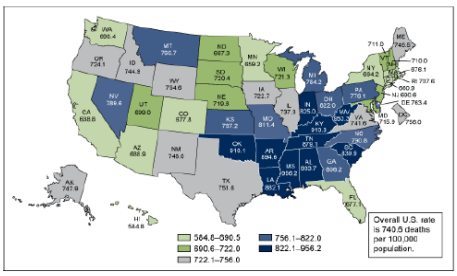
Figure 22. Obesity, Mortality, and Glyphosate Spray Maps in the U.S. Compared

Comparing Obesity and Mortality Rates



NOTICE THAT THE REGIONS WITH THE HEAVIEST PESTICIDES SPRAY HAVE THE HIGHEST OBESITY RATES AND ALSO HAVE THE HIGHEST MORTALITY RATES





Age-adjusted death rates for each state and the District of Columbia: United States, preliminary 2011

1

EVIDENCE LINKING OBESITY AND COMORBIDITIES TO ENDOCRINE-DISRUPTING CHEMICALS

There is no doubt that the state of our environment is linked to diseases affecting the entire planet. This link was established in the 1960s and led to a number of environmental-control measures; however, we are losing ground in our efforts to protect the environment. At the same time, obesity and its comorbidities are slowly ravaging populations worldwide. In the next section, I will discuss the link between

pesticides and household chemicals (including cosmetics and cosmeceuticals) and the obesity and allergy epidemics.

The CDC conducts surveys known as biomonitoring. Biomonitoring involves checking for environmental chemicals in the blood and urine samples of people across the country. The results of these tests are published annually in the *Fourth Report on Human Exposure to Environmental Chemicals.* The first four reports contained cumulative data from national samples collected in 1999-2000, 2001-2002, 2003-2004, and 2005-2006. In these reports, it was found that 90% of the individuals tested had chemicals in their blood and urine. With each year, the number of chemicals on the list gets longer.

THE EFFECTS OF ENDOCRINE-DISRUPTING CHEMICALS ON THE HUMAN BODY

The following information is based on data published on the website of the authors of the book, *Our Stolen Future* (www.ourstolenfuture.org). I am using this data for illustration only.

Persistent organohalogens

Compound(s)	Hormone system affected	Mechanism if known	References
Benzenehexachloride (BHC)	Thyroid		Akhtar *et al.* 1996
1,2-dibromoethane	Reproductive		Brittebo *et al.* 1987
Chloroform	Reproductive		Brittebo *et al.* 1987
Dioxins and furans (in order of antiestrogenic potency : 2,3,7,8-tetrachlorodibenzo-p-dioxin > 2,3,7,8-tetrachlorodibenzofuran > 2,3,4,7,8-pentachlorodibenzo-furan > 1,2,3,7,9-pentachlorodibenzofuran > 1,3,6,8-tetrachlo-rodibenzofuran)	Estrogen	work as anti-estrogen through binding with Ah receptor, which then inhibits estrogen receptor binding to estrogen response elements, thereby inhibiting estrogen action	Krishnan and Safe 1993 Klinge *et al.* 1999
Octachlorostyrene	Thyroid		Sandan *et al.* 2000
PBBs	Estrogen/ Thyroid		Bahn *et al.* 1980 Henderson *et al.* 1995
PCBs (in order of antiestrogenic potency: 3,3' -pentachlorobiphenyl > 3,3,4,4,5,5'-hexachlorobiphenyl 3,3',4,4-tetrachlorobiphenyl > 2,3,3',4,4',5'-hexa, 2,3,3',4,4'- and	Estrogen/androgen/Thyroid Adverse outcomes in reproductive systems.	Inhibits estrogen binding to the receptor; works as anti-estrogen.	Korach *et al.* 1988 Zoeller *et al.* 2000 Grey *et al.* 1999

2,3,4,4',5-pentachlorobiphenyl > Aroclors 1221, 1232. 1248, 1254, and 1260 were inactive as antiestrogens at the highest concentrations used in this study (10-6 Ni)		anti-androgenic via Ah receptor interaction	
PCB, hydroxylated	Thyroid	Binds to thyroid hormone binding protein, but not to the thyroid hormone receptor.	Cheek *et al.* 1999
PBDEs	Thyroid	Interfere with thyroxine (T4) binding with transthryetin	Ilonka *et al.* 2000
Pentachlorophenol	Thyroid	Reduces thyroid hormone possibly through a direct effect on the thyroid gland.	Bear *et al.* 1999 Gerhard *et al.* 1999

Food Antioxidant

Compound	Hormone system affected	Mechanism	References
Butylated hydroxyanisole (BHA)	Estrogen	Inhibits binding to the estrogen receptor.	Jobling *et al.* 1995

Pesticide (for information organized by pesticide class)

Compound	Hormone system affected	Mechanism	References
Acetochlor	Thyroid (decrease of thyroid hormone levels, increase in TSH)		Hurley *et al.* 1998
Alachlor	Thyroid (decrease of thyroid hormone levels, increase in TSH)		Wilson *et al.* 1996
Aldrin	Estrogen	Binds to estrogen receptors; competes with estradiol.	Jorgenson 2001
Allethrin, d-trans	Estrogen		Go *et al.* 1999
Amitrol	Thyroid	Thyroid peroxidase inhibitors; inhibits thyroid hormone synthesis.	Hurley *et al.* 1998
Atrazine	Neuroendocrine-pituitary (depression of LH surge), testosterone metabolism.	Inhibits ligand binding to androgen and estrogen receptors.	Danzo 1997
Carbaryl	Estrogen and progesterone		Klotz *et al.* 1997
Chlofentezine	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998

Chlordane	Testosterone and progesterone		Willingham *et al.* 2000
Cypermethrin	Disruption of reproductive function		Moore and Waring 2001
DDT	Estrogen	DDT and related compounds act in a number of ways to disrupt endocrine function by binding with the estrogen receptor, including estrogen mimickry and antagonism, altering the pattern of synthesis or metabolism of hormones, and (4) modifying hormone receptor levels	Soto *et al.* 1994 Lascombe *et al.* 2000 Kupfer *et al.* 1980 Rajapakse *et al.* 2001
DDT Metabolite, p,p'-DDE	Androgen	Inhibits androgen binding to the androgen receptor, androgen-induced transcriptional activity, and androgen action in developing, pubertal and adult male rats.	Kelce 1995
Dicofol (Kelthane)	Estrogen		Vinggaard *et al.* 1999
Dieldrin	Estrogen	Binds to estrogen receptor;competes with estradiol.	Soto *et al.* 1994 Jorgenson 2001
Endosulfan	Estrogen		Soto *et al.* 1994 Soto *et al.* 1995
Ethylene thiourea	Thyroid	Thyroid peroxidase inhibitor.	Hurley *et al.* 1998
Fenarimol	Estrogen	Estrogen receptor agonist.	Vinggaard *et al.* 1999
Fenbuconazole	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Fenitrothion	Antiandrogen	Competitive androgen receptor antagonist.	Tamura *et al.* 2001
Fenvalerate	Estrogen		Go *et al.* 1999
Fipronil	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Heptachlor	Thyroid		Akhtar *et al.* 1996 Reuber 1987
Heptachlor-epoxide	Thyroid/Reproductive	Metabolite of heptachlor	Reuber 1987

Iprodione	Inhibition of testosterone synthesis		Benhamed 1996
Karate	Thyroid	A decrease of thyroid hormone in serum; direct effect on the thyroid gland?	Akhtar *et al.* 1996
Kepone (Chlordecone)	Estrogen	Displays androgen and estrogen receptor-binding affinities.	Waller *et al.* 1996 Soto *et al.* 1994 McLachlan(ed)
Ketoconazole	Effects on reproductive systems		Marty *et al.* 1999 Marty *et al.* 2001
Lindane (Hexachlorocyclohexane)	Estrogen/Androgen	Inhibits ligand binding to androgen and estrogen receptors.	Danzo 1997
Linuron	Androgen	Androgen receptor antagonist.	Waller *et al.* 1996 Lambright *et al.* 2000 Grey *et al.* 1999
Malathion	Thyroid	Significant decrease of thyroid hormone in serum, with perhaps a direct effect on the thyroid gland.	Akhtar *et al.* 1996
Mancozeb	Thyroid	Thyroid peroxidase inhibitors.	Hurley *et al.* 1998
Maneb	Thyroid	The metabolite ethylenthiourea inhibits thyroid hormone synthesis.	Toppari *et al.* 1995
Methomyl	Thyroid		Porter *et al.* 1993 Klotz *et al.* 1997
Methoxychlor	Estrogen	Through mechanisms other than receptor antagonism. Precise mechanism still unclear.	Pickford and Morris 1999
Metribuzin	Thyroid		Porter *et al.* 1993

Mirex	Antiandrogenic activity; inhibits production of LH. Potentially thyroid.		Chen *et al.* 1986 Chernoff *et al.* 1976
Nitrofen	Thyroid	Structural similarities to the thyroid hormones; nitrofen or its metabolite may have thyroid hormone activities.	Stevens and Summer 1991
Nonachlor, trans-	Estrogen	Estrogen receptor agonist?	Willingham *et al.* 2000
Oxychlordane	Reproductive		Guillette *et al.* 1999
Pendimethalin	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Pentachloronitrobenzene	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Permethrin	Estrogenic		Go et al. 1999
Procymidone	Androgen	Androgen receptor antagonist.	Ostby *et al.* 1999 Grey *et al.* 1999
Prodiamine	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Pyrimethanil	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Sumithrin	Androgen		Go *et al.* 1999
Tarstar	Thyroid	A decrease of thyroid hormone in serum; direct effect on the thyroid gland?	Akhtar *et al.* 1996
Thiazopyr	Thyroid	Enhances secretion of thyroid hormone.	Hurley *et al.* 1998
Thiram	Neuroendocrine-pituitary (depression of LH surge), thyroid (decrease of T4, increase of TSH)		Stoker *et al.* 1993
Toxaphene	Estrogen/ Thyroid		Soto *et al.* 1994
Triadimefon	Estrogen	Estrogen receptor agonist.	Vinggaard *et al.* 1999
Triadimenol	Estrogen	Estrogen receptor agonist	Vinggaard *et al.* 1999
Tributyltin	Reproductive		Horiguchi *et al.* 2000
Trifluralin	Reproductive/ Metabolic		Rawlings *et al.* 1998
Vinclozolin	Androgen	Anti-androgenic. (Competes with androgens for the androgen receptor	Soto *et al.* 1994

		(AR), inhibits AR-DNA binding, and alters androgen-dependent gene expression.)	Soto *et al.* 1995 Kelce *et al.* 1994 Grey *et al.* 1999
Zineb	Thyroid	The metabolite ethylenthiourea inhibits thyroid hormone synthesis.	Toppari *et al.* 1995
Ziram	Thyroid	Inhibits the iodide peroxidase. Structural similarities between ziram and thiram; ziram can be metabolized to thiram in the environment.	Marinovich *et al.* 1997

Phthalate

Compound	Hormones affected	Mechanism	References
Butyl benzyl phthalate (BBP)	Estrogen	Inhibits binding to the estrogen receptor	Jobling *et al.* 1995
Di-n-butyl phthalate (DBP)	Estrogen Androgen	Inhibits binding to the estrogen receptor. anti-androgenic	Jobling *et al.* 1995 Harris *et al.* 1997 Grey *et al.* 1999
Di-ethylhexyl phthalate (DEHP)	Estrogen Androgen	Inhibits binding to the estrogen receptor. anti-androgenic	Jobling *et al.* 1995 Harris *et al.* 1997 Moore *et al.* 2001 Grey *et al.* 1999
Diethyl Phthalate (DEP)	Estrogen		Harris *et al.* 1997

Other Compounds

Compound	Hormones affected	Mechanism	References
Benzophenone	Estrogen	Binds weakly to estrogen receptors, roles of its metabolite remain to be clarified.	Schlumpf *et al.* 2001
Bisphenol A	Estrogen	Estrogenic; binds to estrogen receptor	Fisher *et al.* 1999

			Anderson *et al.* 1999 Rajapakse *et al.* 2001
Bisphenol A	Estrogen	Estrogenic; binds to estrogen receptor	Fisher *et al.* 1999 Anderson *et al.* 1999 Rajapakse *et al.* 2001
Bisphenol F	Estrogen	Estrogenic; b inds to estrogen receptor	Perez *et al.* 1998
Benzo(a)pyrene	Androgen	anti-androgenic	Thomas 1990
Carbendazim	Reproductive		Gray *et al.* 1990
Ethane Dimethane Sulphonate	Reproductive		Gray *et al.* 1999
Perfluorooctane sulfonate (PFOS)	Thyroid, reproductive	suppression of T3,T4; mechanism unknown	3M data
Nonylphenol, octylphenol	Estrogen	Estrogen receptor agonists; reduces estradiol binding to the estrogen receptor.	Soto *et al.* 1991 Soto *et al.* 1995 Danzo 1997 Lascombe *et al.* 2000 Rajapakse *et al.* 2001
Resorcinol	Thyroid		Lindsay *et al.* 1989
Styrene dimers and trimers	Estrogen	Estrogen receptor agonists	Ohyama *et al.* 2001

Metals

Compound	Hormones affected	Mechanism	References
Arsenic	Glucocorticoid	Selective inhibition of DNA transcription normally stimulated by the glucocorticoid-GR complex.	Kaltreider *et al.* 2001

Cadmium	Estrogenic	Activates estrogen receptor through an interaction with the hormone-binding domain of the receptor.	Stoica *et al.* 2000 Johnson *et al.* 2003
Lead	Reproductive		Telisman *et al.* 2000 Hanas *et al.* 1999
Mercury	Reproductive/ Thyroid		Facemire *et al.* 1995

Most pesticides and herbicides used in the U.S. have major effects on the thyroid gland, the reproductive system, and estrogen. Hypothyroidism, hyperestrogenism, and hypoandrogenism in men and women are the major direct effects of these chemicals.

Endocrine-Disrupting Chemicals Timeline

This is a timeline of the appearance and usage of endocrine-disrupting chemicals (pesticides) in the U.S.:

• 1940s to 1970s: Organochlorines (estrogenic chemical compounds such as DDT) were introduced, resulting in the beginning of endocrine-disrupting chemical pollution of the environment. An alarm was sounded about this toxification of the environment by Rachel Carson (1907-1964) in her book, *Silent Spring*, which was published in 1962.

• 1970s to the present: Organochlorines were banned, but still persisted in the environment and continue to persist as xenoestrogens. They were replaced by organophosphate insecticides, which added acetylcholine-related problems to xenoestrogens-related problems.

• 1990s to the present: GMOs were introduced on a large scale, along with Roundup (glyphosate), a potent endocrine-disrupting chemical. Other pesticides such as triazine compounds and chloracetamides were introduced. The term *endocrine disruptor* was coined at the Wingspread Conference Center in Wisconsin in 1991. One of the early papers on the phenomenon was presented by Theo Colborn in 1993. In it, she talked about the effects of environmental chemicals on the development of the endocrine system in fetuses. She and her colleagues further explored the effects of endocrine disruptors in the book, *Our Stolen Future*, which was published in 1996,

at which time the obesity and allergy epidemics began. I issued the alarm about this situation in my book, *Hormone Imbalance Syndrome: America's Silent Plague,* which was published in 2012.

Persistent Organic Pollutants

The efforts of Rachel Carson and others led to the ban of organochlorine insecticides, such as DDT and its metabolites, in 1972. These organochlorine insecticides remain in the environment, however, and are known as persistent organic pollutants (POPs). Organochlorine insecticides are known to be estrogenic and, therefore, act as endocrine disruptors. Organochlorine insecticides were replaced by organophosphate and carbamate insecticides, which are POPs. The total load and synergistic effects of these pesticides accumulate over decades and accelerate endocrine-related problems, such as obesity, diabetes, hypertension, menstrual disturbances, and cancer, as well as the growth of allergy and autoimmune diseases.

Table 6. List of Banned Chemicals That Still Persist in the Environment

The "Dirty Dozen"

POP	Global Historical Use/Source	Overview of U.S. Status
aldrin and dieldrin	Insecticides used on crops such as corn and cotton; also used for termite control.	Under FIFRA: 1. No U.S. registrations; most uses canceled in 1969; all uses by 1987. 2. All tolerances on food crops revoked in 1986. No production, import, or export.
chlordane	Insecticide used on crops, including vegetables, small grains, potatoes, sugarcane, sugar beets, fruits, nuts, citrus, and cotton. Used on home lawn and garden pests. Also used extensively to control termites.	Under FIFRA: 1. No U.S. registrations; most uses canceled in 1978; all uses by 1988. 2. All tolerances on food crops revoked in 1986. No production (stopped in 1997), import, or export. Regulated as a hazardous air pollutant (CAA).

DDT	Insecticide used on agricultural crops, primarily cotton, and insects that carry diseases such as malaria and typhus.	Under FIFRA: No U.S. registrations; most uses canceled in 1. 1972; all uses by 1989. 2. Tolerances on food crops revoked in 1986. No U.S. production, import, or export. DDE (a metabolite of DDT) regulated as a hazardous air pollutant (CAA). Priority toxic pollutant (CWA).
endrin	Insecticide used on crops such as cotton and grains; also used to control rodents.	Under FIFRA, no U.S. registrations; most uses canceled in 1979; all uses by 1984. No production, import, or export. Priority toxic pollutant (CWA).
mirex	Insecticide used to combat fire ants, termites, and mealybugs. Also used as a fire retardant in plastics, rubber, and electrical products.	Under FIFRA, no U.S. registrations; all uses canceled in 1977. No production, import, or export.
heptachlor	Insecticide used primarily against soil insects and termites. Also used against some crop pests and to combat malaria.	Under FIFRA: 1. Most uses canceled by 1978; registrant voluntarily canceled use to control fire ants in underground cable boxes in early 2000. 2. All pesticide tolerances on food crops revoked in 1989. No production, import, or export.
hexachlorobenzene	Fungicide used for seed treatment. Also an industrial chemical used to make fireworks, ammunition, synthetic rubber, and other substances. Also unintentionally produced during combustion and the manufacture of certain chemicals. Also an impurity in certain pesticides.	Under FIFRA, no U.S. registrations; all uses canceled by 1985. No production, import, or export as a pesticide. Manufacture and use for chemical intermediate (as allowed under the Convention). Regulated as a hazardous air pollutant (CAA). Priority toxic pollutant (CWA).

PCBs	Used for a variety of industrial processes and purposes, including in electrical transformers and capacitors, as heat exchange fluids, as paint additives, in carbonless copy paper, and in plastics. Also unintentionally produced during combustion.	Manufacture and new use prohibited in 1978 (TSCA). Regulated as a hazardous air pollutant (CAA). Priority toxic pollutant (CWA).
toxaphene	Insecticide used to control pests on crops and livestock, and to kill unwanted fish in lakes.	Under FIFRA: 1. No U.S. registrations; most uses canceled in 1982; all uses by 1990. 2. All tolerances on food crops revoked in 1993. No production, import, or export. Regulated as a hazardous air pollutant (CAA).
dioxins and furans	Unintentionally produced during most forms of combustion, including burning of municipal and medical wastes, backyard burning of trash, and industrial processes. Also can be found as trace contaminants in certain herbicides, wood preservatives, and in PCB mixtures.	Regulated as hazardous air pollutants (CAA). Dioxin in the form of 2,3,7,8-TCDD is a priority toxic pollutant (CWA).

- **FIFRA:** Federal Insecticide, Fungicide and Rodenticide Act
- **TSCA:** Toxic Substances Control Act
- **CAA:** Clean Air Act
- **CWA:** Clean Water Act

❖ Watch the documentary: BLUE GOLD on YouTube www.youtube.com/watch?v=B1a3tjqQiBI&feature=player_embedded

❖ Watch the documentary: Origins on YouTube www.youtube.com/watch?v=d9HBad0yZHo

Table 7 shows selected endocrine-disrupting chemicals listed in the CDC's 2013 Fourth Report

Table 7. Selected Endocrine Disruptors Listed in the Center for Disease Control and Prevention (CDC), *Fourth National Report on Human Exposure to Environmental Chemicals,* 2013

Chemical	Effect on Body
Carbamate insecticides	Acetylcholine producers
Dioxins and dioxin-like compounds**	Estrogenic
Environmental phenols (bisphenol A, triclosan)	Estrogenic
Metals (cadmium, calcium, cobalt, copper, nickel, chromium, lead, mercury, tin)	Estrogenic
Organochlorine pesticides and metabolite*	Estrogenic
Organophosphorus insecticides	Acetylcholine producers
Parabens**	Estrogenic
Perchlorate	Thyroid disruptor
Perfluorochemicals (PFCs)	Thyroid disruptors
Phthalates and phthalate metabolites**	Estrogenic
Phytoestrogens and metabolites	Estrogenic
Polycyclic aromatic hydrocarbon metabolites	Estrogenic
Pyrethroid pesticides	Neurotoxic

*Banned worldwide, but still persists in the environment.

**Category contains compounds added to list since publication of 2009 CDC *Fourth Report.*

Chemicals in the *Fourth Report: Updated Tables, September 2013*

CDC's *Fourth National Report on Human Exposure to Environmental Chemicals: Updated Tables* provides exposure data on the following chemicals or classes of chemicals. The Updated Tables contain cumulative data from national samples collected beginning in 1999–2000 and as recently as 2009-2010. Not all chemicals were measured in each national sample. The data tables are available at http://www.cdc.gov/exposurereport. An asterisk (*) indicates the chemical has been added since publication of the *Fourth Report* in 2009.

Tobacco Smoke
- Cotinine
- NNAL*

Disinfection By Products
- Bromodichloromethane
- Dibromochloromethane
- Tribromomethane (Bromoform)
- Trichloromethane (Chloroform)

THYROID DISRUPTORS

Environmental Phenols
- Benzophenone-3
- Bisphenol A
- 4-*tert*-Octylphenol
- Triclosan

ESTROGENIC

Fungicides and Metabolites
- *ortho*-Phenylphenol
- Ethylene thiourea*
- Pentachlorophenol
- Propylene thiourea*

Herbicides and Metabolites
- 2,4-Dichlorophenoxyacetic acid
- 2,4,5-Trichlorophenoxyacetic acid

Sulfonyl Urea Herbicides
- Urinary Bensulfuron-methyl*
- Urinary Chlorsulfuron*
- Urinary Ethametsulfuron-methyl*
- Urinary Foramsulfuron*
- Urinary Halosulfuron*
- Urinary Mesosulfuron-methyl*
- Urinary Metsulfuron-methyl*
- Urinary Nicosulfuron*
- Urinary Oxasulfuron*
- Urinary Primisulfuron-methyl*
- Urinary Prosulfuron*
- Urinary Rimsulfuron*
- Urinary Sulfometuron-methyl*
- Urinary Sulfosulfuron*
- Urinary Thifensulfuron-methyl*
- Urinary Triasulfuron*
- Urinary Triflusulfuron-methyl*

Carbamate Pesticide Metabolites
- Carbofuranphenol
- 2-Isopropoxyphenol

Organochlorine Pesticides and Metabolites
- Aldrin
- Dieldrin
- Endrin
- Heptachlor epoxide
- *o,p'*-DDT
- 2,4,5-Trichlorophenol
- 2,4,6-Trichlorophenol

ESTROGENIC

Other Pesticide Metabolites
- 2,4-Dichlorophenol*
- 2,5-Dichlorophenol*

Organophosphorus Insecticides: Specific Metabolites
- Acephate*
- Dimethoate*
- Methamidophos*
- Omethoate*

ACETYLCHOLINE

Organophosphorus Insecticides: Dialkyl Phosphate Metabolites
- Diethylphosphate (DEP)
- Dimethylphosphate (DMP)
- Diethylthiophosphate (DETP)
- Dimethylthiophosphate (DMTP)
- Diethyldithiophosphate (DEDTP)
- Dimethyldithiophosphate (DMDTP)

ACETYLCHOLINE

Metals and Metalloids
- Antimony
- Arsenic, Total
  - Arsenic (V) acid
  - Arsenobetaine
  - Arsenocholine
  - Arsenous (III) acid
  - Dimethylarsinic acid
  - Monomethylarsonic acid
  - Trimethylarsine oxide
- Barium
- Beryllium
- Cadmium
- Cesium
- Cobalt
- Lead
- Mercury, Total
  - Mercury, Inorganic
- Molybdenum
- Platinum
- Thallium
- Tungsten
- Uranium

METALLOESTROGENS
cadmium, calcium, cobalt, copper, nickel, chromium, lead, mercury, and tin

Parabens
- Butyl paraben*
- Ethyl paraben*
- Methyl paraben*
- *n*-Propyl paraben*

ESTROGENIC

Perchlorate and Other Anions
- Nitrate*
- Perchlorate
- Thiocyanate*

Perfluorinated Compounds: Surfactants
- Perfluorobutane sulfonic acid (PFBuS)
- Perfluorodecanoic acid (PFDeA)
- Perfluorododecanoic acid (PFDoA)
- Perfluoroheptanoic acid (PFHpA)
- Perfluorohexane sulfonic acid (PFHxS)
- Perfluorononanoic acid (PFNA)
- Perfluorooctanoic acid (PFOA)
- Perfluorooctane sulfonic acid (PFOS)
- Perfluorooctane sulfonamide (PFOSA)
- 2-(N-Ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH)
- 2-(N-Methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH)
- Perfluoroundecanoic acid (PFUA)

PFCs ARE THYROID DISRUPTORS And Neurotoxins-persistent pollutants

Chemicals in the *Fourth Report: Updated Tables, February 2015*

CDC's *Fourth National Report on Human Exposure to Environmental Chemicals: Updated Tables* provides exposure data on the following chemicals or classes of chemicals. The Updated Tables contain cumulative data from national samples collected beginning in 1999–2000 and as recently as 2011-2012. Not all chemicals were measured in each national sample. The data tables are available at http://www.cdc.gov/exposurereport. An asterisk (*) indicates the chemical has been added since publication of the *Fourth Report* in 2009.

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- Cotinine
- NNAL*

Disinfection By-Products
- Bromodichloromethane
- Dibromochloromethane (Chlorodibromomethane)
- Tribromomethane (Bromoform)
- Trichloromethane (Chloroform)

Environmental Phenols
- Benzophenone-3
- Bisphenol A
- 4-*tert*-Octylphenol
- Triclosan

Fungicides and Metabolites
- *ortho*-Phenylphenol
- Ethylene thiourea*
- Pentachlorophenol
- Propylene thiourea*

Herbicides and Metabolites
- 2,4-Dichlorophenoxyacetic acid
- 2,4,5-Trichlorophenoxyacetic acid

Sulfonylurea Herbicides
- Bensulfuron-methyl*
- Chlorsulfuron*
- Ethametsulfuron-methyl*
- Foramsulfuron*
- Halosulfuron*
- Mesosulfuron-methyl*
- Metsulfuron-methyl*
- Nicosulfuron*
- Oxasulfuron*
- Primisulfuron-methyl*
- Prosulfuron*
- Rimsulfuron*
- Sulfometuron-methyl*
- Sulfosulfuron*
- Thifensulfuron-methyl*
- Triasulfuron*
- Triflusulfuron-methyl*

Carbamate Pesticide Metabolites
- Carbofuranphenol
- 2-Isopropoxyphenol

Organochlorine Pesticides and Metabolites
- Aldrin
- Dieldrin
- Endrin
- Heptachlor epoxide
- *o,p'*-Dichlorodiphenyltrichloroethane (DDT)
- 2,4,5-Trichlorophenol
- 2,4,6-Trichlorophenol

Other Pesticides and Metabolites
- 2,4-Dichlorophenol*
- 2,5-Dichlorophenol*

Organophosphorus Insecticides: Specific Metabolites
- Acephate*
- Dimethoate*
- Methamidophos*
- Omethoate*
- Malathion dicarboxylic acid
- 2-Isopropyl-4-methyl-6-hydroxypyrimidine
- *para*-Nitrophenol
- 3,5,6-Trichloro-2-pyridinol

Organophosphorus Insecticides: Dialkyl Phosphate Metabolites
- Diethylphosphate (DEP)
- Dimethylphosphate (DMP)
- Diethylthiophosphate (DETP)
- Dimethylthiophosphate (DMTP)
- Diethyldithiophosphate (DEDTP)
- Dimethyldithiophosphate (DMDTP)

Pyrethroid Metabolites
- *trans*-3-(2,2-Dichlorovinyl)-2,2-dimethylcyclopropane carboxylic acid
- *cis*-3-(2,2-Dibromovinyl)-2,2-dimethylcyclopropane carboxylic acid
- 4-Fluoro-3-phenoxybenzoic acid
- 3-Phenoxybenzoic acid

Metals and Metalloids
- Antimony
- Arsenic, Total
  - Inorganic Arsenic-related Species*
  - Arsenic (V) acid
  - Arsenobetaine
  - Arsenocholine
  - Arsenous (III) acid
  - Dimethylarsinic acid
  - Monomethylarsonic acid
  - Trimethylarsine oxide
- Barium
- Beryllium
- Cadmium
- Cesium
- Cobalt
- Copper*
- Lead
- Manganese*
- Mercury (total; inorganic; ethyl* and methyl species*)
- Molybdenum
- Platinum
- Selenium*
- Strontium*
- Thallium
- Tin*
- Tungsten
- Uranium
- Zinc*

Parabens
- Butyl paraben*
- Ethyl paraben*
- Methyl paraben*
- *n*-Propyl paraben*

Perchlorate and Other Anions
- Nitrate*
- Perchlorate
- Thiocyanate*

Perfluorinated Compounds: Surfactants
- Perfluorobutane sulfonic acid (PFBuS)
- Perfluorodecanoic acid (PFDeA)
- Perfluorododecanoic acid (PFDoA)
- Perfluoroheptanoic acid (PFHpA)
- Perfluorohexane sulfonic acid (PFHxS)
- Perfluorononanoic acid (PFNA)
- Perfluorooctanoic acid (PFOA)
- Perfluorooctane sulfonic acid (PFOS)
- Perfluorooctane sulfonamide (PFOSA)
- 2-(N-Ethyl-perfluorooctane sulfonamido) acetic acid (Et-PFOSA-AcOH)
- 2-(N-Methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH)
- Perfluoroundecanoic acid (PFUA)

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Phthalate Metabolites

- Mono-benzyl phthalate (MBzP)
- Mono-isobutyl phthalate (MiBP)
- Mono-n-butyl phthalate (MnBP)
- Mono-cyclohexyl phthalate (MCHP)
- Mono-ethyl phthalate (MEP)
- Mono-2-ethylhexyl phthalate (MEHP)
- Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)
- Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)
- Mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP)
- Mono-(carboxynonyl) phthalate (MCNP)*
- Mono-isononyl phthalate (MiNP)
- Mono-(carboxyoctyl) phthalate (MCOP)*
- Mono-methyl phthalate (MMP)
- Mono-(3-carboxypropyl) phthalate (MCPP)
- Mono-n-octyl phthalate (MOP)

ESTRO-GENIC

Phytoestrogens and Metabolites

- Urinary Daidzein
- Urinary Enterodiol
- Urinary Enterolactone
- Urinary Equol
- Urinary Genistein
- Urinary O-Desmethylangolensin

ESTROGENIC

Polycyclic Aromatic Hydrocarbon Metabolites

- 2-Hydroxyfluorene
- 3-Hydroxyfluorene
- 9-Hydroxyfluorene
- 1-Hydroxyphenanthrene
- 2-Hydroxyphenanthrene
- 3-Hydroxyphenanthrene
- 4-Hydroxyphenanthrene
- 1-Hydroxypyrene
- 1-Hydroxynaphthalene (1-Naphthol)
- 2-Hydroxynaphthalene (2-Naphthol)

ESTROGENIC

Volatile Organic Compounds

- 1,1,1-Trichloroethane (Methyl chloroform)
- 1,1,2,2-Tetrachloroethane
- 1,1,2-Trichloroethane
- 1,1-Dichloroethane
- 1,1-Dichloroethene (Vinylidene chloride)
- 1,2-Dibromo-3-chloropropane (DBCP)
- 1,2-Dichlorobenzene
- 1,2-Dichloroethane (Ethylene dichloride)
- *cis*-1,2-Dichloroethene
- *trans*-1,2-Dichloroethene
- 1,2-Dichloropropane
- 1,3-Dichlorobenzene
- 1,4-Dichlorobenzene (Paradichlorobenzene)
- 2,5-Dimethylfuran
- Benzene
- Chlorobenzene
- Dibromomethane
- Dichloromethane (Methylene chloride)
- Ethylbenzene
- Hexachloroethane
- Methyl-*tert*-butyl ether (MTBE)
- Nitrobenzene
- Styrene
- Tetrachloroethene (Perchloroethylene)
- Tetrachloromethane (Carbon tetrachloride)
- Toluene
- Trichloroethene (Trichloroethylene)
- *m*-/*p*-Xylene
- *o*-Xylene

Organochlorine Pesticides and Metabolites (Pooled Samples)

- Oxychlordane
- *trans*-Nonachlor
- *p,p'*-DDT
- *p,p'*-DDE
- Hexachlorobenzene
- *beta*-Hexachlorocyclohexane
- *gamma*-Hexachlorocyclohexane
- Mirex

ESTROGENIC

Polybrominated Diphenyl Ethers and PBB 153 (Pooled Samples)

- 2,2',4-Tribromodiphenyl ether (BDE 17)
- 2,4,4'-Tribromodiphenyl ether (BDE 28)
- 2,2',4,4'-Tetrabromodiphenyl ether (BDE 47)
- 2,3',4,4'-Tetrabromodiphenyl ether (BDE 66)
- 2,2',3,4,4'-Pentabromodiphenyl ether (BDE 85)
- 2,2',4,4',5-Pentabromodiphenyl ether (BDE 99)
- 2,2',4,4',6-Pentabromodiphenyl ether (BDE 100)
- 2,2',4,4',5,5'-Hexabromodiphenyl ether (BDE 153)
- 2,2',4,4',5,6'-Hexabromodiphenyl ether (BDE 154)
- 2,2',3,4,4',5',6-Heptabromodiphenyl ether (BDE 183)
- 2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl ether (BDE 209)*
- 2,2',4,4',5,5'-Hexabromobiphenyl (PBB 153)

ESTROGENIC

Polybrominated Dibenzo-*p*-dioxins (Pooled Samples)

- 1,2,3,4,6,7,8-Heptabromodibenzo-*p*-dioxin (HpBDD)*
- 1,2,3,4,7,8 and 1,2,3,6,7,8-Hexabromodibenzo-*p*-dioxin (HxBDD)*
- 1,2,3,7,8,9-Hexabromodibenzo-*p*-dioxin (HxBDD)*
- 1,2,3,7,8-Pentabromodibenzo-*p*-dioxin (PeBDD)*
- 2,3,7,8-Tetrabromodibenzo-*p*-dioxin (TBDD)*

ESTROGENIC

Polybrominated Dibenzofurans (Pooled Samples)

- 1,2,3,4,6,7,8-Heptabromodibenzofuran (HpBDF)*
- 1,2,3,4,7,8-Hexabromodibenzofuran (HxBDF)*
- 1,2,3,7,8-Pentabromodibenzofuran (PeBDF)*
- 2,3,4,7,8-Pentabromodibenzofuran (PeBDF)*
- 2,3,7,8-Tetrabromodibenzofuran (TBDF)*

ESTROGENIC

Polychlorinated Dibenzo-*p* dioxins (Pooled Samples)

- 1,2,3,4,6,7,8-Heptachlorodibenzo-*p*-dioxin (HpCDD)
- 1,2,3,4,7,8-Hexachlorodibenzo-*p*-dioxin (HxCDD)
- 1,2,3,6,7,8-Hexachlorodibenzo-*p*-dioxin (HxCDD)
- 1,2,3,7,8,9-Hexachlorodibenzo-*p*-dioxin (HxCDD)
- 1,2,3,4,6,7,8,9-Octachlorodibenzo-*p*-dioxin (OCDD)
- 1,2,3,7,8-Pentachlorodibenzo-*p*-dioxin (PeCDD)
- 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD)

ESTROGENIC

Polychlorinated Dibenzofurans (Pooled Samples)

- 1,2,3,4,6,7,8-Heptachlorodibenzofuran (HpCDF)
- 1,2,3,4,7,8,9-Heptachlorodibenzofuran (HpCDF)
- 1,2,3,4,7,8-Hexachlorodibenzofuran (HxCDF)
- 1,2,3,6,7,8-Hexachlorodibenzofuran (HxCDF)
- 1,2,3,7,8,9-Hexachlorodibenzofuran (HxCDF)
- 2,3,4,6,7,8-Hexachlorodibenzofuran (HxCDF)
- 1,2,3,4,6,7,8,9-Octachlorodibenzofuran (OCDF)
- 1,2,3,7,8-Pentachlorodibenzofuran (PeCDF)
- 2,3,4,7,8-Pentachlorodibenzofuran (PeCDF)
- 2,3,7,8-Tetrachlorodibenzofuran (TCDF)

ESTROGENIC

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Phthalate and Phthalate Alternative Metabolites

Mono-benzyl phthalate (MBzP)
Mono-isobutyl phthalate (MiBP)
Mono-n-butyl phthalate (MnBP)
Mono-cyclohexyl phthalate (MCHP)
Mono-ethyl phthalate (MEP)
Mono-2-ethylhexyl phthalate (MEHP)
Mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP)
Mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP)
Mono-(2-ethyl-5-carboxypentyl) phthalate (MECPP)
Mono-(carboxynonyl) phthalate (MCNP)*
Mono-isononyl phthalate (MiNP)
Mono-(carboxyoctyl) phthalate (MCOP)*
Mono-methyl phthalate (MMP)
Mono-(3-carboxypropyl) phthalate (MCPP)
Mono-n-octyl phthalate (MOP)
Cyclohexane-1,2-dicarboxylic acid mono(hydroxy-isononyl ester) (MHNCH)*

Phytoestrogens and Metabolites

Daidzein
Enterodiol
Enterolactone
Equol
Genistein
O-Desmethylangolensin

Polycyclic Aromatic Hydrocarbon Metabolites

2-Hydroxyfluorene
3-Hydroxyfluorene
9-Hydroxyfluorene
1-Hydroxyphenanthrene
2-Hydroxyphenanthrene
3-Hydroxyphenanthrene
4-Hydroxyphenanthrene
1-Hydroxypyrene
1-Hydroxynaphthalene (1-Naphthol)
2-Hydroxynaphthalene (2-Naphthol)

Volatile Organic Compounds (VOCs)

1,1,1-Trichloroethane (Methyl chloroform)
1,1,2,2-Tetrachloroethane
1,1,2-Trichloroethane
1,1-Dichloroethane
1,1-Dichloroethene (Vinylidene chloride)
1,2-Dibromo-3-chloropropane (DBCP)
1,2-Dichlorobenzene (*o*-Dichlorobenzene)
1,2-Dichloroethane (Ethylene dichloride)
cis-1,2-Dichloroethene
trans-1,2-Dichloroethene
1,2-Dichloropropane
1,3-Dichlorobenzene (*m*-Dichlorobenzene)
1,4-Dichlorobenzene (Paradichlorobenzene)
2,5-Dimethylfuran
Benzene
Chlorobenzene (Monochlorobenzene)
Dibromomethane
Dichloromethane (Methylene chloride)
Ethylbenzene
Hexachloroethane
Methyl-*tert*-butyl ether (MTBE)
Nitrobenzene
Styrene
Tetrachloroethene (Perchloroethylene)
Tetrachloromethane (Carbon tetrachloride)
Toluene
Trichloroethene (Trichloroethylene)
m-/*p*-Xylene
o-Xylene

Volatile Organic Compound (VOC) Metabolites

N-Acetyl-S-(benzyl)-L-cysteine*
N-Acetyl-S-(2-carbamoyl-2-hydroxyethyl)-L-cysteine*
N-Acetyl-S-(2-carbamoylethyl)-L-cysteine*
N-Acetyl-S-(2-carboxyethyl)-L-cysteine*
N-Acetyl-S-(3-hydroxypropyl)-L-cysteine*
N-Acetyl-S-(2-cyanoethyl)-L-cysteine*
N-Acetyl-S-(1,2-dichlorovinyl)-L-cysteine*
N-Acetyl-S-(2,2-dichlorovinyl)-L-cysteine*
N-Acetyl-S-(dimethylphenyl)-L-cysteine*
N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine*
N-Acetyl-S-(3,4-dihydroxybutyl)-L-cysteine*
N-Acetyl-S-(2-hydroxy-3-butenyl)-L-cysteine*
N-Acetyl-S-(4-hydroxy-2-butenyl)-L-cysteine*
N-Acetyl-S-(1-hydroxymethyl-2-propenyl)-L-cysteine*
N-Acetyl-S-(2-hydroxyethyl)-L-cysteine*
N-Acetyl-S-(2-hydroxypropyl)-L-cysteine*
N-Acetyl-S-(3-hydroxypropyl-1-methyl)-L-cysteine*
N-Acetyl-S-(phenyl)-L-cysteine*
t,t-Muconic acid*
N-Acetyl-S-(phenyl-2-hydroxyethyl)-L-cysteine*
N-Acetyl-S-(n-propyl)-L-cysteine*
N-Acetyl-S-(trichlorovinyl)-L-cysteine*
2-Aminothiazoline-4-carboxylic acid*
Mandelic acid*
2-Methylhippuric acid*
3- and 4-Methylhippuric acid*
Phenylglyoxylic acid*
2-Thioxothiazolidine-4-carboxylic acid*

Metals and Metalloids (Adult Cigarette Smokers and Nonsmokers: Special Sample)

Antimony
Arsenic, Total
  Arsenic (V) acid
  Arsenobetaine
  Arsenocholine
  Arsenous (III) acid
  Dimethylarsinic acid
  Monomethylarsonic acid
  Trimethylarsine oxide
Barium
Cadmium
Cesium
Cobalt
Lead
Manganese*
Molybdenum
Strontium*
Thallium
Tin*
Tungsten
Uranium

Perchlorate and Other Anions (Adult Cigarette Smokers and Nonsmokers: Special Sample)

Nitrate*
Perchlorate
Thiocyanate*

Polycyclic Aromatic Hydrocarbon Metabolites (Adult Cigarette Smokers and Nonsmokers: Special Sample)

2-Hydroxyfluorene
3-Hydroxyfluorene
9-Hydroxyfluorene
1-Hydroxyphenanthrene
2-Hydroxyphenanthrene
3-Hydroxyphenanthrene
4-Hydroxyphenanthrene
1-Hydroxypyrene
1-Hydroxynaphthalene (1-Naphthol)
2-Hydroxynaphthalene (2-Naphthol)

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Dioxin like Polychlorinated Biphenyls: Coplanar PCBs (Pooled Samples)
3,3',4,4'-Tetrachlorobiphenyl (PCB 77)*
3,4,4',5-Tetrachlorobiphenyl (PCB 81)
3,3',4,4',5-Pentachlorobiphenyl (PCB 126)
3,3',4,4',5,5'-Hexachlorobiphenyl (PCB 169)

Dioxin like Polychlorinated Biphenyls: Mono-*ortho*-substituted PCBs (Pooled Samples)
2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)
2,3,3',4,4'-Pentachlorobiphenyl (PCB 114)*
2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
2',3,4,4',5-Pentachlorobiphenyl (PCB 123)*
2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)
2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)

Non-Dioxin Like Polychlorinated Biphenyls (Pooled Samples)
2,4,4'-Trichlorobiphenyl (PCB 28)
2,2'3,5'-Tetrachloro biphenyl (PCB 44)
2,2',4,5'-Tetrachloro biphenyl (PCB 49)
2,2',5,5'-Tetrachlorobiphenyl (PCB 52)
2,3',4,4'-Tetrachlorobiphenyl (PCB 66)
2,4,4',5-Tetrachlorobiphenyl (PCB 74)
2,2',3,4,5'-Pentachlorobiphenyl (PCB 87)
2,2',4,4',5-Pentachlorobiphenyl (PCB 99)
2,2',4,5,5'-Pentachlorobiphenyl (PCB 101)
2,3,3',4',6-Pentachlorobiphenyl (PCB 110)
2,2',3,3',4,4'-Hexachlorobiphenyl (PCB 128)
2,2',3,4,4',5' and 2,3,3',4,4',6-Hexachlorobiphenyl (PCB 138 & 158)
2,2',3,4',5,5'-Hexachlorobiphenyl (PCB 146)
2,2',3,4',5',6-Hexachlorobiphenyl (PCB 149)
2,2',3,5,5',6-Hexachlorobiphenyl (PCB 151)
2,2',4,4',5,5'-Hexachlorobiphenyl (PCB 153)
2,2',3,3',4,4',5-Heptachlorobiphenyl (PCB 170)
2,2',3,3',4,5,5'-Heptachlorobiphenyl (PCB 172)
2,2',3,3',4,5',6'-Heptachlorobiphenyl (PCB 177)
2,2',3,3',5,5',6-Heptachlorobiphenyl (PCB 178)
2,2',3,4,4',5,5'-Heptachlorobiphenyl (PCB 180)
2,2',3,4,4',5',6-Heptachlorobiphenyl (PCB 183)
2,2',3,4',5,5',6-Heptachlorobiphenyl (PCB 187)
2,2',3,3',4,4',5,5'-Octachlorobiphenyl (PCB 194)
2,2',3,3',4,4',5,6-Octachlorobiphenyl (PCB 195)
2,2',3,3',4,4',5,6' and 2,2',3,4,4',5,5',6-Octachlorobiphenyl (PCB 196 & 203)
2,2',3,3',4,5,5',6-Octachlorobiphenyl (PCB 199)
2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl (PCB 206)
2,2',3,3',4,4',5,5',6,6'-Decachlorobiphenyl (PCB 209)

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Volatile Organic Compound (VOC) Metabolites (Adult Cigarette Smokers and Nonsmokers: Special Sample)

- N-Acetyl-S-(benzyl)-L-cysteine*
- N-Acetyl-S-(2-carbamoyl-2-hydroxyethyl)-L-cysteine*
- N-Acetyl-S-(2-carbamoylethyl)-L-cysteine*
- N-Acetyl-S-(2-carboxyethyl)-L-cysteine*
- N-Acetyl-S-(3-hydroxypropyl)-L-cysteine*
- N-Acetyl-S-(2-cyanoethyl)-L-cysteine*
- N-Acetyl-S-(1,2-dichlorovinyl)-L-cysteine*
- N-Acetyl-S-(2,2-dichlorovinyl)-L-cysteine*
- N-Acetyl-S-(dimethylphenyl)-L-cysteine*
- N-Acetyl-S-(N-methylcarbamoyl)-L-cysteine*
- N-Acetyl-S-(3,4-dihydroxybutyl)-L-cysteine*
- N-Acetyl-S-(2-hydroxy-3-butenyl)-L-cysteine*
- N-Acetyl-S-(4-hydroxy-2-butenyl)-L-cysteine*
- N-Acetyl-S-(1-hydroxymethyl-2-propenyl)-L-cysteine*
- N-Acetyl-S-(2-hydroxyethyl)-L-cysteine*
- N-Acetyl-S-(2-hydroxypropyl)-L-cysteine*
- N-Acetyl-S-(3-hydroxypropyl-1-methyl)-L-cysteine*
- N-Acetyl-S-(phenyl)-L-cysteine*
- *t,t*-Muconic acid*
- N-Acetyl-S-(phenyl-2-hydroxyethyl)-L-cysteine*
- N-Acetyl-S-(n-propyl)-L-cysteine*
- N-Acetyl-S-(trichlorovinyl)-L-cysteine*
- 2-Aminothiazoline-4-carboxylic acid*
- Mandelic acid*
- 2-Methylhippuric acid*
- 3- and 4-Methylhippuric acid*
- Phenylglyoxylic acid*
- 2-Thioxothiazolidine-4-carboxylic acid*

Organochlorine Pesticides and Metabolites (Pooled Samples)

- Oxychlordane
- *trans*-Nonachlor
- *p,p'*-DDT
- *p,p'*-DDE
- Hexachlorobenzene
- *beta*-Hexachlorocyclohexane
- *gamma*-Hexachlorocyclohexane
- Mirex

Polybrominated Diphenyl Ethers and PBB 153 (Pooled Samples)

- 2,2',4-Tribromodiphenyl ether (BDE 17)
- 2,4,4'-Tribromodiphenyl ether (BDE 28)
- 2,2',4,4'-Tetrabromodiphenyl ether (BDE 47)
- 2,3',4,4'-Tetrabromodiphenyl ether (BDE 66)
- 2,2',3,4,4'-Pentabromodiphenyl ether (BDE 85)
- 2,2',4,4',5-Pentabromodiphenyl ether (BDE 99)
- 2,2',4,4',6-Pentabromodiphenyl ether (BDE 100)
- 2,2',4,4',5,5'-Hexabromodiphenyl ether (BDE 153)
- 2,2',4,4',5,6'-Hexabromodiphenyl ether (BDE 154)
- 2,2',3,4,4',5',6-Heptabromodiphenyl ether (BDE 183)
- 2,2',3,3',4,4',5,5',6,6'-Decabromodiphenyl ether (BDE 209)*
- 2,2',4,4',5,5'-Hexabromobiphenyl (PBB 153)

Dioxin-like Polychlorinated Biphenyls: mono-*ortho*-substituted PCBs (Pooled Samples)

- 2,3,3',4,4'-Pentachlorobiphenyl (PCB 105)
- 2,3,3',4,4'-Pentachlorobiphenyl (PCB 114)*
- 2,3',4,4',5-Pentachlorobiphenyl (PCB 118)
- 2',3,4,4',5-Pentachlorobiphenyl (PCB 123)*
- 2,3,3',4,4',5-Hexachlorobiphenyl (PCB 156)
- 2,3,3',4,4',5'-Hexachlorobiphenyl (PCB 157)
- 2,3',4,4',5,5'-Hexachlorobiphenyl (PCB 167)
- 2,3,3',4,4',5,5'-Heptachlorobiphenyl (PCB 189)

Non-Dioxin-Like Polychlorinated Biphenyls (Pooled Samples)

- 2,4,4'-Trichlorobiphenyl (PCB 28)
- 2,2'3,5'-Tetrachlorobiphenyl (PCB 44)
- 2,2',4,5'-Tetrachlorobiphenyl (PCB 49)
- 2,2',5,5'-Tetrachlorobiphenyl (PCB 52)
- 2,3',4,4'-Tetrachlorobiphenyl (PCB 66)
- 2,4,4',5-Tetrachlorobiphenyl (PCB 74)
- 2,2',3,4,5'-Pentachlorobiphenyl (PCB 87)
- 2,2',4,4',5-Pentachlorobiphenyl (PCB 99)
- 2,2',4,5,5'-Pentachlorobiphenyl (PCB 101)
- 2,3,3',4',6-Pentachlorobiphenyl (PCB 110)
- 2,2',3,3',4,4'-Hexachlorobiphenyl (PCB 128)
- 2,2',3,4,4',5' and 2,3,3',4,4',6-Hexachlorobiphenyl (PCB 138 & 158)
- 2,2',3,4',5,5'-Hexachlorobiphenyl (PCB 146)
- 2,2',3,4',5',6-Hexachlorobiphenyl (PCB 149)
- 2,2',3,5,5',6-Hexachlorobiphenyl (PCB 151)
- 2,2',4,4',5,5'-Hexachlorobiphenyl (PCB 153)
- 2,2',3,3',4,4',5-Heptachlorobiphenyl (PCB 170)
- 2,2',3,3',4,5,5'-Heptachlorobiphenyl (PCB 172)
- 2,2',3,3',4,5',6'-Heptachlorobiphenyl (PCB 177)
- 2,2',3,3',5,5',6-Heptachlorobiphenyl (PCB 178)
- 2,2',3,4,4',5,5'-Heptachlorobiphenyl (PCB 180)
- 2,2',3,4,4',5',6-Heptachlorobiphenyl (PCB 183)
- 2,2',3,4',5,5',6-Heptachlorobiphenyl (PCB 187)
- 2,2',3,3',4,4',5,5'-Octachlorobiphenyl (PCB 194)
- 2,2',3,3',4,4',5,6-Octachlorobiphenyl (PCB 195)
- 2,2',3,3',4,4',5,6' and 2,2',3,4,4',5,5',6-Octachlorobiphenyl (PCB 196 & 203)
- 2,2',3,3',4,5,5',6-Octachlorobiphenyl (PCB 199)
- 2,2',3,3',4,4',5,5',6-Nonachlorobiphenyl (PCB 206)
- 2,2',3,3',4,4',5,5',6,6'-Decachlorobiphenyl (PCB 209)

The CDC Fourth Report 2015 updates are reported here for comparison with the 2013 updates and for your appreciation of the changes. More chemicals are found in the blood and urine of all tested individuals.

Endocrine-Disrupting Chemicals Are Largely Estrogenic

Organochlorine insecticides and the 12 persistent organic pollutants are estrogenic.

Phthalates are mostly estrogenic and have effect on the androgens (male hormone such as testosterone).

Some heavy metals are estrogenic (antimony, cadmium, cobalt, mercury, lead, and tin).

Glyphosate in Roundup is estrogenic.

Bisphenol A (found in some plastic containers such as plastic bottles, lining of canned foods, plastic wraps, baby bottles) and Bisphenol F are estrogenic.

There is, therefore, a plethora of estrogens in the world that has reached epidemic proportions. Note that most of the CDC's *Fourth Report* chemicals are estrogenic.

This estrogen epidemic around the world causes multiple symptoms, diseases, and human sufferings.

This epidemic is not fully recognized yet, and many governments are only targeting Bisphenol A to be banned.

There is a silent revolution in the U.S. today: many women are going gluten-free because wheat, barley, rye, spelt, and kamut are plant-based estrogens.

These grains cause estrogen-like symptoms of abdominal bloating, cramping, and diarrhea, even migraine headaches and mood swings.

These symptoms are similar to what some women experience in the late luteal phase of their menstrual cycle and late follicular phase, at the peak of their estradiol, which occurs just before ovulation, when the endogenous progesterone is lower than the estrogens (estradiol and estrones).

The following tables list the many chemicals found in wheat (with a review of the modes of action of some of these chemicals in humans), corn, barley, oats and rye, potatoes, rice, soybeans, and tree nuts.

Table 8.

Chemicals in Wheat

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	ATRAZINE	Spring wheat	65,192,074	21,343	<1	2009	ATRAZINE	Spring wheat	67,005,515	66,707	<1
2009	BROMOXYNIL	Spring wheat	2,003,964	1,261,637	62.96	2009	BROMOXYNIL	Spring wheat	2,586,820	1,277,750	49.39
2008	CARBOFURAN	Spring wheat	302,673	750	<1	2008	CARBOFURAN	Spring wheat	1,123,405	2,393	<1
2009	EPTC	Spring wheat	1,829,047	569	<1	2009	EPTC	Spring wheat	3,500,796	569	<1
2009	GLYPHOSATE	Spring wheat	219,724,312	4,148,299	1.89	2009	GLYPHOSATE	Spring wheat	222,444,180	4,182,043	1.88
2008	LINURON	Spring wheat	212,786	27,982	13.15	2008	LINURON	Spring wheat	349,078	89,272	25.57
2009	METHOMYL	Spring wheat	941,971	56	<1	2009	METHOMYL	Spring wheat	1,477,625	56	<1
2009	METHYL PARATHION	Spring wheat	312,631	57,078	18.26	2009	METHYL PARATHION	Spring wheat	975,383	198,751	20.38
2009	METOLACHLOR-S	Spring wheat	29,215,535	122	<1	2009	METOLACHLOR-S	Spring wheat	33,534,830	122	<1
2008	METRIBUZIN	Spring wheat	1,125,519	69	<1	2008	METRIBUZIN	Spring wheat	2,055,536	69	<1
2009	PROPANIL	Spring wheat	4,847,819	192	<1	2009	PROPANIL	Spring wheat	4,871,388	192	<1
2008	PROPARGITE	Spring wheat	1,036,080	133	<1	2008	PROPARGITE	Spring wheat	2,755,932	133	<1
2009	PROPICONAZOLE	Spring wheat	1,125,968	246,237	21.87	2009	PROPICONAZOLE	Spring wheat	1,552,077	251,737	16.22
2009	TRIALLATE	Spring wheat	188,294	19,869	10.55	2009	TRIALLATE	Spring wheat	625,113	118,312	18.93
2009	TRIFLURALIN	Spring wheat	5,201,075	23,591	<1	2009	TRIFLURALIN	Spring wheat	8,222,225	208,184	2.53

Chemicals in Wheat

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	ATRAZINE	Winter wheat	65,192,074	10,435	<1	2009	ATRAZINE	Winter wheat	67,005,515	142,446	<1
2009	BROMOXYNIL	Winter wheat	2,003,964	363,276	18.13	2009	BROMOXYNIL	Winter wheat	2,586,820	714,542	27.62
2009	CARBOFURAN	Winter wheat	865,095	8,169	<1	2009	CARBOFURAN	Winter wheat	2,123,383	157,851	7.43
2009	GLYPHOSATE	Winter wheat	219,724,312	5,862,177	2.67	2009	GLYPHOSATE	Winter wheat	222,444,180	6,600,249	2.97
2009	METHYL PARATHION	Winter wheat	312,631	21,020	6.72	2009	METHYL PARATI	Winter wheat	975,383	269,852	27.67
2009	METRIBUZIN	Winter wheat	1,388,578	6,487	<1	2009	METRIBUZIN	Winter wheat	2,078,312	48,890	2.35
2008	PHORATE	Winter wheat	1,011,005	98,356	9.73	2008	PHORATE	Winter wheat	1,670,449	415,202	24.86
2009	PROPICONAZOLE	Winter wheat	1,125,968	201,975	17.94	2009	PROPICONAZOI	Winter wheat	1,552,077	326,837	21.06
2009	TRIALLATE	Winter wheat	188,294	22,995	12.21	2009	TRIALLATE	Winter wheat	625,113	275,610	44.09
2009	TRIFLURALIN	Winter wheat	5,201,075	6,899	<1	2009	TRIFLURALIN	Winter wheat	8,222,225	82,689	1.01

Table 9. Chemical Class, Mode of Action, and Effects on Humans

Year	Pesticide	Crop	PESTICIDES CLASS	PESTICIDES MODE OF ACTION	EFFECT ON HUMANS
2009	ATRAZINE	Spring wheat	TRIAZINE	AROMATASE BOOSTER	Testosterone/estrogen
2009	BROMOXYNIL	Spring wheat	HYDROXYBENZONITRILE		
2009	CARBOFURAN	Spring wheat	N-METHYL-CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	EPTC	Spring wheat	THIOCARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	GLYPHOSATE	Spring wheat	PHOSPHONOGLYCINE	EDC	Estrogenic/reproductive
2009	LINURON	Spring wheat	SUBSTITUTED UREA		
2009	METHOMYL	Spring wheat	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METHYL PARATHION	Spring wheat	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METOLACHLOR-S	Spring wheat	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	METRIBUZIN	Spring wheat	SELECTIVE TRIAZINE	EDC	Testosterone/estrogen
2009	PROPANIL	Spring wheat	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PROPARGITE	Spring wheat	UNCLASSIFIED	PAN BAD ACTOR	
2009	PROPICONAZOLE	Spring wheat	TRIAZOLE	PAN BAD ACTOR	
2009	TRIALLATE	Spring wheat	THIOCARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	TRIFLURALIN	Spring wheat	2,6-DINITROANILINE		

Table 10

Chemicals in Cotton

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide use by crop, low, in pounds	Percentage of EPest-low usage estimate applied to crop
2009	BROMOXYNIL	Cotton	2,003,964	43	<1
2009	FLUOMETURON	Cotton	301,242	301,242	100.00
2009	GLYPHOSATE	Cotton	219,724,312	14,392,772	6.55
2009	LINURON	Cotton	172,434	24,164	14.01
2009	METHOMYL	Cotton	941,971	5,975	<1
2009	METHYL PARATHION	Cotton	312,631	19,751	6.32
2009	METOLACHLOR	Cotton	3,545,458	203,119	5.73
2009	METOLACHLOR-S	Cotton	29,215,535	862,640	2.95
2009	NORFLURAZON	Cotton	664,593	8,499	1.28
2009	OXAMYL	Cotton	484,832	110,128	22.71
2009	PHORATE	Cotton	746,198	25,674	3.44
2009	PROPARGITE	Cotton	604,823	2,319	<1
2009	TRIFLURALIN	Cotton	5,201,075	2,458,241	47.26
2008	FLUOMETURON	Cotton	290,122	290,122	100.00
2008	GLYPHOSATE	Cotton	212,705,214	13,975,066	6.57
2008	LINURON	Cotton	212,786	44,470	20.90
2008	METHOMYL	Cotton	916,997	2,873	<1
2008	METHYL PARATHION	Cotton	412,495	8,013	1.94
2008	METOLACHLOR	Cotton	2,574,583	281,044	10.92
2008	METOLACHLOR-S	Cotton	28,453,494	661,521	2.32
2008	NORFLURAZON	Cotton	343,647	5,296	1.54
2008	OXAMYL	Cotton	857,837	245,101	28.57
2008	PHORATE	Cotton	1,011,005	69,304	6.85
2008	PROPARGITE	Cotton	1,036,080	19,656	1.90
2008	TRIFLURALIN	Cotton	5,082,337	2,280,668	44.87

Table 11

Chemical Class, Mode of Action and Effect on Humans

Year	Pesticide	Crop	PESTICIDES CLASS	PESTICIDES MODE OF ACTION	EFFECT ON HUMANS
2009	BROMOXYNIL	COTTON	HYDROXYBENZONITRILE	PAN BAD ACTOR	Cancer/Develop/Reprod
2009	FLUOMETURON	COTTON	UREA	Acetylcholine	Insulin/mast cells/Allergy
2009	GLYPHOSATE	COTTON	PHOSPHONOGLYCINE	EDC	Estrogenic/reproductive
2009	LINURON	COTTON	SUBSTITUTED UREA	Acetylcholine	Insulin/mast cells/Allergy
2009	METHOMYL	COTTON	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METHYL PARATHION	COTTON	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METOLACHLOR	COTTON	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	METOLACHLOR-S	COTTON	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	NORFLURAZON	COTTON	PYRIDAZINONE	EDC	Testosterone/estrogen
2009	OXAMYL	COTTON	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PHORATE	COTTON	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PROPARGITE	COTTON	UNCLASSIFIED	PAN BAD ACTOR	Cancer/Develop/Reprod
2009	TRIFLURALIN	COTTON	2,6-DINITROANILINE		Headache/Dizzy/MetHgb

Table 12

Chemicals in Peanuts

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide use by crop, low, in pounds	Percentage of EPest-low usage estimate applied to crop
2009	ACIFLUORFEN	Peanuts	155,619	49,275	31.66
2009	ALACHLOR	Peanuts	2,421,529	818	<1
2009	BENTAZON	Peanuts	1,184,951	116,172	9.80
2009	CHLORIMURON	Peanuts	104,968	116	<1
2009	ETHALFLURALIN	Peanuts	1,277,080	265,942	20.82
2009	GLYPHOSATE	Peanuts	219,724,312	232,498	<1
2009	METHOMYL	Peanuts	941,971	15,081	1.60
2009	METOLACHLOR	Peanuts	3,545,458	20,481	<1
2009	METOLACHLOR-S	Peanuts	29,215,535	392,034	1.34
2009	OXAMYL	Peanuts	484,832	11,547	2.38
2009	PHORATE	Peanuts	746,198	212,190	28.44
2009	PROPARGITE	Peanuts	604,823	1	<1
2009	PROPICONAZOLE	Peanuts	1,125,968	37,720	3.35
2009	TRIFLURALIN	Peanuts	5,201,075	24,973	<1
2008	ACIFLUORFEN	Peanuts	69,495	34,655	49.87
2008	ALACHLOR	Peanuts	1,718,157	882	<1
2008	BENTAZON	Peanuts	1,110,676	107,641	9.69
2008	CHLORIMURON	Peanuts	107,446	97	<1
2008	ETHALFLURALIN	Peanuts	1,265,891	178,405	14.09
2008	GLYPHOSATE	Peanuts	212,705,214	307,269	<1
2008	METHOMYL	Peanuts	916,997	17,254	1.88
2008	METOLACHLOR	Peanuts	2,574,583	22,686	<1
2008	METOLACHLOR-S	Peanuts	28,453,494	414,309	1.46
2008	PHORATE	Peanuts	1,011,005	331,054	32.75
2008	PROPANIL	Peanuts	4,248,406	3,904	<1
2008	PROPARGITE	Peanuts	1,036,080	6	<1
2008	PROPICONAZOLE	Peanuts	1,489,774	71,086	4.77
2008	TRIFLURALIN	Peanuts	5,082,337	52,453	1.03

Table 13

Chemical Class, Mode of Action and Effect on Humans

Year	Pesticide	Crop	PESTICIDES CLASS	PESTICIDES MODE OF ACTION	EFFECT ON HUMANS
2009	ACIFLUORFEN	PEANUTS	DIPHENYL ETHER	PAN BAD ACTOR	Cancer
2009	ALACHLOR	PEANUTS	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	BENTAZON	PEANUTS	UNCLASSIFIED		
2009	CHLORIMURON	PEANUTS	**SULFONYLUREA**	EDC	INSULIN/CARDIAC EVENTS
2009	ETHALFLURALIN	PEANUTS	2,6-DINITROANILINE		Headache/Dizzy/MetHgb
2009	GLYPHOSATE	PEANUTS	PHOSPHONOGLYCINE	EDC	Estrogenic/reproductive
2009	METHOMYL	PEANUTS	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	METOLACHLOR	PEANUTS	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	METOLACHLOR-S	PEANUTS	CHLORACETAMIDE	EDC	thyroid/nasal turbinates
2009	OXAMYL	PEANUTS	CARBAMATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PHORATE	PEANUTS	ORGANOPHOSPHATE	Acetylcholine	Insulin/mast cells/Allergy
2009	PROPARGITE	PEANUTS	UNCLASSIFIED	PAN BAD ACTOR	Cancer/Develop/Reprod
2009	PROPICONAZOLE	PEANUTS	TRIAZOLE	PAN BAD ACTOR	
2009	TRIFLURALIN	PEANUTS	2,6-DINITROANILINE		Headache/Dizzy/MetHgb

- You see that Glyphosate is used in both cotton and peanut production in addition to other estrogenic chemicals.
- As noted in the nasal allergy pathophysiology, estrogens have receptors on mast cells and basophils that directly affect degranulation of these cells. The estrogens also make the effect of IgE stronger. It is, therefore, possible that the increased estrogen load in our environment may be one of the reasons for the growing food and environmental allergies.
- Many of the chemicals used in both cotton and peanut production are organophosphates and carbamates that tend to cause an increase in acetylcholine.
- Recall that acetylcholine has receptors on mast cells and basophils and can cause a degranulation of these cells to cause allergic reactions.
- Acetylcholine also causes insulin release that leads to mast cell and basophil proliferation and, hence, increase allergic reactions.
- You can see that the synergistic effects of these chemicals may explain not only the growing allergy epidemic, but also the growing obesity epidemic and growing obesity comorbidities worldwide.

Table 14

Chemicals in Corn 2008-2009

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide use by crop, low, in	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by crop, high, in	Percentage of EPest-high
2009	ACETOCHLOR	Corn	28,331,622	28,082,750	99.12	2009	ACETOCHI	Corn	30,681,109	30,163,034	98.31
2009	ALACHLOR	Corn	2,421,529	1,465,241	60.51	2009	ALACHLOI	Corn	7,138,070	4,475,714	62.70
2009	ATRAZINE	Corn	65,192,074	57,285,676	87.87	2009	ATRAZINE	Corn	67,005,515	57,776,134	86.23
2009	BENTAZON	Corn	1,184,951	47,160	3.98	2009	BENTAZOI	Corn	1,970,758	470,095	23.85
2009	BROMOXYNIL	Corn	2,003,964	32,638	1.63	2009	BROMOXY	Corn	2,586,820	211,893	8.19
2009	BUTYLATE	Corn	29,240	8,697	29.74	2009	BUTYLATE	Corn	154,864	47,355	30.58
2009	CARBOFURAN	Corn	865,095	361,537	41.79	2009	CARBOFUI	Corn	2,123,383	579,038	27.27
2008	CYANAZINE	Corn	114,541	114,541	100.00	2008	CYANAZIN	Corn	1,460,951	1,460,951	100.00
2009	EPTC	Corn	1,829,047	43	<1	2009	EPTC	Corn	3,500,796	43	<1
2009	ETHALFLURALIN	Corn	1,277,080	183	<1	2009	ETHALFLU	Corn	1,690,635	183	<1
2009	GLYPHOSATE	Corn	219,724,312	69,213,738	31.50	2009	GLYPHOSA	Corn	222,444,180	69,378,517	31.19
2009	LINURON	Corn	172,434	12,749	7.39	2009	LINURON	Corn	356,079	123,369	34.65
2009	METHOMYL	Corn	941,971	47,581	5.05	2009	METHOM\	Corn	1,477,625	47,581	3.22
2009	METHYL PARATHION	Corn	312,631	55,318	17.69	2009	METHYL P.	Corn	975,383	126,801	13.00
2009	METOLACHLOR	Corn	3,545,458	2,203,490	62.15	2009	METOLACI	Corn	10,450,056	6,587,596	63.04
2009	METOLACHLOR-S	Corn	29,215,535	22,238,799	76.12	2009	METOLACI	Corn	33,534,830	23,361,577	69.66
2008	METRIBUZIN	Corn	1,125,519	15,108	1.34	2008	METRIBUZ	Corn	2,055,536	135,080	6.57
2009	NICOSULFURON	Corn	36,501	35,879	98.30	2009	NICOSULF	Corn	73,141	72,269	98.81
2009	PHORATE	Corn	746,198	10,260	1.37	2009	PHORATE	Corn	908,117	33,226	3.66
2009	PROPARGITE	Corn	604,823	366,761	60.64	2009	PROPARG	Corn	3,310,430	3,027,592	91.46
2009	PROPICONAZOLE	Corn	1,125,968	245,176	21.77	2009	PROPICOI	Corn	1,552,077	395,432	25.48
2009	TERBUFOS	Corn	640,805	422,273	65.90	2009	TERBUFOS	Corn	1,864,375	1,487,460	79.78
2009	TRIFLURALIN	Corn	5,201,075	373	<1	2009	TRIFLURAI	Corn	8,222,225	373	<1

Corn is estrogenic by contamination only. Atrazine and glyphosate are estrogenic; alachlor, acetochlor, metolachlor and metolachlor-S are thyroid disruptors and also make nasal turbinates larger (they involved in nasal allergies). Avoiding GMO corn products may alleviate nasal symptoms and help in weight control.

Table 15

Chemicals in Barley

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	BROMOXYNIL	Barley	2,003,964	252,614	12.61	2009	BROMOXYNIL	Barley	2,586,820	270,006	10.44
2009	GLYPHOSATE	Barley	219,724,312	573,993	<1	2009	GLYPHOSATE	Barley	222,444,180	675,508	<1
2008	METHOMYL	Barley	916,997	28	<1	2008	METHOMYL	Barley	1,290,152	28	<1
2008	METHYL PARA	Barley	412,495	111	<1	2008	METHYL PARATH	Barley	1,334,581	111	<1
2009	METRIBUZIN	Barley	1,388,578	234	<1	2009	METRIBUZIN	Barley	2,078,312	2,397	<1
2009	PROPICONAZ(	Barley	1,125,968	1	<1	2009	PROPICONAZOL	Barley	1,552,077	1	<1
2009	TRIALLATE	Barley	188,294	89,082	47.31	2009	TRIALLATE	Barley	625,113	143,263	22.92
2008	TRIFLURALIN	Barley	5,082,337	3,272	<1	2008	TRIFLURALIN	Barley	7,697,724	24,522	<1

Barley is a phytoestrogen and has a major contaminant Triallate which causes acetylcholine release. Recall that acetylcholine causes insulin release and this may be contributing to the ventral obesity seen in beer drinkers.

Table 16

Chemicals in Oats and Rye

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	BROMOXYNIL	Oats and rye	2,003,964	6,302	<1	2009	BROMOXYNIL	Oats and rye	2,586,820	6,302	<1
2007	CARBOFURAN	Oats and rye	512,893	53	<1	2007	CARBOFURAN	Oats and rye	2,198,171	53	<1
2009	GLYPHOSATE	Oats and rye	219,724,312	3,094	<1	2009	GLYPHOSATE	Oats and rye	222,444,180	3,094	<1
2008	METHOMYL	Oats and rye	916,997	121	<1	2008	METHOMYL	Oats and rye	1,290,152	121	<1
2009	METHYL PARATHION	Oats and rye	312,631	10	<1	2009	METHYL PARATHION	Oats and rye	975,383	10	<1
2007	NICOSULFURON	Oats and rye	104,555	1	<1	2007	NICOSULFURON	Oats and rye	121,644	1	<1
2008	TRIFLURALIN	Oats and rye	5,082,337	88	<1	2008	TRIFLURALIN	Oats and rye	7,697,724	88	<1

Notice that oat and rye use less pesticides and that is the reason many individuals can tolerate gluten-free oats.

Table 17

Chemicals in Potatoes

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	CARBOFURAN	Potatoes	865,095	731	<1	2009	CARBOFURAN	Potatoes	2,123,383	1,062	<1
2009	EPTC	Potatoes	1,829,047	844,541	46.17	2009	EPTC	Potatoes	3,500,796	952,812	27.22
2009	ETHALFLURALIN	Potatoes	1,277,080	939	<1	2009	ETHALFLURALIN	Potatoes	1,690,635	939	<1
2009	ETHOPROPHOS	Potatoes	346,912	76,416	22.03	2009	ETHOPROPHOS	Potatoes	501,306	138,418	27.61
2009	GLYPHOSATE	Potatoes	219,724,312	113,181	<1	2009	GLYPHOSATE	Potatoes	222,444,180	139,737	<1
2009	LINURON	Potatoes	172,434	51,122	29.65	2009	LINURON	Potatoes	356,079	66,902	18.79
2009	METHOMYL	Potatoes	941,971	15,227	1.62	2009	METHOMYL	Potatoes	1,477,625	90,593	6.13
2009	METHYL PARATHION	Potatoes	312,631	487	<1	2009	METHYL PARATHION	Potatoes	975,383	867	<1
2009	METOLACHLOR	Potatoes	3,545,458	106,242	3.00	2009	METOLACHLOR	Potatoes	10,450,056	297,803	2.85
2009	METOLACHLOR-S	Potatoes	29,215,535	186,270	<1	2009	METOLACHLOR-S	Potatoes	33,534,830	241,867	<1
2009	METRIBUZIN	Potatoes	1,388,578	357,918	25.78	2009	METRIBUZIN	Potatoes	2,078,312	364,257	17.53
2009	OXAMYL	Potatoes	484,832	202,813	41.83	2009	OXAMYL	Potatoes	652,059	273,192	41.90
2009	PHORATE	Potatoes	746,198	68,541	9.19	2009	PHORATE	Potatoes	908,117	101,709	11.20
2009	PROPARGITE	Potatoes	604,823	40,487	6.69	2009	PROPARGITE	Potatoes	3,310,430	68,877	2.08
2009	TRIFLURALIN	Potatoes	5,201,075	8,693	<1	2009	TRIFLURALIN	Potatoes	8,222,225	28,396	<1

Potatoes have 9 major contaminants and most are organophosphates, carbamates or fungicides. Organophosphates and carbamates cause acetylcholine release. Recall the effects of acetylcholine excess on humans.

Table 18

Chemicals in Rice

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	ACIFLUORFEN	Rice	155,619	4,639	2.98	2009	ACIFLUORFEN	Rice	385,298	7,283	1.89
2009	BENTAZON	Rice	1,184,951	3,441	<1	2009	BENTAZON	Rice	1,970,758	8,769	<1
2008	BROMOXYNIL	Rice	2,200,325	16	<1	2008	BROMOXYNIL	Rice	3,034,629	16	<1
2009	GLYPHOSATE	Rice	219,724,312	825,594	<1	2009	GLYPHOSATE	Rice	222,444,180	831,077	<1
2009	METHYL PARATHION	Rice	312,631	25,424	8.13	2009	METHYL PARATHION	Rice	975,383	46,442	4.76
2009	PROPANIL	Rice	4,847,819	4,847,362	99.99	2009	PROPANIL	Rice	4,871,388	4,870,930	99.99
2009	PROPICONAZOLE	Rice	1,125,968	152,912	13.58	2009	PROPICONAZOLE	Rice	1,552,077	154,517	9.96
2009	THIOBENCARB	Rice	494,951	494,783	99.97	2009	THIOBENCARB	Rice	650,515	650,347	99.97
2007	TRIFLURALIN	Rice	6,084,681	2	<1	2007	TRIFLURALIN	Rice	10,568,360	2	<1

Rice has 5 major contaminants and the carbamate insecticide Thiobencarb and organophosphate insecticide Propanil are the most significant. The carbamate coupled with the organophosphates Methyl Parathion and Propanil can lead to acetycholine increase, insulin increase, craving for sweets, and obesity and its comorbidities may follow. Propiconazole is a fungicide. There was also the story about arsenic contamination of rice and it is recommended to wash your rice thoroughly before cooking it.

Table 19

Chemicals in Soybeans

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide use by crop, low, in pounds	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by crop, high, in pounds	Percentage of EPest-high
2009	ACIFLUORFEN	Soybeans	155,619	101,424	65.17	2009	ACIFLUORFEN	Soybeans	385,298	318,277	82.61
2009	ALACHLOR	Soybeans	2,421,529	277,577	11.46	2009	ALACHLOR	Soybeans	7,138,070	1,376,354	19.28
2009	BENTAZON	Soybeans	1,184,951	117,897	9.95	2009	BENTAZON	Soybeans	1,970,758	435,745	22.11
2009	CARBOFURAN	Soybeans	865,095	15,929	1.84	2009	CARBOFURAN	Soybeans	2,123,383	159,240	7.50
2009	CHLORIMURON	Soybeans	104,968	104,852	99.89	2009	CHLORIMURON	Soybeans	115,338	115,074	99.77
2009	ETHALFLURALIN	Soybeans	1,277,080	30,623	2.40	2009	ETHALFLURALIN	Soybeans	1,690,635	157,624	9.32
2009	GLYPHOSATE	Soybeans	219,724,312	96,970,922	44.13	2009	GLYPHOSATE	Soybeans	222,444,180	96,996,656	43.60
2007	LINURON	Soybeans	291,094	5,246	1.80	2007	LINURON	Soybeans	891,383	32,053	3.60
2008	METHOMYL	Soybeans	916,997	8,363	<1	2008	METHOMYL	Soybeans	1,290,152	53,449	4.14
2009	METHYL PARATHION	Soybeans	312,631	21,746	6.96	2009	METHYL PARATHION	Soybeans	975,383	109,306	11.21
2009	METOLACHLOR	Soybeans	3,545,458	263,205	7.42	2009	METOLACHLOR	Soybeans	10,450,056	1,916,314	18.34
2009	METOLACHLOR-S	Soybeans	29,215,535	2,538,210	8.69	2009	METOLACHLOR-S	Soybeans	33,534,830	4,829,692	14.40
2009	METRIBUZIN	Soybeans	1,388,578	445,120	32.06	2009	METRIBUZIN	Soybeans	2,078,312	687,816	33.09
2009	PROPICONAZOLE	Soybeans	1,125,968	135,255	12.01	2009	PROPICONAZOLE	Soybeans	1,552,077	251,074	16.18
2009	TRIFLURALIN	Soybeans	5,201,075	1,331,398	25.60	2009	TRIFLURALIN	Soybeans	8,222,225	2,114,350	25.72

Soybean is a major phytoestrogen and it is also contaminated with glyphosate (estrogenic), thyroid disruptors (alachlor, metolachlor, metolachlor-S, acifluorfen) and cancer causing agent (Acifluorfen). Chlorimuron causes an increase in insulin and you already know the effects of too much insulin in the body. Trifluralin causes headaches and dizziness and Metribuzin increases estrogen and decreases testosterone. Avoid soy products to improve your health.

Table 20

Chemicals in Lettuce

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	GLYPHOSATE	Lettuce, all	219,724,312	8,634	<1	2009	GLYPHOSATE	Lettuce, a	222,444,180	8,634	<1
2009	LINURON	Lettuce, all	172,434	19	<1	2009	LINURON	Lettuce, a	356,079	19	<1
2009	METHOMYL	Lettuce, all	941,971	90,903	9.65	2009	METHOMYL	Lettuce, a	1,477,625	90,903	6.15
2009	METHYL PARATHION	Lettuce, all	312,631	127	<1	2009	METHYL PARATHION	Lettuce, a	975,383	127	<1
2009	PROPYZAMIDE	Lettuce, all	107,721	104,522	97.03	2009	PROPYZAMIDE	Lettuce, a	107,998	104,522	96.78
2008	TRIFLURALIN	Lettuce, all	5,082,337	0	<1	2008	TRIFLURALIN	Lettuce, a	7,697,724	0	<1

Table 21

Chemicals in Other Tree Nuts

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2008	ALACHLOR	Nut trees, other	1,718,157	3	<1	2008	ALACHLOR	Nut trees, other	5,220,252	3	<1
2009	ATRAZINE	Nut trees, other	65,192,074	1	<1	2009	ATRAZINE	Nut trees, other	67,005,515	1	<1
2008	BENOMYL	Nut trees, other	3,357	58	1.72	2008	BENOMYL	Nut trees, other	4,560	58	1.26
2009	BENTAZON	Nut trees, other	1,184,951	1	<1	2009	BENTAZON	Nut trees, other	1,970,758	1	<1
2009	BROMOXYNIL	Nut trees, other	2,003,964	175	<1	2009	BROMOXYNIL	Nut trees, other	2,586,820	175	<1
2008	CARBOFURAN	Nut trees, other	302,673	738	<1	2008	CARBOFURAN	Nut trees, other	1,123,405	738	<1
2009	ETHOPROPHOS	Nut trees, other	346,912	181	<1	2009	ETHOPROPHOS	Nut trees, other	501,306	181	<1
2009	GLYPHOSATE	Nut trees, other	219,724,312	365,547	<1	2009	GLYPHOSATE	Nut trees, other	222,444,180	365,547	<1
2009	LINURON	Nut trees, other	172,434	534	<1	2009	LINURON	Nut trees, other	356,079	534	<1
2009	METHOMYL	Nut trees, other	941,971	34	<1	2009	METHOMYL	Nut trees, other	1,477,625	34	<1
2009	METHYL PARATHION	Nut trees, other	312,631	0	<1	2009	METHYL PARATHION	Nut trees, other	975,383	0	<1
2009	METOLACHLOR	Nut trees, other	3,545,458	8	<1	2009	METOLACHLOR	Nut trees, other	10,450,056	8	<1
2009	METOLACHLOR-S	Nut trees, other	29,215,535	1,053	<1	2009	METOLACHLOR-S	Nut trees, other	33,534,830	1,053	<1
2009	METRIBUZIN	Nut trees, other	1,388,578	1	<1	2009	METRIBUZIN	Nut trees, other	2,078,312	1	<1
2009	NORFLURAZON	Nut trees, other	664,593	156	<1	2009	NORFLURAZON	Nut trees, other	745,805	156	<1
2009	ORYZALIN	Nut trees, other	627,479	70,044	11.16	2009	ORYZALIN	Nut trees, other	674,219	70,091	10.40
2009	OXAMYL	Nut trees, other	484,832	33	<1	2009	OXAMYL	Nut trees, other	652,059	33	<1
2009	PHORATE	Nut trees, other	746,198	260	<1	2009	PHORATE	Nut trees, other	908,117	260	<1
2009	PROPARGITE	Nut trees, other	604,823	886	<1	2009	PROPARGITE	Nut trees, other	3,310,430	886	<1
2009	PROPICONAZOLE	Nut trees, other	1,125,968	8,938	<1	2009	PROPICONAZOLE	Nut trees, other	1,552,077	8,978	<1
2009	PROPYZAMIDE	Nut trees, other	107,721	4	<1	2009	PROPYZAMIDE	Nut trees, other	107,998	4	<1
2009	THIOBENCARB	Nut trees, other	494,951	3	<1	2009	THIOBENCARB	Nut trees, other	650,515	3	<1
2009	TRIFLURALIN	Nut trees, other	5,201,075	3,059	<1	2009	TRIFLURALIN	Nut trees, other	8,222,225	3,059	<1

Table 22

Chemicals in Apples

Year	Pesticide	Crop	Estimated total annual use, low, in pounds	Estimated total annual pesticide	Percentage of EPest-low	Year	Pesticide	Crop	Estimated total annual use, high, in pounds	Estimated total annual use by	Percentage of EPest-high
2009	BENOMYL	Apples	1,717	0	<1	2009	BENOMYL	Apples	3,110	0	<1
2009	GLYPHOSATE	Apples	219,724,312	472,508	<1	2009	GLYPHOSATE	Apples	222,444,180	482,867	<1
2009	METHOMYL	Apples	941,971	26,386	2.80	2009	METHOMYL	Apples	1,477,625	44,035	2.98
2009	NORFLURAZON	Apples	664,593	72,863	10.96	2009	NORFLURAZON	Apples	745,805	116,875	15.67
2009	ORYZALIN	Apples	627,479	73,821	11.76	2009	ORYZALIN	Apples	674,219	86,672	12.86
2009	OXAMYL	Apples	484,832	7,307	1.51	2009	OXAMYL	Apples	652,059	21,433	3.29
2007	PROPARGITE	Apples	833,444	1,298	<1	2007	PROPARGITE	Apples	2,970,502	8,823	<1
2009	PROPICONAZOLE	Apples	1,125,968	105	<1	2009	PROPICONAZOLE	Apples	1,552,077	568	<1
2009	PROPYZAMIDE	Apples	107,721	177	<1	2009	PROPYZAMIDE	Apples	107,998	454	<1
2009	TERBACIL	Apples	28,300	9,925	35.07	2009	TERBACIL	Apples	119,198	38,935	32.66
2008	TRIFLURALIN	Apples	5,082,337	0	<1	2008	TRIFLURALIN	Apples	7,697,724	0	<1

Apples are contaminated by Terbacil, Oryzalin, Norflurazon, Methomyl, and Oxamyl. We should, therefore, say an apple a day keeps the doctor away if organic.

PART IID: PUTTING IT ALL TOGETHER: HOW EXOGENOUS ESTROGENS LEAD TO HORMONE IMBALANCE, AND HOW HORMONE IMBALANCE LEADS TO OBESITY AND ALLERGIC DISEASES

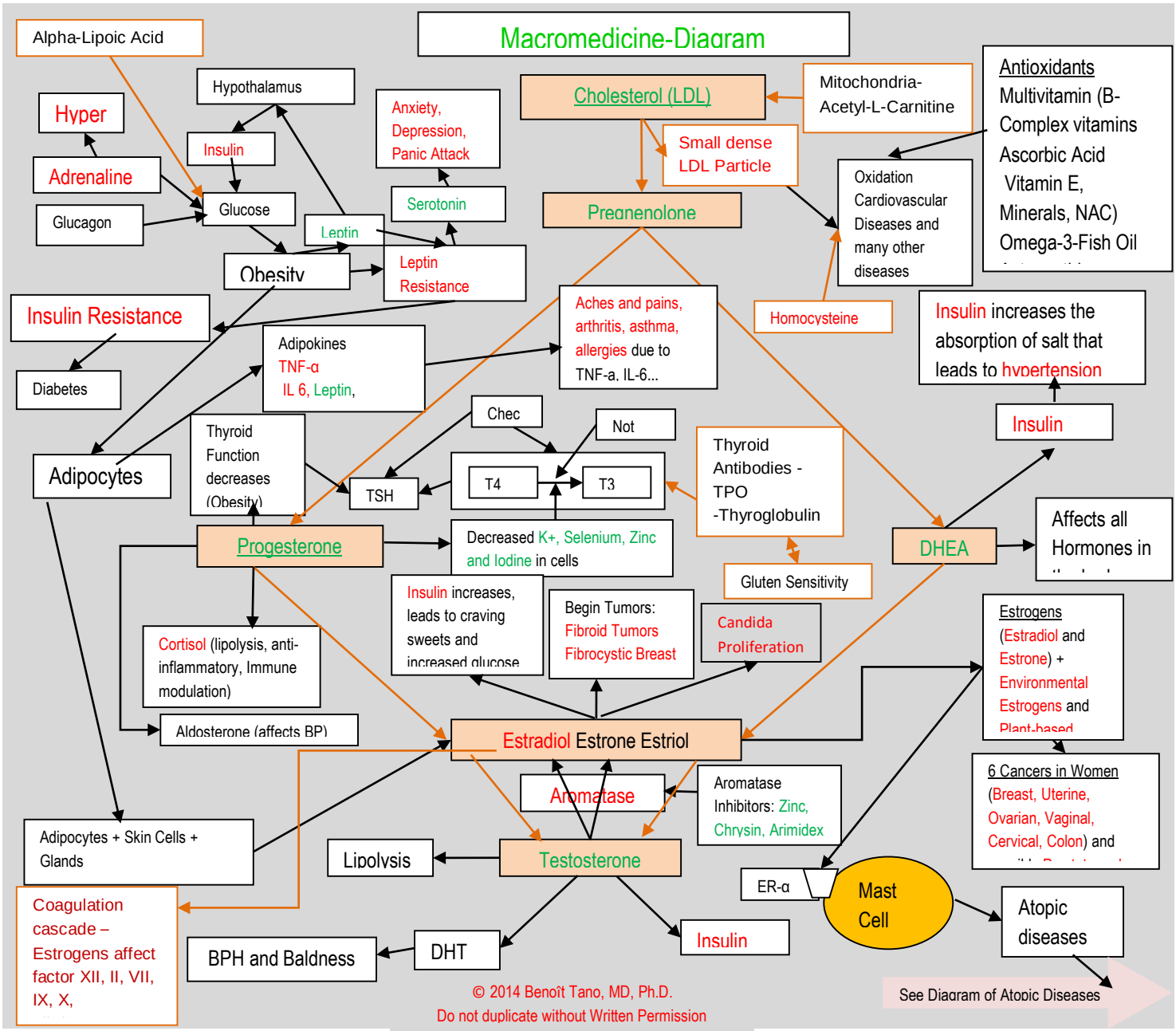
INTRODUCTION: THE CONNECTION BETWEEN THE ESTROGEN EPIDEMIC AND THE OBESITY AND ALLERGY EPIDEMICS

As we have seen in the previous sections, the endocrine system is intimately linked with the immune system; therefore, when hormones are out of balance, immunity is compromised. The *Integrative Immunity* analysis points to the estrogen epidemic as one of the major causes and driving forces of most common diseases. It is necessary for all of us to understand how our environment and foods have been contaminated by endocrine-disrupting chemicals, so we can learn how to build a healthier lifestyle.

USING THE MACROMEDICINE DIAGRAM AS A GUIDE TO UNDERSTANDING THE ROLE OF HORMONE IMBALANCE IN DISEASE AND OBESITY

Study the Macromedicine Diagram to understand the relationship of hormone imbalance syndrome on various diseases and obesity.

Figure 23. The Macromedicine Diagram



THE IMPORTANCE OF PROGESTERONE

Begin reading the Macromedicine Diagram from the middle, left side, starting with progesterone. Both men and women produce progesterone. In men, progesterone is produced by the adrenal glands and testis. In women, it is produced during the menstrual cycle by the ovaries and, in small amount, by the adrenal glands. The

progesterone gene is inherited from the mother, and low progesterone in the mother can lead to low progesterone in the daughter or son.

When girls inherit the low-progesterone gene, they develop progesterone deficiency symptoms early in life. Right after menarche (onset of the menstrual cycle), these girls start experiencing menorrhagia (heavy menstrual periods) and/or dysmenorrhea (painful menstrual cramps), often accompanied by nausea, vomiting, and diarrhea. This is quite debilitating for many young girls around the world. As adults, these women often have difficulty conceiving: and, if they do conceive, they have difficulty carrying the pregnancy to term. They tend to have multiple miscarriages. If an astute OB/GYN physician realizes that low progesterone is the problem and administers progesterone to the pregnant woman, this significantly increases the chances that the pregnancy will be carried to term.

If a woman with low progesterone becomes pregnant, the first trimester could represent a real challenge. She may have nausea and vomiting throughout the pregnancy, but these symptoms are worse in the first trimester when progesterone production remains average. During the second trimester, progesterone is produced by the placenta at a rate of about 400 mg/day, and this increase in progesterone helps to alleviate the nausea and vomiting. Once the woman has delivered and the placenta is expelled, progesterone production ceases and the woman may become depressed. This may explain much of the postpartum depression experienced by women soon after giving birth. Low progesterone also causes many of the symptoms seen in estrogen dominance, because progesterone deficiency is often accompanied by estrogen dominance.

Progesterone deficiency includes some of these common effects:

Cortisol initially decreases.
Thyroid function decreases.
TSH measure is insufficient to determine thyroid function.
T4 does not always convert to T3 (T4 may convert to a storage form called reverse T3 (rT3). Make sure your healthcare practitioner measures free T4, free T3, reverse T3, TSH, and thyroid antibodies—thyroglobulin and thyroid peroxidase antibodies—in your thyroid function test).
Development of estrogen dominance.
Estrogen dominance leads to increased insulin.
Increased insulin, initially, causes hypoglycemic episodes.
Adrenaline is then produced to increase glucose.
Glucagon is also produced to increase glucose.

Carbohydrate consumption increases due to sugar cravings, creating more glucose

Insulin pushes the excess glucose into adipocytes (fat cells).

Saturated adipocytes produce leptin (a messenger to the hypothalamus), adiponectin (a fat - reducing chemical), visfatin, and apelin.

Lepin signals the hypothalamus that adipocytes are saturated with glucose.

Too much leptin, however, overwhelms the hypothalamus, which stops responding to signals from the leptin.

Excess leptin is known as leptin resistance

Leptin resistance causes a decrease of serotonin (there is an inverse relationship between leptin and serotonin)

Low serotonin causes depression, anxiety, panic attacks, obsessive-compulsive disorder (OCD), bipolar disorder, and many so-called mental disorders

Leptin resistance leads to insulin resistance

Insulin resistance leads to type 2 diabetes

Leptin resistance is related to ghrelin and NPY (Neuropeptide Y, a neurotransmitter in the brain)

Ghrelin is the hunger hormone, and we tend to eat a lot when ghrelin is abundant

Adipocytes make inflammatory cytokines such as IL-6 and TNF-alpha, which cause aches and pains as well as allergic reactions such as adult-onset nasal allergies and asthma

Adipocytes help skin cells and adrenal glands to produce estradiol and estrone

Adipocytes produce aromatase, which converts testosterone to estrogens; hence, obese individuals may have low testosterone and estrogen dominance

Estrogens increase thyroid-binding globulin (TBG), which reduces thyroid function (if you are obese, then you have an underactive thyroid due to the high TBG)

Estrogen has receptors on mast cells and basophils and causes mediators such as histamine and leukotrienes to be released. These mediators cause allergic reactions including urticaria (hives) and angioedema. This is the reason these conditions are more prevalent in women.

In women, low progesterone is associated with the following symptoms:

Dysmenorrhea
Menorrhagia
Infertility
Anovulatory menstrual cycles
Irregular periods
Endometriosis
Early miscarriage
Carbohydrate cravings (due to estrogen dominance)
Breast tenderness (due to estrogen dominance)
Ovarian cysts (due to estrogen dominance)
Puffiness/bloating (due to estrogen dominance)
Water retention (due to estrogen dominance)
Lower body temperature (due to ineffective thyroid function; low iodine and low iron may be implicated here)
Internally anxious, outwardly calm
Premenstrual backache (due to estrogen dominance)
Frequent complaining (never happy about any situation; negative thinking)
Excessive crying (due to estrogen dominance)
Fibrocystic breasts (due to estrogen dominance)
Defective luteal phase
Fibroid tumors (due to estrogen dominance)
Excessive facial and body hair (androgen excess due to estrogen dominance and ovarian cysts that produce androgens)
Feeling of loneliness
Unjustified fear

Even if a woman does not have low progesterone early in life, she may develop progesterone deficiency because of anovulatory menstrual cycles as reported earlier in the book. Women tend to produce progesterone after ovulation. When women ovulate, the pocket from which the egg was released does not disappear. That pocket begins to produce progesterone. The pocket becomes so engorged with progesterone that it changes color and becomes yellow. It is, therefore, often called the "yellow body" or *corpus luteum* in Latin.

Because of the activities of the *corpus luteum,* the second half of the menstrual cycle (ovulation to start of the period) is known as the luteal phase. Progesterone production in the luteal phase peaks around day 21 of the cycle (count from first day of menstrual bleeding to day 21) in women who have a normal cycle of 28 days.

If a woman suspects that she has low progesterone and would like to test it, this test (a spot blood test performed in a lab) should be done at day 21. If she has low progesterone at the peak of her progesterone production, she definitely has low progesterone, which is known as a blunted luteal phase. When women enter menopause, their bodies cease to produce progesterone, since ovulation has stopped.

Because ovulation is essential for progesterone production, women who take birth control pills will have low progesterone. Women who use synthetic progesterone implants such as Depo Provera or progestin-impregnated IUDs such as Mirena are at risk for prolonged progesterone deficiency. Some women have difficulty conceiving for a long period of time after discontinuation of these progestin-based products. I have also seen many women on these forms of contraceptives develop hives or lip swelling/body swelling called angioedema. Allergists often call this type of hormone-induced disorder angioedema type III. Many of these women also gain weight.

Why do women develop these symptoms when their progesterone production is suppressed? The reason is that low progesterone frequently results in estrogen dominance. When progesterone is declining, many women continue to produce their own estrogen. Earlier in this book, we discussed at length the endocrine-disrupting effects of pesticides, and you are now aware that many household products, including cosmetics and cosmeceuticals, and many of the foods that we eat are estrogenic. Endogenous estrogens and xenoestrogens add up to cause symptoms, and progesterone deficiency complicates this situation of estrogen dominance.

THE RELATIONSHIP AMONG PROGESTERONE, OBESITY, AND DIABETES

Some women begin to have anovulatory menstrual cycles as early as their late twenties, and most women experience this phenomenon by their midthirties. Anovulation leads to progressive dwindling of the protective hormone progesterone, and the ensuing unopposed estrogen leads to a persistent—instead of transient, as occurs in the normal menstrual cycle—insulin increase. This persistent insulin increase, in turn, leads to a constant craving for sweets. Eating sweets or high-sugar-

content foods leads to an increase in blood glucose. Insulin has to quickly dispose of the glucose to prevent it from spilling into the blood. The fastest way of getting rid of the glucose in the blood is to put it into fat cells. Fat cells convert the glucose into fatty acids for storage, and the end result is weight gain and obesity; hence, it is not surprising to see women gain weight after age thirty-five or even sooner if their progesterone deficiency starts earlier.

Remember that the decrease in progesterone leads to estrogen dominance, which leads to an increase in insulin. Insulin causes craving for carbohydrates and is also responsible for putting glucose into adipocytes. Initially, when insulin increases, blood glucose decreases. This prompts cravings for carbohydrates (sugar-containing foods). In order to replenish the decreased glucose, adrenaline is also produced and most people become "hyper." When the fat cells are at capacity with glucose, they signal to the brain that they have enough glucose by producing hormones such as leptin. Leptin tells the hypothalamus to put the brakes on the appetite and sugar cravings. (Bios Life Slim—listed in the *Physicians' Desk Reference* [PDR]—boosts leptin to help adipocytes communicate better with the hypothalamus). After a while, the message is ignored by the hypothalamus and most people continue to gain weight. When the fat cells are full, the excess glucose spills into the blood, and high levels of glucose in the blood is what is known as diabetes (insulin resistance).

THE RELATIONSHIP BETWEEN PROGESTERONE AND THYROID FUNCTION

When progesterone decreases, thyroid function also decreases, which compounds the weight-gain problem. Progesterone has many effects on the thyroid. It aids in the retention of iodine, zinc, potassium, and selenium in our cells. Iodine is needed to create the thyroid prohormone T4, and zinc, potassium, and selenium allow T4 to enter cells, where it is converted to the active form, T3, which regulates metabolism. Low progesterone decreases the retention of these minerals in the cells. As a result, T4 does not convert to T3 readily. Instead, T4 converts to a storage form called reverse T3 (rT3), which tends to compete with T3 for its binding sites.

Patients with hypothyroidism are given a T4 equivalent, levothyroxine (Synthroid). If the patient has low progesterone, as occurs in menopausal women, this treatment will be ineffective. These patients do not readily lose weight and may feel tired all the time. If T3 is measured, it is found to be low. T3 may be within the standardized specified range that has been carved in stone by orthodox medical standards, but thyroid stimulating hormone (TSH) in this case is often greater than 2. If 0.3–3 (new range by the endocrinology society) is the normal range for TSH and 0.3 is considered perfectly optimal thyroid function, then anything greater than

0.3 is not perfect. The thyroid stimulating hormone (TSH) is inversely related to your thyroid function. The higher the TSH, the worse your thyroid function. If the TSH is greater than 2, then the thyroid function is not optimal. Furthermore, if the free T3 is below 3.5 (range: 2.3-4.2) and the reverse T3 is greater than 15 (range: 8-25), then thyroid supplementation may be considered. These are simple guidelines; but, the true guide for therapy is the patients' symptoms. We should treat the patient and not the lab values. Unfortunately, the rigid standard of care established by conventional medicine does not consider patients' low thyroid symptoms such as fatigue, rapid weight gain and difficulty losing weight, dry skin, anxiety, headaches, insomnia, and low body temperature. Practitioners around the world continue to treat patients with the same medications (Synthroid) even though improvement is not seen and patients continue to complain. This leads to patients becoming frustrated and seeking alternative therapies on their own.

Copper is required for the synthesis and release of estrogen and also forms enzymes in the liver that help to break down leftover estrogen into harmless substances. Estrogen can cause copper retention if zinc or progesterone levels are too low or calcium level is high. Since copper contributes to an increase in estrogen, an increase in the copper level in the body may decrease thyroid function. If calcium and copper increase together, they can have a detrimental effect on thyroid function.

Progesterone, by antagonizing estrogen and allowing potassium, selenium, and zinc to remain in cells, reverses all these negative mineral effects on thyroid function. Unfortunately, TSH levels, which are used by most practitioners to assess thyroid function, often do not capture this imbalance.

THE RELATIONSHIP BETWEEN PROGESTERONE DEFICIENCY (ESTROGEN DOMINANCE) AND ALLERGIES

Fat cells also produce adipokines such as TNF-α, interleukin-6, and many others that lead to inflammatory processes such as asthma, allergic rhinitis, and aches and pains. That is the reason most obese individuals experience these symptoms. One adipokine, adiponectin, is known to be inversely related to asthma symptoms and to obesity. It has been suggested that African mango extracts help boost adiponectin for weight loss. Fat cells also produce estrogens (estradiol and estrone) by converting testosterone to these estrogens through aromatase. The estrogens, in turn, increase the insulin level and decrease thyroid function (by increasing the thyroid-binding globulin) and, therefore, lead to more weight gain.

OTHER DISEASES AND DISORDERS CAUSED BY HORMONE IMBALANCE

In women, the decrease in progesterone and increase in estrogen can lead to several types of benign tumors such as fibroid tumors, fibrocystic breast disease, ovarian cysts, polycystic ovary syndrome (PCOS), Lymphangioleiomyomatosis (LAM, benign tumors in the lungs), Dercum's Disease (debilitating, multiple, painful fatty tumors—lipomas—also known as Adiposis Dolorosa), and malignant tumors, such as breast, vaginal, cervical, uterine, ovarian, and colon cancers, among others. Benign, but devastating tumors such as fibroid tumors are a source of heavy and often prolonged menstrual bleeding (dysfunctional uterine bleeding) and anemia, which can lead to hysterectomies (surgical removal of the uterus).

Both men and women lose their testosterone as they age, and this leads, in turn, to decreased libido, decreased muscle mass, increased insulin, and abdominal weight gain.

When men's testosterone levels begin to decline in their midfifties (some men experience a decline as early as their thirties), they experience an increased production of estrogens via the enzyme aromatase. Also, if they abuse alcohol or are obese, many tend to show evidence of what is colloquially known as "man boobs" or gynecomastia. More significantly, men in their fifties and older with high estrogen levels and without the protective effects of testosterone are at risk for prostate, colon, and other cancers.

The bottom line: Too much estrogen is not good for the body.

THE EFFECTS OF TESTOSTERONE DEFICIENCY

Xenoestrogens and phytoestrogens, coupled with endogenous estrogens, have similar effects in men and women. Younger men do not develop prostate cancer because of the protective effect of testosterone. Prostate enlargement, called benign prostatic hyperplasia (BPH), which develops in the majority of older men, may be due to too much estrogen. This condition is usually linked to dihydrotestosterone, a more potent form of testosterone that has been blamed for causing male pattern baldness and prostate enlargement. Low testosterone also leads to foggy thinking in men and women. Low testosterone in men causes the "angry man syndrome"

(grumpy old men). Bioidentical progesterone supplementation in men calms them down, and they concentrate better. Progesterone supplementation also decreases the conversion of testosterone to dihydrotestosterone and, hence, improves BPH symptoms.

HCUP data show higher numbers of patients discharged with a primary complaint of BPH in the South and Midwest, where use of estrogen-causing chemicals is greater than in the Northeast and West; hence, endogenous estrogens, xenoestrogens, and phytoestrogens have an impact on the prostate in terms of hypertrophy or cancer.

THE EFFECT OF DHEA DEFICIENCY ON INSULIN

Decreases in levels of dehydroepiandrosterone (DHEA) lead to increased insulin and, thus, potentiates lipogenesis (the formation of fat cells). Insulin increase not only leads to diabetes, but also to increased blood pressure secondary to salt retention by renal tubules. DHEA decreases with age and stress. DHEA and progesterone together are known to increase testosterone, estradiol, and estrone. Decreases in DHEA often lead to a decrease in testosterone, although estrogen levels may be normal because estrogens are produced by sources other than DHEA (adipocytes, skin cells, and adrenal glands).

DHEA is known as the mother of all hormones and affects all other hormones in the body. It is also known as the longevity hormone. Studies have shown that people who have adequate amounts of DHEA tend to live longer, on average, compared to people who have less. When DHEA goes down, the individual develops gray hair and ages faster. This explains why some young individuals who are under intense stress develop gray hair. A decrease in DHEA, which occurs with age and stress, has direct effects on overall well-being.

THE EFFECT OF ESTROGEN ON CANDIDA OVERGROWTH

Candida is a genus of yeasts that occur naturally in the human body. In healthy amounts, candida, which is primarily found in the intestines, aids in digestion and nutrient absorption; however, overgrowth of candida is the most common cause of fungal infections. Candida infections (candidiasis) typically affect the skin, genitals, throat, skin, and blood.

Estrogens feed candida growth, and this is the reason women have more candidiasis than men. Candida produces more than 79 major toxins that cause health problems, and candida overgrowth has become epidemic as the estrogen load in our environment has increased. Many now believe that candida participates in development of some cancers because it ferments glucose that cancer cells need to grow. That is the reason it is recommended to avoid sugars to prevent candida proliferation.

THE EFFECT OF STRESS ON THE DISTRIBUTION OF PREGNENOLONE

Stress causes pregnenolone (the precursor of progestogens, mineralocorticoids, glucocorticoids, androgens, and estrogens) to form cortisol in a process called pregnenolone steal. Initially, this process was intended to be a defense mechanism. When our ancestors faced bears, lions, tigers, and other dangers in their environment, cortisol shot up to increase blood glucose, which was converted into energy to flee or fight these dangers.

Nowadays, chronic stress makes it seem to our bodies that bears, lions, and tigers are lurking everywhere—at home, at work, at the mall, on the freeway—and, therefore, cortisol is constantly being generated. As a result, insulin and blood glucose also go up. Insulin may direct the excess glucose into the fat cells of the omentum, which causes the ventral type of obesity. These individuals are at risk for diabetes, hypertension, and heart disease.

When pregnenolone is used to make cortisol, DHEA production goes down. When insulin goes up, many individuals retain salt in the kidney tubules and high blood pressure follows. Insulin, therefore, may be one of the major contributing factors of high blood pressure. Individuals who have diabetes or high blood pressure do well and achieve better glycemic and blood pressure control when their DHEA is supplemented. Cortisol also reduces the thyroid function by preventing the conversion of T4 to T3.

Thyroid function, therefore, decreases five ways:

1. Too much estrogens cause an increase in TBG, which reduces thyroid function.
2. Too much estrogens cause production of thyroid antibodies (TG antibodies and TPO) that lead to inflammation of the thyroid gland and cause suboptimal thyroid function. If you have thyroid antibodies or a diagnosis of Hashimoto's thyroiditis, you should strictly follow the Estrogen-Free Lifestyle. Do not wait until your thyroid dies before taking action. Unfortunately, "wait and see" is the recommendation of many practitioners.
3. Too much estrogens (extrinsic and intrinsic) and other environmental toxins cause T4 to convert into reverse T3, a storage form of thyroid hormone that competes with the T3 receptors and renders T3 ineffective.
4. Low progesterone prevents useful minerals from entering our cells and, therefore, reduces the conversion of T4 to T3 and leads to suboptimal thyroid function.
5. Too much cortisol also prevents the conversion of T4 to T3 and leads to suboptimal thyroid function.

WHY LDL ("BAD") CHOLESTEROL INCREASES WITH AGE AND OBESITY

When hormones are out of balance, the body is triggered to make more to try to maintain the health of the individual. Low-density lipoprotein (LDL) cholesterol is used for this process. When LDL cholesterol increases, many practitioners' reaction is to assume the patient is at risk of myocardial infarction (heart attack). The patient is prescribed a statin drug to lower the "bad" LDL cholesterol; however, if the LDL goes down too much, what happens to hormone production? Many healthcare providers do not ask this question.

In fact, when LDL decreases, the patient may be at even higher risk of death. It does not really matter whether your cholesterol is high or low. You can die of a heart attack if your cholesterol comprises primarily small, dense particles. The lipoprotein particle (LPP) test, offered by SpectraCell and other laboratories, fractionates LDL cholesterol into small, dense particles and large particles. It is thought that the small, dense particles are the ones involved in heart disease.

In order for the small dense LDL particles to cause damage, however, another element called homocysteine must be present. Homocysteine is an amino acid that circulates in the blood and pokes holes into the arteries. Small, dense LDL particles lodge in these holes and cause inflammation. Antigen-presenting cells (APC) (macrophages, monocytes, and dendritic cells) respond to the area of inflammation and eat so much of the cholesterol remnants that they become obese. These obese APCs are known as foam cells. These foam cells die in the inflamed area and more APCs are sent to clean up these dead foam cells, and they, too, die. As time goes by, these dead cells form a cemetery of white blood cells known as plaque.

If the plaque is extensive enough, it obstructs the interior of the arterial vessels that provide blood to the heart. If the heart muscles do not receive blood, that part of the heart dies. That is called a heart attack. If it is a part of the brain that does not receive blood, that part also dies, and it is called a stroke.

We can prevent this process—known as oxidation of LDL cholesterol—by eating foods rich in antioxidants or by taking good antioxidant supplements including B-vitamins and folate. Deficiency of B-vitamins and folate lead to an increase in homocysteine. I, therefore, recommend antioxidant supplements to many of my patients. We know now that methylated formulations of B12 and folate may be better absorbed by individuals with MTHFR (methylenetetrahydrofolate reductase) gene mutation. Your practitioner may order an MTHFR mutation test for you if your practitioner suspects such a problem and makes the appropriate recommendations for you.

Cholesterol is made in the liver from fat and glucose. Insulin serves as the catalyst for the rate-limiting enzyme HMG-CoA reductase, which assists in regulating cholesterol production. This is the reason many diabetics have cholesterol problems and why obese men and women (who invariably have hormone imbalances) also have fatty liver. When cholesterol is produced, it goes to the gallbladder to mix with bile to aid in the digestion of fat; however, sometimes when the cholesterol reaches

the gallbladder, there are no bile salts available for the mixing to take place. This leads to cholecystitis (inflammation of the gallbladder), which is one of the main reasons for a cholecystectomy (surgical removal of the gallbladder).

In medical school, we learned that cholecystitis is often found in individuals described by the following adjectives: female, fat, fertile, forty—the four Fs. The only thing peculiar about women around the age of forty is anovulatory menstrual cycles that lead to progesterone deficiency. Oral contraceptives are also a risk factor, since they tend to prevent progesterone production. As we have learned, progesterone deficiency leads to estrogen dominance, and this eventually leads to obesity; hence, the four Fs perfectly fit the hormone imbalance state in women in their forties. Many women with hormone imbalance have already had a cholecystectomy when they come to my clinic. Looking at the HCUP database, it appears that women, on average, have slightly higher cholesterol than men; however, coronary artery disease and myocardial infarction are much higher in men than women.

Since cholesterol is made from glucose and from fatty acids that convert to acetyl-CoA, good glucose metabolism may also help with blood glucose and fatty acid control and, therefore, cholesterol control. I often prescribe alpha-lipoic acid to my patients for better glucose metabolism. Pure Encapsulations carries two combination vitamins—RevitalAge Ultra or RevitalAge Nerve—that combine alpha-lipoic acid and acetyl-l-carnitine to improve both glucose and cholesterol metabolism.

THE MOST PREVALENT DISEASES IN THE U.S. ARE DUE TO HORMONE IMBALANCE

Coronary artery disease and myocardial infarction (MI) are more prevalent in the South and Midwest than in the Northeast and West. This trend is the same for all types of MI. MI is more likely to occur as men get older. Men who convert their testosterone to high estrogens are at increased risk of developing MI. This group tends to have low testosterone, which makes sense because low testosterone leads to high insulin, which, in turn, leads to ventral obesity in men and, hence, higher risk of MI. Testosterone is often lost to estrogens in older men and, when estrogen increases, insulin increases. As they age, men begin to crave sweets, just as women do in their late luteal phase, and, therefore, become obese. The high endogenous estrogens in aging men coupled with environmental estrogens lead to obesity, benign prostatic hyperplasia, prostate cancer, and colon cancer. The low testosterone leads some of these men to use Viagra or Cialis to fix their erectile

dysfunction. These medications tend to make the heart disease worse and may even cause death if they are taken in conjunction with nitrates. Some men with erectile dysfunction also seek help from their healthcare practitioners. Since many practitioners are not well-verse in hormone evaluation and treatment, they tend to just measure testosterone level. If testosterone is low, then they prescribe testosterone gels. There is a danger in this practice because the exogenous testosterone administration may convert to estrogens and you can see how this may lead to MI. Men should, therefore, exercise caution if they have erectile dysfunction by finding an Integrative Immunity practitioner or Functional Medicine practitioner to test their hormones thoroughly and provide BHRT suitable for men.

Men with low testosterone and high estrogen have many of the same symptoms as women with estrogen dominance. The most common of these symptoms are fatigue, irritability, apathy, lack of ambition, decreased libido (no testosterone means diminished libido for both men and women), anxiety, nervousness, depression, weight gain (pot bellies, which can be made worse by beer because the hops in beer is a phytoestrogen), high blood pressure, diabetes, and hypercholesterolemia.

These conditions represent the bulk of what primary care physicians treat. These disorders are mostly hormone related, and hormones are not checked by most physicians. Some physicians begin patients on testosterone gels and injections when patients complain of decreased libido without even measuring the testosterone level. Some healthcare providers measure the testosterone level and then start their patients on testosterone replacement therapy, if necessary, but they never check estradiol, estrone, or dihydrotestosterone (DHT, a potent androgen), or DHEA levels. In many patients, the testosterone in the gel or injection is converted by aromatase into estrone and, to a lesser extent, estradiol; in others, the testosterone is converted to DHT. When testosterone converts to estrogens via aromatase, insulin increases, and that can lead to the hormonal cascade, which causes heart disease; hence, giving testosterone to men and not checking what happens to it can be disastrous.

These estrogenic by-products of testosterone can cause harm by contributing to prostate enlargement or even prostate cancer if these estrogens become too high and remain imbalanced over a long period of time. It is important to measure hormone levels prior to starting anyone on hormone supplements. When a patient is on bioidentical hormones, the follow-up should be every three to six months, and the patient should have full access to the physician for adjustment of these hormones. Reassessment of hormones at six months or sooner should be conducted if the patient still complains of symptoms. The major reason for not doing well on

bioidentical hormone therapy is nonadherence to the Estrogen-Free Lifestyle, which is discussed later in this book.

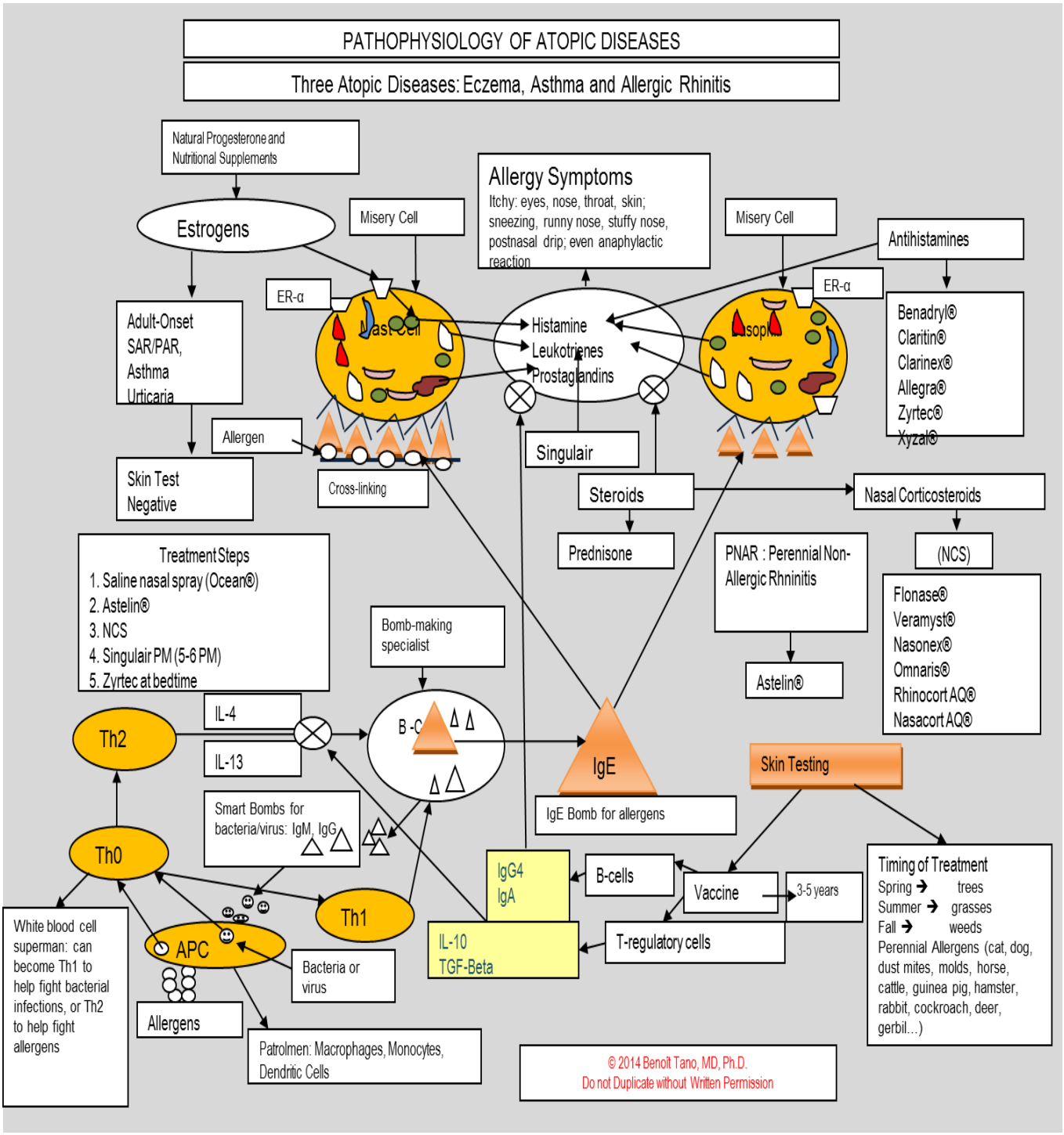
THE END RESULT OF ENDOGENOUS HORMONE PRODUCTION IMBALANCE

To summarize, the end result of endogenous hormone production imbalance includes:

- Progesterone deficiency
- Estrogen dominance
- Thyroid deficiency
- Leptin resistance
- Insulin resistance
- Hypertension
- Hyperlipidemia
- DHEA deficiency
- Testosterone deficiency

While obesity and its comorbidities are increasing rapidly worldwide, allergic diseases seem to follow the same pattern. In the case of growing atopic diseases, environmental pollution and the estrogen epidemic are critical contributing factors.

The two following diagrams, which show the pathophysiology of atopic diseases and the pathophysiology of food allergies, shed some light on this growing problem.


PATHOPHYSIOLOGY OF ATOPIC DISEASES
Three Atopic Diseases: Eczema, Asthma and Allergic Rhinitis
Natural Progesterone and Nutritional Supplements
Estrogens
Misery Cell
Allergy Symptoms
Itchy: eyes, nose, throat, skin; sneezing, runny nose, stuffy nose, postnasal drip; even anaphylactic reaction
Misery Cell
Antihistamines
ER-α
ER-α
Adult-Onset SAR/PAR, Asthma Urticaria
Mast Cell
Histamine
Leukotrienes
Prostaglandins
Basophil
Benadryl®
Claritin®
Clarinex®
Allegra®
Zyrtec®
Xyzal®
Allergen
Singulair
Skin Test Negative
Cross-linking
Steroids
Nasal Corticosteroids
Prednisone
(NCS)
Treatment Steps
1. Saline nasal spray (Ocean®)
2. Astelin®
3. NCS
4. Singulair PM (5-6 PM)
5. Zyrtec at bedtime
PNAR : Perennial Non-Allergic Rhninitis
Flonase®
Veramyst®
Nasonex®
Omnaris®
Rhinocort AQ®
Nasacort AQ®
Bomb-making specialist
Astelin®
IL-4
IL-13
Th2
B-C
IgE
Skin Testing
Smart Bombs for bacteria/virus: IgM, IgG
IgE Bomb for allergens
Th0
IgG4
IgA
B-cells
Vaccine
3-5 years
Timing of Treatment
Spring → trees
Summer → grasses
Fall → weeds
Perennial Allergens (cat, dog, dust mites, molds, horse, cattle, guinea pig, hamster, rabbit, cockroach, deer, gerbil…)
Th1
IL-10
TGF-Beta
T-regulatory cells
White blood cell superman: can become Th1 to help fight bacterial infections, or Th2 to help fight allergens
APC
Bacteria or virus
Allergens
Patrolmen: Macrophages, Monocytes, Dendritic Cells
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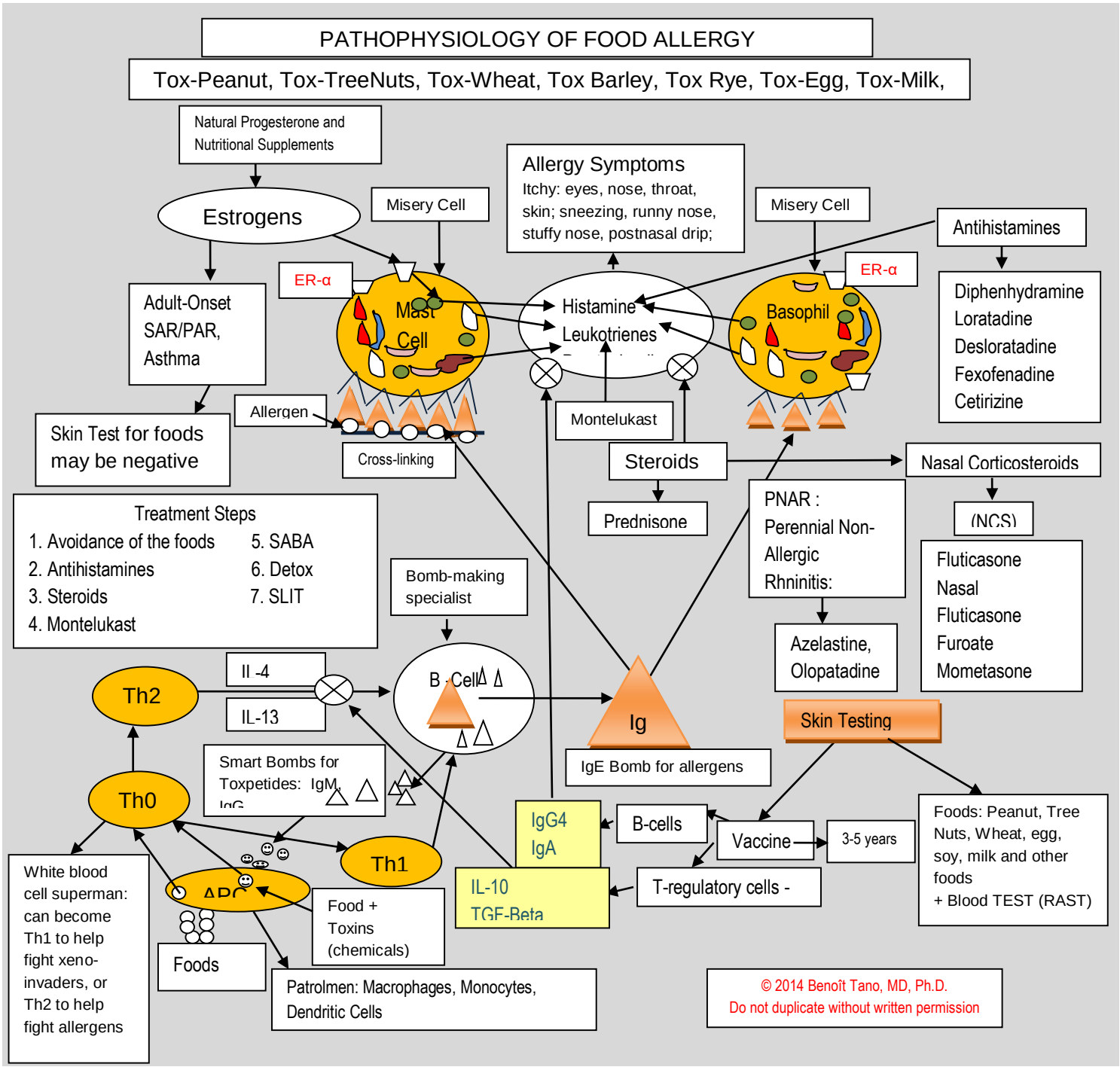
PATHOPHYSIOLOGY OF FOOD ALLERGY
Tox-Peanut, Tox-TreeNuts, Tox-Wheat, Tox Barley, Tox Rye, Tox-Egg, Tox-Milk,
Natural Progesterone and Nutritional Supplements
Estrogens
Misery Cell
Allergy Symptoms
Itchy: eyes, nose, throat, skin; sneezing, runny nose, stuffy nose, postnasal drip;
Misery Cell
Antihistamines
ER-α
Mast Cell
Histamine
Leukotrienes
Basophil
ER-α
Diphenhydramine
Loratadine
Desloratadine
Fexofenadine
Cetirizine
Adult-Onset SAR/PAR, Asthma
Allergen
Montelukast
Skin Test for foods may be negative
Cross-linking
Steroids
Nasal Corticosteroids
Prednisone
(NCS)
Treatment Steps
1. Avoidance of the foods
2. Antihistamines
3. Steroids
4. Montelukast
5. SABA
6. Detox
7. SLIT
PNAR : Perennial Non-Allergic Rhninitis:
Fluticasone Nasal
Fluticasone Furoate
Mometasone
Bomb-making specialist
Azelastine, Olopatadine
Th2
Il -4
IL-13
B -Cell
Ig
Skin Testing
Smart Bombs for Toxpetides: IgM,
IgE Bomb for allergens
Th0
IgG4
IgA
B-cells
Vaccine
3-5 years
Foods: Peanut, Tree Nuts, Wheat, egg, soy, milk and other foods
+ Blood TEST (RAST)
Th1
IL-10
TGF-Beta
T-regulatory cells -
White blood cell superman: can become Th1 to help fight xeno-invaders, or Th2 to help fight allergens
Food + Toxins (chemicals)
Foods
Patrolmen: Macrophages, Monocytes, Dendritic Cells
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MY ROAD MAP FOR TREATING ALLERGIC DISEASES

Successful treatment of environmental and food allergies is based on the pathophysiology of atopic diseases as depicted in the first diagram above. The pathophysiology of food allergies is depicted in the second diagram. These diagrams show the interactions among the white blood cells that lead to the release of toxic mediators from the Misery Cells (mast cells and basophils).

When patients come to my clinic complaining of allergic rhinitis, or other atopic symptoms, I take a thorough history and perform a physical examination. After the history and physical examination, I discuss the pathophysiology (the allergy treatment "road map" for atopic diseases and food allergies). I then explain how to block each of these toxic mediators: antihistamines to block histamine, leukotriene receptor antagonists to block leukotrienes, and corticosteroids to block all of these mediators. Antihistamine nasal sprays and combination nasal corticosteroid/antihistamine nasal sprays are used to treat multiple chemical sensitivities.

Adjuvant therapy with nutritional supplements, vitamins, and minerals is also covered, as well as the Estrogen-Free Lifestyle (EFL), discussed below.

For long-term relief of atopic diseases, skin testing is performed to identify the environmental allergens involved. The allergens found determine the seasonality of the allergies and, hence, clarify the treatment approach.

The long-term relief and most cost-effective treatment of atopic diseases involves using allergen immunotherapy. In the U.S., doctors use two kinds: subcutaneous immunotherapy (SCIT), also known as allergy shots and sublingual immunotherapy (SLIT), known as allergy drops. Both are equally effective. Either form of this allergy vaccine stimulates T-regulatory cells and B-cells to produce four major chemicals (IL-10, TGF-Beta, IgA, and IgG4), which induce tolerance to allergens.

QUESTIONS ANSWERED IN THIS BOOK

WHY AM I HAVING ALLERGY SYMPTOMS?

This question has been addressed by offering the genetic and epigenetic underpinnings of allergic rhinitis, sinusitis, conjunctivitis, atopic dermatitis, asthma, and food allergies.

WHAT IMMUNE CELLS ARE INVOLVED?

This question has been addressed by explaining the workings of the immune defense system:

- Action of the antigen-presenting cells (patrolmen)
- Action of the Th0-cell (superhero or superman)
- Action of the B-cells (bomb-making cells)
- Role of the IgE bomb
- Role of the IgM and IgG bombs
- Role of mast cells (Misery Cell no. 1)
- Role of basophils (Misery Cell no. 2)

HOW DO I GET RID OF MY ENVIRONMENTAL AND FOOD ALLERGY SYMPTOMS?

This question has been addressed by showing you how you can block the actual chemicals involved (medication therapy):

- Block the histamine
- Block the leukotrienes
- Block all chemicals involved
- Block multiple chemical sensitivities

HOW DO I ACHIEVE LONG-TERM ALLERGY RELIEF

Perform skin testing and use allergy vaccine to:

- Block the actions of the Th0-cell (by SCIT or SLIT)
- Block the actions of the bomb-making cells (by SCIT or SLIT)
- Block the actions of mast cells and basophils (Misery Cells) by SCIT or SLIT

Determine the effect of allergen immunotherapy (AIT):

- Action of the T-regulatory cells
- Action of the bomb-making cells

Adjuvant therapy with nutritional supplements, vitamins, and minerals is also suggested (see Appendix B). For long-term relief, skin testing and a radioallergosorbent test (RAST) for suspected allergenic foods are performed to identify immediate hypersensitivity food reactions that are IgE-mediated and delayed food sensitivity reactions that are IgG-mediated. The long-term relief and

most cost-effective treatment of food allergies involve avoidance of certain foods, adopting the Estrogen-Free Lifestyle (discussed later), and using food allergen immunotherapy. In the U.S., a few doctors use sublingual immunotherapy (SLIT) in food allergy immunotherapy and others use oral immunotherapy (OIT). Studies are underway to determine the efficacy of these long-term treatment modalities.

These oral vaccines stimulate T-regulatory cells and B-cells to produce the four major chemicals (IL-10, TGF-Beta, IgA, and IgG4) that induce tolerance to foods that cause allergic reactions.

REFERENCES AND NOTES

For complete references, please visit the Integrative Immunity website: www.integrativeimmunity.com, which also has information about my other books, *The Allergy Detective: Allergic Rhinitis Treatment Secrets Your Doctor May Not Tell You* and *Hormone Imbalance Syndrome: America's Silent Plague.* The latter book uncovers the roots of the obesity epidemic and most common diseases. These books are also available at Amazon.

www.amazon.com/Hormone-Imbalance-Syndrome-Americas-Silent/dp/0983419205/

www.amazon.com/Hormone-Imbalance-Syndrome-Americas-ebook/dp/B00CLHIEY4/

www.amazon.com/Allergy-Detective-Allergic-Rhinitis-Treatment/dp/0983419221/ and www.amazon.com/Allergy-Detective-Allergic-Treatments-ebook/dp/B006C2C7CO/

Not all vitamins are created equal and, to get your money's worth, you may want to purchase professional vitamins on the Integrative Immunity website: www.integrativeimmunity.com, or from the Pure Encapsulations website: www.purecapspro.com/toai.

Ortho Molecular, Metagenics, and other professional nutritional supplement companies also offer excellent products.

PART III: KNOW YOUR FOODS AND BEVERAGES: THE ESTROGEN-FREE LIFESTYLE

This book has demonstrated the adverse effects of endogenous estrogens and exogenous estrogens in the pathophysiology of common diseases. Based on this knowledge, I have created the Estrogen-Free Lifestyle (EFL). This lifestyle change consists of simply avoiding xenoestrogens and phytoestrogens and their associates. Nutritional supplements are also added for a complete, life-changing experience.

It must be assumed that if you are obese, estrogen dominance exists; therefore, anyone who wishes to lose weight is well advised to follow the EFL. This means avoiding estrogenic carbohydrates, estrogenic proteins, and estrogenic fats—i.e., avoiding xenoestrogens, phytoestrogens, and their associates. Certain dietary supplements are also recommended, and, in some cases, hormones need to be brought into balance.

AVOID XENOESTROGENS AND PHYTOESTROGENS

Plant-based estrogens (phytoestrogens) are found in barley, rye, wheat, spelt, kamut, millet, and GMO corn. These grains are plant-based estrogens. Avoid these grains. Eat oats (gluten-free), rice (wild or brown, preferably organic), amaranth (has high glycemic index), and quinoa. Brown rice pasta and rice-based bread is readily available in most natural grocery stores and in many chain supermarkets.

Corn is estrogenic by association: it is a GMO that has been developed to tolerate Roundup. The main ingredient in Roundup is glyphosate, which is known to be estrogenic. Several other chemicals are also used in the production of corn, as shown in the corn pesticides table in the previous section. Atrazine is an aromatase booster and converts testosterone to estrogen. Alachlor, acetochlor, metolachlor, and metolachlor-S are toxic to the thyroid and cause nasal turbinate tumors in animals and, perhaps, nasal turbinate enlargement; hence, allergies in humans. High fructose GMO corn syrup is ubiquitous and found in many processed foods, sodas, and juices.

As much as possible, avoid the foods and chemicals listed in Table 23 below.

Table 23. Common Xenoestrogens and Phytoestrogens

Alfalfa Sprouts	Garlic	Sodas high in caffeine
Algin	Goldenseal	Soy
Aconite	Hemp Oil	Spelt
American Pennyroyal	Hops (in beer)	Spermicide (Nonoxynol-9 on condoms)
Anise	Horseradish	Squill
Anti-Ulcer Drugs (Tagamet)	Hyssop	Squirting Cucumber
Arnica	Jequirity	Stinging Nettle
Artemisia	Juniper	Sunflower Oil
Asafetida	Kamut	Sunflower Seeds
Asarum	Laundry Detergent	Sunscreens
Bael Fruit	Lavender Oil	Tansy
Barley	Licorice	Tea
Bay Leaf	Lovage	Tea Tree Oil
Birthwort	Marjoram	Thyme
Bisphenol A	Mandrake	Tribulus
Bitter Melon	Marijuana	Turmeric
Blessed Melon	Millet	Verbena
Blessed Thistle	Mint	Watercress
Bloodroot	Mistletoe	Wheat
Buchu	Motherwort	Willow
Caffeine	Mugwort	Yarrow
Calamus Root	Neem	Yew
Canola Oil	Nutmeg	
Celery	Ocotillo	
Chamomile	Oleander	
Chamomile Tea	Oregano	
Chaparral	Parabens (methyl, Propyl, butyl)	
Chicory	Peppermint	
Chocolate	Phenoxyethanol	
Cinnamon	Plastic Heated	
Clover	Podophyllum	
Cloves	Pokeweed	
Coffee Decaffeinated	Pomegranate	
Corn (GMO)	Poplar	
Cottonseed Oil	Queen Anne's Lace	
Cumin	Red Clover	
Cypress	Red Clover Tea	
Damiana	Rhubarb	
Dates	Rosemary	
Essential Oils	Rue	
European Pennyroyal	Rye	
Fenugreek	Safflower	
Ferns	Safflower Oil	
Feverfew	Sage	
Flax Oil	Slippery Elm	
Fragrance		

Phytoestrogen Content of Foods Consumed in Canada, Including Isoflavones, Lignans, and Coumestan: Lilian U. Thompson , Beatrice A. Boucher , Zhen Liu , Michelle Cotterchio & Nancy Kreiger Published online: 18 Nov 2009. NUTRITION AND CANCER, 54(2), 184–201

Phytoestrogens and Endocrine Disruptors in Breast Cancer: Albrini et al. Current Medicinal Chemistry, 2014, Vol. 21, No. 9

AVOID DAIRY

Cows are fed GMO corn and soy that contain endocrine-disrupting chemicals that are transferred into the milk of these animals. Cows may also be injected with growth hormones that are estrogenic and cause inflamed mammary glands. These cows are then treated with antibiotics that also find their way into the milk. The estrogenic milk causes mast cell and basophil degranulation, leading to hypersensitivity reactions (profuse mucus production and even anaphylactic reactions—remember that milk is one of the major foods that cause allergic reactions in children). Since the estrogen load has increased in the environment, so has dairy sensitivity in children and adults.

There are many delicious milk substitutes available today—coconut milk, rice milk, almond milk, cashew milk, hemp milk, and, of course, soy milk. It is wise to avoid soy milk, however, as soy is estrogenic, often GMO, and treated with glyphosate—making it double or triple estrogenic. Hemp is also listed in the table above and is to be avoided.

AVOID "DIRTY" FOODS

In my opinion, there are far more than a dozen foods that are dirty. I would make a list called "The Dirty Thousand." All foods produced with nonorganic chemicals are dirty until proven otherwise. Eat organic and whole foods as much as possible, and avoid all processed foods.

We all know that it is good to eat organic foods. Even though the U.S. Government passed the Organic Food Production Act in 1990, farmers have been reluctant to switch to organic production because of the work involved, and many also cite low yield. Demand for organic food is, therefore, higher than supply. As a result, organic food prices are higher than those for pesticide-treated foods. These higher prices dissuade many consumers from buying organic foods.

There is also the phenomenon of so-called food islands. In inner-city America, there is often a lack of grocery stores with fresh produce, and many inner-city dwellers rely primarily on convenience stores for their food purchases. Healthy and organic foods are rarely available in these stores.

AVOID REFINED SUGARS AND ARTIFICIAL SWEETENERS

Half of all refined sugars in America are produced from sugar beets. Sugar beet is the number one GMO food in this country (see GMO crops table below) and, therefore, estrogenic (remember: GMO=Roundup=glyphosate=estrogen).

What Kind of Foods are Genetically Engineered?

There are currently six major foods sold in the US that are typically genetically engineered. These are listed below with the percent that are GE:

Food	% GE
Sugar beets	95%
Soybeans	93%
Cotton (Cottonseed oil)	78%
Canola	75%
Corn	70%
Papaya	50%

Weed killers (herbicides-especially Glyphosate) account for about one-third of the global pesticide market, and around 80% of GMO seeds involve herbicide-resistance.

Avoid sodas of all types. Regular sodas contain High-fructose-GMO corn-syrup (HF-GMO-CS) and GMO sugar, and diet sodas contain artificial sweeteners such as aspartame, which may convert into formaldehyde, a toxic chemical that is highly concentrated in corn. Avoid all candy unless it is sweetened with xylitol, a natural sugar from the birch tree that is known to help get rid of bacteria. [Do not over-ingest xylitol because it carries a warning that it is toxic to animals; since we are in the mammalian family, the verdict regarding its safety may change.] Avoid juices and dried fruits. For additional reading, see list of hidden sugars in "JJ Virgin's Sugar Impact Diet".

OTHER SUGGESTIONS IN ADDITION TO LIVING AN ESTROGEN-FREE LIFESTYLE

In addition to avoiding the food products mentioned above, here are a few other recommendations:

Limit acidic foods
Eat more alkaline foods (start with the cruciferous vegetables that are mostly alkaline—see the table below. Remember, cruciferous vegetables can increase your protective estrogen, 2-hydroxyestrone).

Table 24. Cruciferous Vegetables

Arugula (wild and rocket)	Komatsuna
Bok Choy	Land Cress
Broccoli (includes rabe, wild, Romanesco, Broccoflower, Chinese)	Maca
Brussels Sprouts	Mizuna
Cabbage (Napa, Chinese, Savoy, wrapped heart - mustard)	Mustard (Ethiopian)
Cauliflower	Mustard Seeds (brown, white, greens)
Collard Greens	Radish
Daikon	Rapeseed
Field Pepper weed	Rutabaga (has high glycemic index)
Garden Cress	Tatsoi
Kale (include Siberian)	Turnip
Kohlrabi	Wasabi

Drink plenty of filtered water (consider drinking alkaline water occasionally)

Find an Integrative Immunity or Functional Medicine practitioner to measure steroid hormones and do a thorough thyroid function test (test TSH, free T4, free T3, reverse T3 and thyroid antibodies, thyroglobulin antibodies, and thyroid peroxidase antibodies). If progesterone is low, your

practitioner may start you on bioidentical progesterone. If DHEA, progesterone, or testosterone are low, your practitioner may prescribe appropriate bioidentical hormone replacement (BHRT) for you. Your practitioner should know that progesterone, DHEA, and testosterone can convert to estrogens, and, hence, should consider that in your BHRT formulation. If all three hormones (progesterone, DHEA, and testosterone) are low, the precursor of these hormones, your pregnenolone may also be low and your practitioner may test and add pregnenolone supplementation to your BHRT regimen.

If free T4 and free T3 are low, reverse T3 is high, and TSH is greater than 2, then your practitioner may consider thyroid supplementation with Armour thyroid, Nature-Throid, or any of the natural, desiccated thyroid supplementation products, or with compounded T4/T3. If T4 is really low, Synthroid or levothyroxine may be added to your natural thyroid replacement. You may add a thyroid-support supplement, or iodine and tyrosine supplement, or Mineral 650 with copper and iron if you have low free T3, high reverse T3, and low ferritin levels.

If your thyroid antibodies are elevated, you have what is known as Hashimoto's thyroiditis. Hashimoto's, in my opinion, goes hand-in-hand with estrogen dominance and estrogen sensitivity. I, therefore, urge my patients with Hashimoto's thyroiditis to strictly follow the Estrogen-Free Lifestyle.

If your hormone tests show high estradiol and estrone, then avoid phytoestrogens and xenoestrogens. If your hormone tests show that your estrogen metabolites are off (low 2-hydroxyestrone-protective estrogen metabolite and high to very high 16-alpha-hydroxyestrone—dangerous—estrogen metabolite, which is involved in estrogen-induced cancers), you should, in addition to your BHRT, consume plenty of cruciferous vegetables (see list above) and adhere to the EFL strictly.

If you have challenges with your weight in the form of increasing weight gain, you have estrogen dominance and you may experience symptoms of low thyroid. You should ask your Integrative Immunity or Functional Medicine practitioner to start you on adequate thyroid supplementation. Remember that fat cells convert your testosterone to estrogens and the estrogens make your TBG (thyroid-binding globulin) levels higher and prevent your thyroid from working properly; hence, the EFL plus thyroid supplementation, and BHRT may help you lose unwanted weight, and decrease your estrogen load. You should eat low-glycemic-index carbohydrates and proteins low in glucogenic amino acids such as fish, plant proteins (except

phytoestrogens such as soy proteins), hormone-free organic poultry, and grass-fed, organic beef. A diet rich in ketogenic proteins may also offer benefit.

In addition, consider high quality nutritional supplements (discussed later in this section), including indole-3-carbinol (I3C) + DIM (dindolylmethane) and bioflavonoids. Indole-3-carbinol and DIM are cruciferous-vegetable extracts. These two supplements may or may not increase your 2-hydroxyestrone level. Over the years of giving these supplements to women who have low 2-hydroxyestrone, I have found that these good estrogen boosters may have the unintended effect of reducing the protective 2-hydroxyestrone in some patients. I, therefore, recommend eating plenty of cruciferous vegetables in addition to these extracts.

Ask your practitioner to measure your fasting insulin and leptin levels. If fasting insulin and leptin are high, insulin and leptin resistance is a problem. You may start on leptin boosters such as Bios Life Slim (which helps with blood glucose metabolism), and can be found in the *Physician Desk Reference* (PDR).

Take adiponectin (which helps reduce fat) boosters such as *Irvingia Gabonensis* (African mango) and raspberry ketone.

Take broad-spectrum antioxidants and minerals such as Nutrient 950 with NAC (a multivitamin that includes a good dose of B-vitamins, NAC—liver detox—the precursor for glutathione, and several important minerals).

Take vitamin D3, 5,000-10,000 IU daily (for adults) if your measured vitamin D level is low. Studies have shown that an average of 8,000 IU is optimal for most adults.

Take Digestive Enzymes Ultra with Betaine HCL if you have GERD (acid reflux).

Take Probiotics-50 B and augment this with the prebiotic fructooligosaccharide (FOS) supplement.

ADDITIONAL STEPS TO EFFECTIVELY TREAT OBESITY

Since obesity has such dire consequences, most individuals who notice weight gain decide to lose the weight. The first thing often recommended by physicians and attempted by patients is the unnatural and difficult route of dieting, which means restricting food intake.

When the body senses a sustained lack of food, it goes into an energy-conserving mode. This means that the body, a very smart machine, tries to store as much energy in the form of fat as possible. The glucose-alanine cycle shifts into gear to continue the supply of glucose to provide energy for body tissues. The glucose-alanine cycle converts muscle proteins to glucose and, therefore, muscle mass is lost. This energy-saving mode causes loss of water and muscle mass, but not fat. At a certain point, the individual realizes that he or she is not losing any more weight. Out of desperation and discouragement, the individual resumes his or her normal diet. The individual, therefore, gains even more weight, and the individual finds another diet program again. This is the origin of what is known as the yo-yo diet. There are many diet programs out there and as you notice, many individuals continue to fail these protocols because diets are cumbersome and do not get to the root causes of the obesity epidemic.

In the quest to lose weight, there is often a total failure to consider the hormones that initiate the weight-gain cascade. Losing weight and keeping it off entails correcting the underlying hormone imbalance, eating from healthy food categories in the right proportions, and getting exercise.

Here are the basic rules for eating to lose weight:

Eat for your hormone type
Choose foods that are optimal for weight loss
Use nutritional supplements to assist in weight control
Exercise

Many weight-loss programs recommend low-carbohydrate diets, some focus on high-protein diets, and most recommend low-fat diets, but few experts incorporate balancing the hormones as part of a weight-loss strategy. The failure of most weight-loss programs stems from not looking at all the determinants in the obesity equation. Based on my knowledge of the body, our environment, and the foods and beverages that people consume, I recommend the inclusion of all the variables identified in the obesity model to formulate a weight-loss plan that works.

Eating to Lose Weight

Many books on diet and hormones contain recipes. Considering that all patients do not like the same foods and food preferences are as varied as those for clothing and may depend on the culture and region of patients' origin, you will not find any recipes in this book. Instead, I recommend, in addition to the EFL, low-glycemic-index food choices to minimize the surge in glucose that tends to fuel the obesity problem and create leptin and insulin resistance, which are culprits in the bulk of the diseases treated by all physicians in the industrialized world.

The Importance of the Glycemic Index in Losing Weight

I frequently recommend Jennie Brand-Miller's *New Glucose Revolution Shopper's Guide* to my patients. The glycemic index (GI) is a measure of the power of foods (or, specifically, the carbohydrate in a food) to raise blood sugar (glucose) levels. Stated simply, foods with a high GI score contain rapidly digested carbohydrates, which produce a large and rapid rise and fall in the level of blood glucose. In contrast, foods with a low GI score contain slowly digested carbohydrates, which produce a gradual, relatively low rise in the level of blood glucose.

The GI values of foods must be measured using valid scientific methods; the GI cannot be guessed by looking at the composition of the food. Currently, only a few nutrition research groups around the world provide a legitimate testing service. Professor Brand-Miller of the Human Nutrition Unit at Sydney University in Australia has been at the forefront of glycemic index research for over a decade, and her research group has determined the GI values of more than 400 foods.

The GI value of a food is determined by feeding ten or more healthy people a portion of the food containing 50 grams of digestible (available) carbohydrate and then measuring the effect of the food on their blood glucose levels (glucose AUC – Area Under the Curve) over the next two hours. On another occasion, the same ten people consume an equal-carbohydrate portion of glucose sugar (the reference food) and their two-hour blood glucose response is measured. A GI value for the test food is then calculated for each person by dividing their glucose AUC for the test food by their glucose AUC for the reference food. The final GI value for the test food is the average GI value for the ten people.

The Role of Carbohydrates in Achieving and Maintaining a Healthy Weight

A patient with leptin resistance and/or insulin resistance should choose carbohydrates judiciously. All carbohydrates are not created equal. The carbohydrates high on the glycemic index and with a high-glycemic load should be avoided.

Although many diets focus on low-carbohydrate intake, a low-carbohydrate diet is useless if you are eating a small amount of carbohydrates with a high-glycemic index. For example, dry dates have a glycemic index of 103; a few dates will transform more quickly into glucose than a bowl of Uncle Ben's converted rice, which has a glycemic index of 44. Fruits and vegetables are highly recommended by nutritionists and doctors all over the world; however, you cannot ignore the glycemic index of fruits and vegetables if you are trying to lose weight or fight candida. For example, if you eat grapes, ripe bananas, watermelon, dates, potatoes, corn or other grains, such as barley, rye, wheat, very often, you are likely to gain weight due to their high-glycemic index and glycemic load.

The Role of Proteins in a Healthy Diet

Avoid eating proteins with a high-glucogenic amino acids content such as GMO grains-fed beef, GMO grain-fed poultry, farm-raised fish, and shellfish (salmon, tilapia, shrimp, etc.). A glucogenic amino acid is an amino acid that can be converted into glucose via gluconeogenesis. This is in contrast to ketogenic amino acids, which are converted into ketone bodies. Eat more ketogenic proteins.

The production of glucose from glucogenic amino acids involves the conversion of these amino acids into alpha-keto acids and then to glucose, with both processes occurring in the liver. This mechanism predominates during catabolysis (a severe type of malnutrition in which there is no longer any source of protein, carbohydrate, or vitamin nourishment feeding all body systems) and rises as fasting and starvation increase in severity.

The Role of Fats in a Healthy Diet

"Fat" is a bad word for most people trying to lose weight, but you can and should eat saturated fats such as coconut oil and palm oil (these are now known to increase your large-particle LDL cholesterol). Other good sources of fats that your body needs to stay healthy are organic monounsaturated fatty acid (MUFA)-rich foods such as avocados; organic nuts such as almonds, cashews, pecans, and macadamias; nut

butters; olive oil; olives; organic peanut oil; and sesame seeds or oil (beware of food allergies or sensitivities with agrochemical-contaminated nuts). If you can afford organic, buy and eat organic foods.

Avoid *trans* fats or hydrogenated fats. They tend to increase your small particle LDL cholesterol, which is implicated in the increased risk of heart attacks.

Follow the Prophet Daniel's Diet (Vegetables and Water Prescription)

For patients who have obesity problems, I often recommend what I call the Prophet Daniel's Diet. Following this diet for a month will bring the weight down a bit and encourage the patient to follow the low-glycemic index/load diet and the moderate-to-low protein diet. The Prophet Daniel's Diet will not work if patients love potatoes and hate water. Patients should drink enough water a day for success in any diet. Studies in England have shown that drinking half a liter (500 ml=16.9 fl oz) of water, thirty minutes before meals may induce a faster weight loss.

Here's the story of the Prophet Daniel's Diet (taken from Daniel 1: 1–21):

In the third year of the reign of Jehoiakim, King of Judah, Nebuchadnezzar (the King of Babylon) besieged Jerusalem. The Lord gave Jehoiakim into his hand, with some of the articles of the house of his god, which he carried into the land of Shinar to the house of his god; and he brought the articles into the treasure house of his god.

Then the king instructed Ashpenaz, the master of his eunuchs, to bring some of the children of Israel and some of the king's descendants and some of the nobles, young men in whom there was no blemish, but good-looking, gifted in all wisdom, possessing knowledge and quick to understand, who had ability to serve in the king's palace, and whom they might teach the language and literature of the Chaldeans. And the king appointed for them a daily provision of the king's delicacies and of the wine which he drank, and three years of training for them, so that at the end of that time they might serve before the king. Now from among those of the sons of Judah were Daniel, Hananiah, Mishael, and Azariah. To them the chief of eunuchs gave names: to Daniel, the name Belteshazzar; to Hananiah, Shadrach; to Mishael, Meshach; and to Azariah, Abed-Nego.

But Daniel purposed in his heart that he would not defile himself with the portions of the king's delicacies, nor with the wine that he drank; therefore, he requested of

the chief of eunuchs that he might not defile himself. Now God had brought Daniel into the favor and goodwill of the chief of the eunuchs. And the chief of the eunuchs said to Daniel, "I fear my lord the king, who has appointed your food and drink. For why should he see your faces looking worse than the young men who are your age? Then you would endanger my head before the king."

So Daniel said to the steward whom the chief of the eunuchs had set over Daniel, Hananiah, Mishael, and Azariah, "Please test your servants for ten days, and let them give us vegetables to eat and water to drink. Then let our appearance be examined before you, and the appearance of the young men who eat the portion of the king's delicacies; and as you see fit, so deal with your servants." So he consented with them in this matter, and tested them in ten days.

And at the end of ten days their features appeared better and fatter in flesh than all the young men who ate the portion of the king's delicacies. Thus the steward took away their portion of delicacies and the wine that they were to drink, and gave them vegetables. As for these four young men, God gave them knowledge and skill in all literature and wisdom; and Daniel had understanding in all visions and dreams.

Now at the end of the days, when the king had said that they should be brought in, the chief of the eunuchs brought them in before Nebuchadnezzar. Then the king interviewed them, and among them all none was found like Daniel, Hananiah, Mishael, and Azariah; therefore, they served before the king. And in all matters of wisdom and understanding about which the king examined them, he found them ten times better than all the magicians and astrologers who were in his entire realm. Thus Daniel continued until the first year of King Cyrus.

This may be considered the first clinical trial to compare delicacies and wine to vegetables and water. The conclusion is that vegetables and water are better for building muscle than delicacies and wine (alcoholic beverages in general). To lose weight, build muscle, and think better, it is imperative to start a diet of vegetables (organic, alkaline vegetables, and cruciferous vegetables to boost your good estrogen metabolite, 2-hydroxyestrone) and drink plenty of filtered water.

Table 25 is a list of alkaline vegetables and fruits, which are a great addition to any weight-loss diet.

Table 25. Eat Alkaline Vegetables and Fruits (make sure they are organic)

ALKALINE VEGETABLES		ALKALINE FRUITS
Beet Greens	Spinach, green	Apple
Beets	Spirulina	Apricot
Broccoli (cruciferous)	Sprouts	Avocado
Cabbage (cruciferous)	Sweet Potatoes	Banana (ripe banana has high glycemic)
Carrot	Tomatoes	Berries
Cauliflower (cruciferous)	Onions	Blackberries
Chard Greens		Cantaloupe
Chlorella	ORIENTAL VEGETABLES	Cherries, sour
Collard Greens (cruciferous)	Dandelion Root	Coconut, fresh
Cucumber	Kombu	Currants
Dandelions	Maitake	Figs, dried (avoid all dried fruits)
Dulce	Nori	Grapes
Edible Flowers	Reishi	Grapefruit
Eggplant	Shitake	Honeydew Melon
Fermented Vegetables	Umeboshi	Lemon
Green Beans	Wakame	Lime
Green Peas		Muskmelons
Kale		Nectarine
Kohlrabi (cruciferous)		Orange
Lettuce		Peach
Mushrooms		Pear
Mustard Greens		Pineapple
Nightshade Vegetables (tomatoes, pepper, paprika, eggplant, potatoes – avoid all these nightshade vegetables if you have arthritis)		Raspberries
Onions		Rhubarb
Parsnips (high glycemic)		Strawberries
Peas		Tangerine
Peppers		Tomato
Pumpkin		Umeboshi
Radishes		Plums
Rutabaga (cruciferous)		Watermelon
Sea Vegetables		

Weight-Loss Supplements

Supplementation with antioxidants such as vitamins C and L-carnitine has been growing rapidly in the past few years as research on harmful free radicals intensifies. Free radicals are atoms, ions, or molecules with one or more unpaired electrons that bind to and destroy cellular compounds. Dietary antioxidants disarm free radicals through a number of different mechanisms. Foremost, they bind to free electrons, "pairing up" with them, creating an innocuous cellular compound that the body eliminates as waste. The antioxidants in this formula also support and enhance the body's natural defense mechanisms against free radicals—the enzymes superoxide dismutase, catalase, and glutathione peroxidase. Recent research points to the fact that a synergistic combination of antioxidants is more effective than the total effect of each antioxidant taken alone.

In addition to antioxidants, supplementation with vitamin D and minerals is recommended. The benefit of antioxidants, minerals, and vitamin D goes beyond oxidation and achieves improved blood pressure as well as healthy glucose and lipid balance.

These are the ingredients of my recommended weight-loss supplements:

Caralluma Fimbriata, an appetite suppressant

Irvingia Gabonensis (African mango), an adiponectin booster

Bios Life Slim, a leptin booster

CLA (conjugated linoleic acid)

DIM + I-3-C + calcium-D-glucarate (2-hydroxyestrone boosters)

Chromium Polynicotinate

Fucoxanthin (Xanthitrim)

5-HTP (precursor for serotonin, reduces sugar craving)

NAC (N-acetyl-l-cysteine)

7-keto DHEA

Garcinia Cambogia

Gymnema Sylvestre

Apple cider vinegar

Ginger

L-arginine + ornithine = growth hormone support

Kelp

Selenium

Zinc

Glucomannan

Dandelion root

Chitosan

Raspberry ketones

Green tea extract

Potassium iodide

L-Tyrosine

Coleus forskholii

Ashwagandha

B-complex vitamins

Buffered ascorbic acid (vitamin C)

CoQ10

EPA/DHA

Vitamin E (mixed tocopherols)

Calcium

Vitamin D3

Magnesium

Zinc

Other minerals

Lowering Cholesterol Along with Losing Weight

High cholesterol is usually a signal that hormones in the body are out of balance and, therefore, need to be tested and corrected. Since LDL cholesterol is the principal ingredient used by the body to manufacture our good hormones, it may be a mistake to try to lower it. Many researchers are now arguing that it is not the high LDL in itself that causes atherosclerosis and, therefore, myocardial infarction (heart attack) and cerebral vascular accidents (strokes). It is the oxidation of the LDL cholesterol that represents the danger, and the LDL does not have to be high to oxidize.

The solution to the oxidation problem should, therefore, be prevention of the oxidation itself and not lowering the cholesterol level. Lowering the cholesterol level can be detrimental to health and may even lead to decreased longevity. DHEA is known as the longevity hormone. It is produced from pregnenolone, which, in turn, comes from cholesterol. Lowering the LDL or total cholesterol levels may lead to lowering our good hormone levels, and the hormone disequilibrium can lead to problems.

It is known that more people with low cholesterol die of myocardial infarction than people with high cholesterol. Cholesterol, if channeled to the right hormones, may be lifesaving. I highly recommend Uffe Ravnskov's book, *Ignore the Awkward! How the Cholesterol Myths Are Kept Alive.*

If oxidation is the problem, why don't we prevent this oxidation? It is rare to see healthcare providers recommending therapy that will prevent oxidation. I recommend to all my patients, including those as young as sixteen, that if they have obesity problems and high cholesterol, they should start on high-dose antioxidants, vitamin D, and minerals.

I usually recommend the following:

B-complex vitamins

Buffered Ascorbic Acid (vitamin C)

CoQ10

Vitamin E (make sure this is in the form of mixed tocopherols)

Omega-3 fish oil (EPA/DHA)

Acetyl-l-carnitine

Vitamin D

I also recommend multiminerals such as calcium, magnesium, potassium, zinc, manganese, iron, boron, iodine, chromium, selenium, vanadium, and molybdenum—and vitamin K2 when vitamin D and calcium are recommended. I try to avoid copper in patients with obesity, estrogen dominance, and hypothyroidism.

For patients with diabetes (remember that diabetics tend to have high cholesterol and high blood pressure due to hyperinsulinemia and high blood glucose levels), I also recommend the following:

Alpha lipoic acid

Cinnamon

Chromium and Benfotiamine (BenfoMax by Pure Encapsulations)

Broad-spectrum digestive enzymes (Digestive Enzymes Ultra or Digestive Enzymes Ultra with Betaine HCL) and probiotics (probiotic-50 B by Pure Encapsulations or Ortho Biotic 100 billion CFU by Orto Molecular) and prebiotics (FOS is commonly used). Many of you take probiotics without feeding them and them do not grow. Taking a prebiotic in addition to the probiotic insures growth of these beneficial bacteria.

There are many other nutritional supplements out there that aid in achieving optimal blood glucose, lipid, and blood pressure control.

Heavy Metal Decontamination Recommendations

According to CDC reports and many other accounts, heavy metal contamination is a real threat to the health of the U.S. population. Many healthcare providers, especially members of the Integrative Immunity, Functional Medicine, Anti-Aging and Regenerative Medicine groups, are recommending heavy metal decontamination via chelation and liver detoxification.

Since biomonitoring by the CDC has revealed that we are all contaminated, we should all obtain heavy metal testing periodically and engage in detoxification. Core Restore by Ortho Molecular is a good start. Any supplements that are used to detoxify the liver such as NAC, milk thistle, and spirulina/chlorella will also be useful. I also recommend HM Chelate by Pure Encapsulations for slow detoxification of heavy metals.

To antagonize estrogens, bioidentical progesterone should be administered along with foods such as cruciferous vegetables and nutritional supplements that reduce estrogen load (DIM, I3C, CDG, EstroDIM); and above all follow the Estrogen-Free Lifestyle.

Thyroid and androgen supplementation are already available. All these treatments will lead to an increased lifespan along with healthy aging. Remember that in biblical times, humans lived up to 350 years, and women were able to give birth at ninety!

Increased longevity and improved quality of life will not happen if healthcare professionals and the scientific community as a whole do not move outside the box to contemplate, observe, and discover new technologies and treatment modalities that will positively impact our health. This will require the coordination of efforts of all private and governmental institutions working together to save precious human lives. Discovering environmental woes and offering viable solutions seems small in comparison to the mountain of problems that are only getting worse; however, this step is a necessary beginning along the path of better living now and tomorrow. We have failed to achieve the “healthy people” goal set for 2010, but, by changing course now, this goal might be achievable by 2050.

PART IV: TREATMENT REGIMENS FOR ALLERGIC RHINITIS, ALLERGIC CONJUNCTIVITIS, AND FOOD ALLERGIES

ALLERGIC RHINITIS

The Orthomolecular Approach

Orthomolecular medicine, as conceptualized by double-Nobel laureate Linus Pauling, aims to restore the optimum environment of the body by correcting imbalances or deficiencies based on individual biochemistry using substances natural to the body such as vitamins, minerals, amino acids, trace elements, and fatty acids. The term "orthomolecular" was first used by Pauling in a paper he published in the journal, *Science,* in 1968.

The key idea in orthomolecular medicine is that genetic factors affect not only the physical characteristics of individuals, but also their biochemical milieu. Biochemical pathways of the body have significant genetic variability, and diseases such as atherosclerosis, cancer, schizophrenia, and depression are associated with specific biochemical abnormalities that are causal or contributing factors of the illness (definition from www.orthomed.org). Through anti-aging training programs, many healthcare providers are now learning the principles of orthomolecular medicine to better help their patients.

Biomonitoring, as reported by the CDC, has found harmful chemicals in human blood and urine, and many of these chemicals have been shown by scientific studies to increase estrogen, decrease thyroid function, and decrease androgens. These, as we have discussed, are endocrine-disrupting chemicals. Endocrine-disrupting chemicals are not only implicated in the obesity epidemic (and, obviously, obesity comorbidities such as diabetes, hypertension, hyperlipidemia, heart disease, and hypothyroidism), but also in the growing allergy epidemic (which we understand is chemical or estrogen-driven until proven otherwise).

How do you protect yourself and your family against pollution and this epidemic of internal biochemical disruption? I recommend nutritional supplements to enhance your immune system and for overall well-being. Nutritional supplements I have found useful in an allergy-treatment protocol are the following:

Nutrient 950 by Pure Encapsulations, a multivitamin that contains NAC (N-acetyl-l-cysteine), all the B-complex vitamins, ascorbic acid, vitamin D3, minerals (calcium, magnesium, zinc, manganese, molybdenum, chromium, and selenium, etc.), and other nutrients.

EFA Essentials (EPA/DHA, borage oil). Omega-3 fish oil blocks the inflammatory pathway that aspirin and ibuprofen block and, therefore, can help alleviate nasal symptoms.

Vitamin D3 helps to alleviate many inflammatory processes, but many people worldwide are deficient in this essential vitamin (hormone). If you live in the northern hemisphere, especially if you are inside most of the time, you are likely to have vitamin D deficiency. The 400 IU RDA (recommended daily allowance) is not enough, and many patients need 5,000-10,000 IU or more per day to keep their vitamin D level between the 50-100 ng/ml level deemed to be adequate (normal range is 30-100 ng/ml). If you have levels below 50 ng/ml, you should supplement.

Buffered ascorbic acid or vitamin C helps to alleviate many inflammatory processes and is also a natural laxative. If you have chronic constipation issues, you may be able to see improvement by using large doses of vitamin C and/or magnesium citrate. Start with buffered ascorbic acid 1,000 mg and increase progressively until you have a normal bowel movement, and keep taking that dose. If you develop diarrhea, it means you are taking too much. Vitamin C is also a good antioxidant and may help to clear free radicals from your body.

Co-enzyme Q10 (CoQ10) is a powerful antioxidant that is very useful. Sometimes, I recommend a combination of CoQ10 and its reduced form, ubiquinol.

The new kid on the block in terms of antioxidants is astaxanthin. You can try this product to help in the free-radical-detoxification process.

Vitamin E (make sure to use mixed tocopherols) is also an excellent antioxidant.

Aller-Essentials by Pure Encapsulations (which contains quercetin, vitamin C, Indian *Tinospora cordifolia,* hesperidin methyl chalcone, apple polyphenols), nettle (*Urtica dioica*), beta-glucan (1,3/1,6 glucan), and ubiquinol or CoQ10 are highly recommended for allergies.

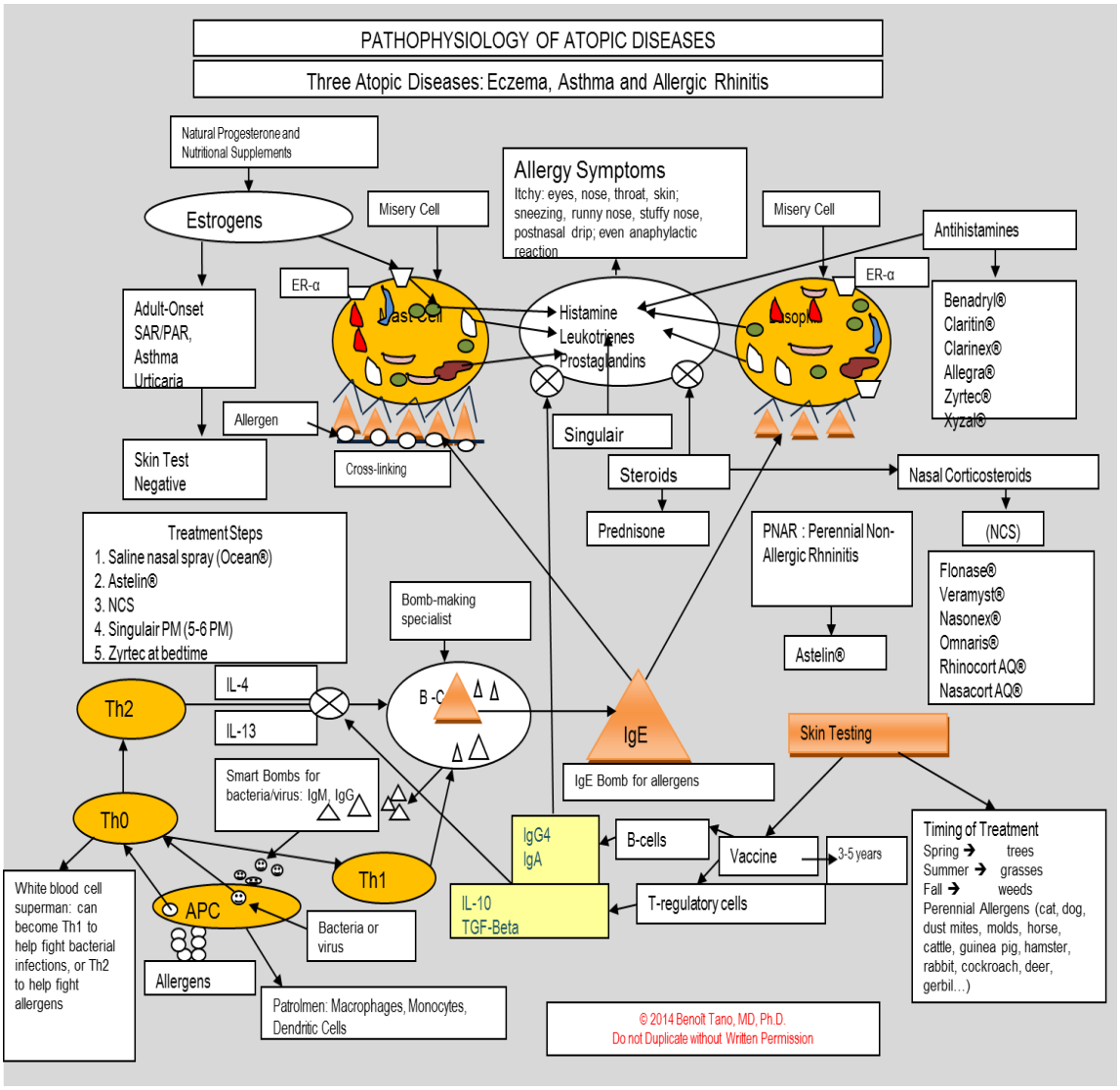
Please note: Nutritional supplements may interact with prescribed medications; therefore, you should consult with your healthcare provider prior to taking these supplements.

My Clinical Experience in Treating Allergic Rhinitis

Success in treating allergic rhinitis is based on the pathophysiology of this disease. When patients come to my clinic, I take a thorough history and perform a physical examination, after which I discuss the pathophysiology (the allergy-treatment roadmap) of atopic diseases.

The pathophysiology of atopic diseases diagram in figure 24 shows the interactions among the white blood cells that lead to the release of toxic mediators from the Misery Cells (mast cells and basophils). It explains how to block each of these toxic mediators (antihistamines to block histamine, leukotriene receptor antagonists to block leukotrienes, and nasal corticosteroids to block all these mediators). Antihistamine nasal sprays and combination nasal corticosteroid/antihistamine nasal sprays are used to treat multiple chemical sensitivities.

Figure 24. Atopic Disease Diagram



Immunotherapy for the Treatment of Allergic Rhinitis

All medication treatments for allergies are palliative and will not eliminate the allergy symptoms for a long period of time. To achieve long-term relief, it is necessary to identify the allergens involved in allergic reactions. Skin testing and blood testing are used for this purpose. Determining the offending allergens assists in specifying the timing of medication treatment. In general, fall allergy symptoms are due to weed pollens and year-round (perennial) allergy symptoms are due to animal dander, dust mites, molds, etc.

Spring allergy symptoms are usually due to tree pollens, and summer allergy symptoms are often due to grass pollens. Patients with spring or fall allergies should act pre-emptively by starting medication therapy two weeks prior to the beginning of the season instead of waiting for symptoms to arise. Patients with year-round allergies should treat symptoms year-round.

In addition to being used as a guide for the timing of the medication therapy, skin testing is used to determine the need for allergen immunotherapy (AIT), also known as the allergy vaccine. Allergen immunotherapy, conceived as long-term relief, is designed to specifically restore normal immunity against allergens. This treatment takes two forms: subcutaneous immunotherapy (SCIT), which is provided by injection, and sublingual immunotherapy (SLIT), which is taken orally in liquid or tablet form.

The vaccine stimulates T-regulatory cells and B-cells (the two groups of cells comprising the bomb squad unit; they disarm the bombs) to produce four major chemicals that induce tolerance of the offending allergens. T-regulatory cells produce IL-10 and TGF-beta, and B-cells produce IgG4 and IgA in the presence of allergen immunotherapy. These four "good chemicals" block IL-4 and IL-13 released from the Th2 cells and, therefore, block the communication line between the Th2 cells and the B-cells. If the Th2 cells do not communicate with the B-cells, IgE is not released from the B-cells. If there is no IgE, the mast cells and the basophils generally do not pour out their chemicals (degranulate). These good chemicals also block the mediators released from mast cells and basophils (histamine, leukotrienes, prostaglandins, and many more chemicals).

In response to allergen-specific immunotherapy, the effects of IL-10 and TGF-beta released by the T-regulatory cells include the following:

- Decreased IgE production by B-cells and increased production of IgA and IgG4
- Decreased IgE-dependent activation of mast cells and basophils
- Decreased activation and survival of eosinophils
- Suppressed mucus production by the epithelial cells and reduced airway hyperreactivity
- Decreased antigen presentation activity of dendritic cells

- Decreased Th2 cytokine production and suppression of its proliferation

Beneficial effects of allergen-specific immunotherapy include the following:

- Decrease in IL-4 and IL-5 production by CD4+ T cells
- A shift from Th2 cytokine pattern toward increased interferon-γ (IFN-γ) production

An increase in allergen-blocking IgG antibodies, particularly of the IgG4 class, which purportedly block allergens and IgE-facilitated antigen presentation

- Generation of IgE-modulating CD8+ T cells
- Reduction in the number of mast cells and eosinophils, including the release of mediators

Induction of a tolerant state in peripheral T-cells, an essential step in allergen-specific immunotherapy

Recent articles in prestigious American medical journals have indicated that SLIT is as effective as SCIT in stimulating T-regulatory cells and B-cells in treating allergies. SLIT is more popular in Europe, but it is making its way into allergy practice in the U.S. SCIT and SLIT are good, cost-effective, long-term treatment choices for patients with atopic diseases; however, SCIT carries a higher risk of anaphylactic reaction than SLIT.

I have treated patients with sublingual immunotherapy since 2007. My clinic (Integrative Immunity Health System [I-IHS], PC in Edina, Minnesota; website: www.integrativeimmunity.com) offers sublingual immunotherapy (SLIT) as well as subcutaneous immunotherapy (SCIT) or a combination of both SCIT and SLIT. Sublingual immunotherapy, should be administered frequently. The rationale for frequent administration is that, the more often the T-regulatory and B-cells are stimulated, the more they produce their good chemicals to induce tolerance. I often tell my patients to do their drops three to five times daily. Patients who follow the high frequency administration report significantly better results.

Chemical sensitivity and estrogen dominance are more often than not the culprits in allergies. Individuals who have estrogen dominance and nasal allergies and/or food allergies or food sensitivities can benefit from SLIT for the treatment of environmental and food allergies. Although medication therapy (antihistamines,

LTRA, nasal corticosteroids) used to prevent estrogen-induced mast cell and basophil-mediator release is the same as that used to prevent pollen-induced mediator release, patients with estrogen dominance may also benefit from bioidentical hormone therapy. I-IHS, therefore, offers treatment for obesity by balancing the hormones, especially neutralizing the estrogens through the EFL protocol and following a well-conceived BHRT protocol.

The Most Efficient Method for Alleviating Nasal Allergy Symptoms

Nasal allergy symptoms can be alleviated by blocking the chemicals involved in the allergic process—histamine and leukotrienes.

Immunotherapy (SCIT or SLIT) can be used to block the actions of the Th0 cell, the B-cells, mast cells, and basophils.

Treatment of Histamine-Related Symptoms

My first recommendation for treating histamine-related allergic rhinitis is saline spray.

If the saline spray is not sufficiently effective, there are many over-the-counter (i.e., nonprescription) oral antihistamines including diphenhydramine (Benadryl), loratadine (Claritin), fexofenadine (Allegra), and cetirizine (Zyrtec). Zyrtec, which became available over the counter in 2008, is one of the best antihistamines. Allegra is also very good.

If a patient has concomitant seasonal/perennial allergic rhinitis, combination therapy may be necessary for better relief of symptoms.

The most optimal combinations are the following:

1. Saline Nasal spray

2. Astelin or Patanase or Dymista (Astelin + Flonase combination) for people with chemical sensitivity

3. Nasal corticosteroids

4. Singulair (leukotriene-receptor antagonist, LTRA, for chronic stuffy nose, postnasal drip, or asthma sufferers)

5. Zyrtec or Xyzal

Treatment of Leukotriene-Related Symptoms

Top leukotriene blockers on the market include montelukast (Singulair), zafirlukast (Accolate), and others. These drugs are known as leukotriene-receptor antagonists (LTRA). Children and adults who have allergic rhinitis and asthma symptoms should use a combination of a good antihistamine, such as Zyrtec or Allegra, and a good LTRA, such as Singulair. This combination therapy will, in most cases, be optimal.

The mistake made by most patients who self-treat and also by many primary care providers is using these medications one at a time. Many people first try antihistamines alone and, if they do not get good relief, they switch to an LTRA. This stepwise approach to allergy treatment is not effective.

To block prostaglandins, which are released as part of the inflammatory process and cause pain, Advil Cold and Sinus is a good option.

Systemic Steroids

The master blockers of all the chemicals released by the Misery Cells are systemic steroids, which is the reason many healthcare providers inject steroids or give oral prednisone to their patients when they present with allergy symptoms. These systemic steroids, however, have multiple undesirable side effects in both adults and children and should be reserved only for short-term bursts for treatment of brittle asthma or other severe atopic inflammation.

For the treatment of allergic rhinitis, nasal corticosteroids (NCs) that act locally are more desirable. These include Flonase (fluticasone), which is now generic, OTC, and approved for ages four and older; Veramyst (fluticasone), also approved for ages four and older; Nasonex, which is most desirable for children is approved for ages two and older; Omnaris and Zetonna (ceclosenide), approved for ages six and older; QNasal (beclomethasone dipropionate), approved for ages twelve and older; Rhinocort AQ (budesonide), approved for ages six and older, and pregnant women with allergies; and Nasacort AQ (triamcinolone), approved for ages six and older and now available over-the-counter.

The bottom line: Treatment of allergic diseases requires combination therapy: antihistamines to block the histamine released from mast cells and basophils, LTRA to block leukotrienes, and nasal corticosteroids to block all of the chemicals that participate in allergic reactions. Orthomolecular, BHRT, and EFL are all used in the treatment of atopic diseases for better results.

ALLERGIC CONJUNCTIVITIS

Nasal allergy symptoms often include eye symptoms (itchy, watery, puffy, red eyes), known as allergic conjunctivitis. For allergic conjunctivitis, you may use an over-the-counter eye drop, such as Zaditor or prescription eye drops such as Patanol, Elestat, or Pataday. If you use prescription eye drops, do not expect results immediately. These prescription medications contain an antihistamine and a mast-cell stabilizer. It takes about two weeks for mast-cell stabilization, so be patient. In addition to these nasal medications and eye drops, I often recommend orthomolecular therapy, described earlier.

The Estrogen-Free-Lifestyle

Refer to Part III of this book to learn how to live an Estrogen-Free Lifestyle, which will eliminate many of the causative agents in allergic rhinitis; abdominal issues such as bloating, cramping, diarrhea, constipation, nausea, and vomiting; migraine headaches and other types of headaches; and mood swings.

FOOD ALLERGIES

The treatment of food allergies is the same as that for allergic rhinitis: blocking the chemicals released by the Misery Cells (mast cells and basophils). Antihistamines mentioned in the discussion of allergic rhinitis are used to block histamine and leukotriene-receptor antagonists are used to block leukotriene. Systemic steroids may sometimes be used to block the chemicals released by the Misery Cells.

As with allergic rhinitis, all medication treatments for food allergies are palliative. In addition, attempts to avoid certain foods may not prevent accidental exposures that could lead to anaphylactic reactions. To identify the allergens involved in allergic reactions, skin testing and blood testing are performed.

If a patient has both food allergies and environmental allergies, skin testing for the environmental allergies and a radioallergosorbent test (RAST) for suspected allergenic foods should be obtained. RAST should be used to test both the specific IgE for the suspected foods and the specific IgG for these foods.

Testing for the specific IgG or IgM (which is currently not done) for suspected foods is strongly encouraged. Often, adults present with a complaint of food sensitivities. Testing for the specific IgE of the foods will typically show negative or mildly positive results. It is the specific IgG for the foods that reveals an allergic response. As a general rule, if the specific IgG for the food has a concentration of 10 or greater (Class III), it is significant.

Individuals with food allergies often benefit from avoiding the offending foods or by receiving immunotherapy. I have found that foods that cause sensitivities (more common in women than in men) are those that are either estrogenic (phytoestrogens) or have become estrogenic because they have been contaminated by estrogenic environmental chemicals (pesticides such as atrazine, glyphosate, phthalates, etc.). I usually recommend avoiding these foods.

My recommendation hinges on the principle that we have an estrogen epidemic in the world today. Anyone with estrogen dominance should, therefore, avoid all forms of estrogens (phytoestrogens and xenoestrogens included) and adopt the Estrogen-Free Lifestyle.

Is It Gluten Sensitivity or Estrogen Sensitivity?

Many individuals complain of gluten sensitivity; therefore, health food and grocery stores are now stocked with multiple, gluten-free products to satisfy the needs of

these individuals. This gluten sensitivity has reached epidemic proportions, and it is more common in women. My hypothesis is that this gluten sensitivity may be due to estrogen sensitivity, especially in women. In the 1970s and even early 1980s when the estrogen load in our environment was not as high as it is now, we all ate gluten grains without negative consequences. In the 1990s when the estrogen load increased tremendously with the extensive use of Glyphosate and other estrogenic chemicals in our food production, many individuals, and especially women, started to feel the extreme estrogen-dominance effects of the estrogenic foods (phytoestrogens).

Wheat, barley, rye, spelt, kamut, and millet are weak phytoestrogens. As are other foods and common chemicals listed in the Xenoestrogens and Phytoestrogens list shown earlier. Women who already have too much estrogen will experience abdominal bloating, cramping, constipation and/or diarrhea, migraine headaches, and mood swings as well as many of the other estrogen-dominance symptoms when they consume gluten products. Women with estrogen dominance who experience estrogen-related symptoms often undergo celiac disease testing that turns out to be negative, which is often baffling to their practitioners. These patients are referred to allergists in the hope that skin testing will reveal an IgE-mediated wheat or other gluten-containing grain allergy. In these cases, the skin test is almost always negative. Patients insist they have a gluten allergy and will not eat gluten products in the hope of alleviating their symptoms; however, balancing their hormones and avoiding the gluten-containing foods strictly is what is necessary to resolve their gluten-sensitivity problem. Rotation diet will not help in this case. Rotation diet means going back to gluten at some point. When patients go back to gluten, they tell me that they paid the price (their symptoms return). Patients with fibromyalgia do better when they eliminate gluten-containing grains, nightshade vegetables, balance their hormones, and adhere to EFL strictly.

Treatment of Food Allergies

As with allergic rhinitis, skin testing and the allergy vaccine may be used to treat food allergies and sensitivities. Allergen immunotherapy is also an option. SLIT is the only form of immunotherapy used to treat food allergies. It is well tolerated by patients and has fewer side effects than SCIT, even in severe food allergy cases. Over time, the T-regulatory cells and B-cells are able to recognize food allergens and induce tolerance.

The Estrogen-Free Lifestyle

Refer to Part III of this book to learn how to live an Estrogen-Free Lifestyle, which will eliminate many of the causative agents in food allergies and sensitivities.

Diagnosis and Treatment of Food Allergies and Sensitivities

Successful treatment of food allergies or food sensitivities is based on the pathophysiology of these immune reactions. When patients come to my clinic, I take a thorough history, and perform a physical examination, and then discuss with the patient the pathophysiology of food allergies along with diagnosis and treatment strategies, as described previously in the section on allergic rhinitis. These strategies include skin testing to determine offending allergens, avoidance of allergy-inducing foods, and immunotherapy that is focused on testing for the specific IgE and IgG in suspected foods.

A WORD ABOUT AUTOIMMUNE DISEASES

I personally think that we should get rid of the term "autoimmune disease" because the body is not crazy and it is not attacking itself. The body is attacking substances that it does not recognize as safe. The body is attacking the environmental chemicals and the chemical-food complexes that have become antigenic. These food antigen-antibody complexes circulate in the blood and lodge in small vessels to cause inflammation. This inflammation leads to immune-cell reactions that perpetuate the inflammation and lead to immune complex diseases (ICDs) such as lupus erythematosus, rheumatoid arthritis, multiple sclerosis, and many others.

Antigen-presenting cells know how to deal with clean peptides; however, with the mounting environmental pollution, food and water now contain chemical toxins. When these toxins link up with food molecules, they form a blob that the immune cells recognize as an antigen. B-cells make antibodies (IgE, IgG, IgA, and IgM) against these chemical-laden food molecules. The antibodies bind the food antigens and form chemical complexes that circulate in the blood and set off the classical complement pathway that leads to direct cell damage. These antibody-antigen complexes are often involved in what is generally called autoimmune diseases.

Prior to the massive pollution of Earth, antigen-presenting cells were seeing only clean peptides and had a normal, noninflammatory response to these peptides. Pollution of our environment has caused antigen-presenting cells to identify these toxic food molecules as dangerous and antigenic. It is as if the body is attacking itself, but remember, there is no smoke without fire. The body is attacking something, but not itself; and that is the reason I do not like the term "autoimmune."

Estrogens make up the bulk of environmental chemicals. Because women have more estrogen than men, they have a greater likelihood of developing immune complex diseases (commonly called autoimmune diseases).

Th2 cells cause B-cells to produce IgE, which is involved in immediate hypersensitivity allergic reactions such as anaphylactic reactions. Again, adult-onset food allergies, urticaria, and angioedema (body swelling) are more prevalent in women because of the estrogen effect. Many women who react to foods tend to react to phytoestrogen foods such as soybeans, barley, rye, wheat, GMO corn, dairy (currently, 25 chemicals are found in milk in addition to growth hormones), sugars (GMO sugar beets), and spices such as rosemary, thyme, and some vegetables such as celery. If you have eaten shellfish all of your life and then, one day, you have a reaction to shellfish, question the spices used in preparing the shellfish and not the shellfish itself. For example, Old Bay seasoning, which is often used in a crab boil, has five ingredients that are phytoestrogens.

In these cases, skin testing for the offending foods is often negative. This indicates that estrogens are causing the reactions, not the proteins in the foods. Recall that estrogens have receptors on mast cells and basophils and can cause direct histamine and leukotriene release just like true antigens do.

SUMMARY

Environmental pollution continues due to population pressures and efforts to feed a growing population. Farming for the masses uses synthetic chemicals that contaminate foods and water sources. This pollution leads to hypothyroidism, hyperestrogenism, and hypoandrogenism, which contribute to obesity and its comorbidities. High estrogen levels lead to high insulin which leads to craving for sugar-containing foods that quickly convert to glucose in the body. High blood glucose must be quickly disposed of to prevent damage to blood vessels and organs; insulin takes care of this excess glucose by storing it in adipocytes. When adipocytes (fat cells) become saturated with glucose, they release leptin to signal to the hypothalamus to stop the body from producing glucose.

The hypothalamus acts to reduce the appetite and cravings to allow the adipocytes to decompress. This decompression process is slow, and stresses the adipocytes, which release more leptin to make their case. Unfortunately, this plea falls on deaf ears. The message is "plenty, but ineffective." This inefficiency is known as leptin resistance. When the hypothalamus does not respond, the adipocytes shield themselves, and insulin, although sufficient, has little capacity to push the glucose into the cells. This insulin incapacity is known as insulin resistance.

Insulin resistance leads to excess glucose in the bloodstream. This is known as type 2 diabetes. Treatment of diabetes uses three groups of medications: one group, called sulfonylureas, increases the amount of insulin produced by the pancreas. A second group, thiazolinediones, increases the capacity of the insulin to deposit more glucose into the adipocytes. The third group suppresses new glucose formation in the liver; the prototype in this group is the drug metformin**.** This group also has a second effect, which is to help insulin move glucose into the adipocytes.

In advanced type 2 diabetes, the pancreas becomes burned out and is unable to produce insulin. When this happens, insulin injections must be given. All these medications lead to a greater risk of obesity and exacerbate the vicious cycle of insulin resistance and increased blood glucose. It is, therefore, no surprise that diabetic patients never get better; eventually, they meet their demise in the form of end-stage organ failure.

Leptin resistance leads to a decrease in serotonin, the "feel-good hormone." Decreased serotonin leads to nervousness, anxiety, panic attacks, depression, and mood swings as well as conditions such as bipolar disorder. These psychiatric symptoms are often treated with antidepressants such as selective serotonin reuptake inhibitors (SSRIs) and mood stabilizers. SSRIs include fluoxetine and paroxetine. Notice the "Fluo" in fluoxetine is the same "fluo" in the thyroid-killing chemicals (PFCs) that are abundant in the environment. SSRIs and mood stabilizers may result in weight gain and, therefore, also perpetuate the vicious cycle.

Obesity seems to strike young adults, especially women in their prime (ages 18–44). This group is at increased risk because of the elevated levels of endogenous estrogens that couple with xenoestrogens and phytoestrogens to raise insulin levels and contribute to hypothyroidism, both of which are major causes of obesity. High insulin levels also contribute to obesity-related comorbidities such as diabetes, hypertension, hyperlipidemia, and heart disease, which also require treatment. Hypertension medications such as beta-blockers lead to decreased thyroid function

and often cause weight gain and many other unwanted side effects such as decreased libido.

Treatment of hyperlipidemia frequently requires the use of statin drugs, which are effective in reducing total cholesterol and LDL cholesterol; however, LDL is the starting material for sex hormones. Low cholesterol, therefore, leads to low testosterone and even lower progesterone; hence, making estrogen dominance worse. Recall that estrogen may be produced by the adrenal glands, skin cells, and conversion of androgens such as testosterone to estrogen via aromatase, which is produced in large quantities by adipocytes. This is in addition to ovarian production. In menopause, the ovarian function stops, but the estrogen-making machinery and estrogen production do not.

Low testosterone leads to decreased libido, a source of marital discord and divorce, but low testosterone also leads to obesity due to the increased insulin, which is a side effect. When testosterone gets converted to estrogens (estradiol and estrone), these estrogens couple with xenoestrogens and phytoestrogens in men to cause "man boobs," benign prostatic hyperplasia, and even prostate cancer.

Too much estrogen in women causes benign and malignant tumors; however, treatment of estrogen-related tumors rarely involves measurement of estrogens. Many estrogen-induced conditions that lead to hysterectomies are the result of endogenous estrogens, xenoestrogens, phytoestrogens, and synthetic hormone replacement therapy (i.e., estrogen supplementation prescribed to treat perimenopause and postmenopausal symptoms such as hot flashes). This exogenous estrogen, paired with the already high endogenous estrogen, xenoestrogens, and phytoestrogens, leads to ongoing estrogen dominance symptoms. Because estrogen dominance is never addressed, the symptoms are never corrected.

Estrogen dominance can be suspected if a patient has had a hysterectomy, a cholecystectomy, menstrual disturbances, tumors and cancers, infertility issues, and weight gain.

A basic hormone panel and treatment of any hormone imbalance will be cost-effective in treating a plethora of diseases and disorders. Hormone evaluation should be initiated at age thirty for most women and men, or earlier if patients exhibit symptoms of progesterone deficiency, androgen deficiency or excess (as in PCOS, for example), and estrogen dominance.

In this book, we have seen how pesticides are leading contributors to the epidemic of estrogen dominance and its resulting disorders and diseases, in particular, obesity, atopic diseases (eczema, asthma, and allergic rhinitis), and food allergies. Success in treating these diseases lies in recognizing the sources of estrogens (endogenous and/or exogenous) and eliminating them, as much as possible, from our diet and our environment, and by living the Estrogen-Free Lifestyle.

APPENDICES

APPENDIX A

ILLUSTRATIVE CASE STUDY

Chief Complaint: Evaluation of Seasonal/Perennial Allergic Rhinitis, Perennial Nonallergic Rhinitis (Multiple Chemical Sensitivities), and Asthma

History of Present Illness

Tina is a 45-year-old woman who grew up in the Midwest. She was diagnosed with bronchitis when she was in high school. She has continued to have shortness of breath with upper respiratory infections and season changes. She also has runny nose, stuffy nose, sneezing, itchy and watery eyes, postnasal drip, coughing, wheezing, and shortness of breath. These symptoms have become progressively worse since her midthirties. She does not get any sinus infections.

Allergy triggers are pollens, dust, molds, cigarette smoke, perfumes, household cleaning agents, and petrochemicals (car exhaust and diesel fuel). Symptoms are year-round, but tend to get worse in the spring and fall. She uses OTC (over-the-counter) antihistamines (especially Zyrtec) and occasionally that partially helps.

When she was in high school, she developed aches and pains and had painful menstrual cramps, heavy bleeding, and endometriosis. She briefly took birth control pills to control the menstrual symptoms, but had to stop because she developed a blood clot. At age 21, she had her first pregnancy and had hyperemesis gravidarum (nausea and vomiting during the first to second trimester of pregnancy) and weight loss until about five months. She was not overweight at that time. At age 27, she became pregnant again after taking fertility drugs and felt better during that pregnancy. Over the next 10 years, the menstrual pain became so intense that she requested a hysterectomy. She also developed cholecystitis (inflammation of the gallbladder) and had a cholecystectomy around the time of the hysterectomy.

She felt better after the hysterectomy, but quickly gained more than100 pounds and, since that time, she has continued to gain weight. She now weighs 250 pounds. She has tried diets, exercise, and whole foods; she lost some weight, but then gained all of it back. She also has anxiety, depression, and insomnia, and has been diagnosed with fibromyalgia.

She was having migraine headaches before her periods. Since her midthirties, she has migraines all the time and takes Imitrex sometimes for these headaches. She was

recently started on Prozac for her anxiety and depression and propranolol for her chronic headaches. She has seen a chiropractor and tried food supplements, but these have not helped. She is in my clinic for evaluation of her allergies. She indicates that she has gone from practitioner to practitioner without any symptoms relief. She relates that she is frustrated, and she is sick and tired of feeling sick all the time and not being able to lose the weight.

Allergy Profile

She has no eczema, but she has asthma and food sensitivities. She reacted to Penicillin (hives) when she was a child and has not received any Penicillin treatment as an adult. She has no insect sting allergies.

Family History

Both parents are alive and have allergies. She has a brother and a sister who also have allergies. She has a son and a daughter. Her daughter has allergies, but her son has no allergies.

Social history

Tina is married, she lived on a farm as a child and continues to live in a farming community. She is a high school teacher and has a lot of stress at work. She has noticed that her allergy symptoms get worse during the spring when the farmers spray chemicals on their crops and in the fall during harvest, when farmers are "combining corn and soybeans." She does not smoke. She drinks beer occasionally. Whenever she drinks beer, she develops flushing on the face and chest. She has also noted flushing of the chest when she is nervous.

Review of Systems

General: No activity change, has increased appetite, no fevers but has chills, has excessive sweating and hot flashes. She has weight gain, decreased libido, fatigue, some degree of apathy, and foggy thinking.

No ear discharge, ear pain, or fullness in the ears, but has ringing in the ears. No nosebleeds, but has stuffy nose and sneezing. No lip swelling or tongue swelling. No dental problems, drooling, or mouth sores, but has sore throat without difficulty swallowing or voice change. No eye discharge, has itchy, watery eyes, without redness of the eyes, light sensitivity, or visual disturbance.

Neck: She has neck pain and neck stiffness; and she sees the chiropractor, which helps.

Cardiovascular: She has no chest pain, but gets occasional palpitations. She has no leg swelling. She has been diagnosed with hyperlipidemia, and she is on Lipitor.

Respiratory: She has sleep apnea, but no chest tightness, choking, coughing, shortness of breath, or wheezing today.

Abdomen: She has abdominal distention, bloating, pain in the abdomen, but no bloody stools. She has alternating constipation and diarrhea, and has been diagnosed with IBS. She has no nausea or vomiting.

Genitourinary: No difficulty urinating, no painful urination, increased frequency of urination, genital sores, or blood in the urine. She has some urinary incontinence. Menstrual history is as above.

Musculoskeletal: She has arthritis, back pain, gait problems, joint swelling, and muscle aches.

Skin: She has darkening of the skin in the neck and both axillary areas, no pallor, no rash, and no wounds/sores.

Neurological: She has headaches frequently, and dizziness; she gets lightheaded and shaky (especially around noon when she feels hungry); she has no numbness, seizures, speech difficulty, syncope (passing out), tremors, or weakness.

Hematological: She has no lumps in the neck, nor does she bruise or bleed easily.

Psychiatric: She has agitation, behavioral problems, decreased concentration, and depressed mood. She has nervousness and anxiety. She takes Prozac and sees a therapist, but reports no hallucinations or hyperactivity. She has had insomnia for a long time. She has no suicidal ideation.

Physical Exam

Vitals: Temperature 97.5 BP: 110/78 (she has always had low blood pressure) Heart Rate: 98 Respiratory Rate: 12 Weight: 248.

General: Well-developed, well-nourished, obese-appearing woman in no apparent distress.

Head: Atraumatic, normocephalic

Eyes: Pupils are of equal size, round, reactive to light, direct and consensual, no conjunctiva injection, or sclera icterus.

Ears: Canals are patent, tympanic membranes are not injected.

Nose: Nares are patent, there is moderate swelling of the inferior turbinates bilaterally, mucus noted in both nostrils, mild excoriation of the nasal membranes noted in the left nostril.

Mouth: Mucosa is moist, no lesions noted.

Throat: Has mild erythema secondary to postnasal drip.

Neck: Supple, no lymphadenopathy, no thyromegaly, carotid pulses are equal with no bruits. The neck has darkening of the skin.

Lungs/Chest: Lungs are clear to auscultation bilaterally, no wheezing, rales or rhonchi noted today.

Heart/Cardiovascular: Normal S1, S2, regular rate and rhythm, no murmurs, rubs, or gallops.

Abdomen: Normal bowel sounds, nontender, nondistended obese abdomen with linear striae (stretch marks) diffusely distributed on the abdomen.

Extremities: Normal pulses, no cyanosis, clubbing or edema noted, and striae noted on the upper extremities. The upper extremities also have fine bumps on the upper arms area consistent with keratosis pilaris (due to vitamin A and/or vitamin D deficiency).

Skin: No rash noted, but she has dry skin.

Neuro: Tina is alert, oriented x 3. DTRs are equal and adequate in upper and lower extremities. CN II-XII grossly intact.

Analysis of the Case: How did I use Integrative Immunity technique to address Tina's presenting complaints? Before going into the case analysis, look at an inventory of the most common complaints that patients presented within 21st century medical clinics and hospitals worldwide.

Common 21st Century Symptoms and Signs of Diseases Seen in Clinics and Hospitals Worldwide

Among the most common symptoms seen in patients worldwide in the 21st century are the following: environmental allergies, food allergies and food sensitivities, chronic sinusitis, asthma, atopic dermatitis (eczema), migraine headaches, hair loss (alopecia), hyperlipidemia, hypertension, diabetes, cardiovascular diseases, abdominal issues (bloating, cramping, constipation/diarrhea, and IBS symptoms), candidiasis, candida-related vulvovaginitis, recurrent UTIs, dysmenorrhea (painful menstrual cramps), menorrhagia (heavy menstrual bleeding), irregular menses, ovarian cysts, polycystic ovary syndrome (PCOS), fibroid tumors, fibrocystic breast disease, endometriosis, cervical dysplasia with abnormal Pap smear, BPH (benign prostatic hypertrophy) in men with increased frequency of urination, nocturia (nighttime frequent urination that affects sleep), arthritis, fibromyalgia (body aches and pains), urticaria (hives), angioedema (facial swelling, lip swelling, tongue swelling, and even whole body swelling), hypoglycemic episodes and hypotension leading to lightheadedness and dizziness, agitation, anger, behavioral problems, grumpiness, decreased concentration, depression, anxiety, nervousness, panic attacks, decreased libido, sleep disturbances, eating disorders (eating too much or too little), abnormal weight gain, morbid obesity, hypothyroidism, Graves' disease, Hashimoto's thyroiditis, autoimmune diseases (such as lupus, rheumatoid arthritis, multiple sclerosis and many others), hot flashes, night sweats, foggy thinking, fatigue, cancers, and many other signs and symptoms not listed here.

Clinical Approach

How did I approach Tina's complaints? She came to the clinic for allergy evaluation, but as you notice, she has more than allergies to address. Should I simply focus on her allergies? What should be done about all her other symptoms? She clearly shows her frustration about not getting any help from previous practitioners.

1. Tina grew up in the Midwest and lives in a farming community.
   If you have read this book to this point, then you know that the Midwest and the South have the highest obesity rates and higher obesity comorbidities than other regions. It is, therefore, not surprising that Tina is obese and has many obesity comorbidities. Her obesity started in her midthirties. That gave me a clue that her hormones were shifting in her midthirties, and she became estrogen dominant. Estrogen dominance led to an increase in her insulin, an increase in her thyroid-binding globulin (TBG) and both led to the weight gain. As she was gaining weight, she developed insulin resistance, which led

to her hypoglycemic episodes (lightheadedness and shaking around noon). She also developed leptin resistance that led to a decrease in her serotonin and, therefore, led to the moods swings (depression, anxiety, and panic attacks). The weight gain also led to decreased libido, fatigue, apathy, and foggy thinking—consistent with low testosterone and low thyroid function. The low testosterone was due to three things: low progesterone, low DHEA, and activity of the fat cells that were converting her testosterone to estrogen.

2. When Tina was in her late twenties, she had menstrual cycles without ovulation and, therefore, had low progesterone, which was the reason for her infertility. She became pregnant after receiving fertility drugs. During the next 10 years, she progressively gained weight and had menstrual disturbances because of her low progesterone and increased estrogen. The result was the hysterectomy. The low progesterone also contributed to her weight gain because her thyroid function decreased. Because of the low progesterone, she could not keep important minerals, such as zinc, potassium, iodine, and selenium in her cells and, therefore, was unable to convert her thyroid prohormone T4 (thyroxine) to the active form T3 (triiodothyronine).
   She lives in a farming community and her minerals may be low due to chelation of these minerals by many of the pesticides (Roundup, for example, is a mineral chelator). Iron, magnesium, zinc, iodine, and many other mineral deficiencies are now common in the U.S. and the rest of the world.

3. The estrogen dominance also led to the chronic migraine headaches that required propranolol (a beta-blocker that slows down thyroid function) and made her low- functioning thyroid even worse and resulted in additional weight gain. She was started on Prozac (fluoxetine) and as you have learned, all the fluo-containing drugs and environmental PFCs (perfluorocarbons) have a negative effect on the thyroid. She, therefore, has two medications that are contributing to low thyroid, weight gain, fatigue, more anxiety, depression, and aches and pains (fibromyalgia symptoms). Remember that the fat cells are pro-inflammatory and can increase IL-6 and TNF-alpha, which can cause aches and pains.

4. In her midthirties, her allergies became worse, and she was having more asthma flare and more nasal symptoms. Notice that she was still living in her same environment. So what changed that made her allergy symptoms worse?

If you have read this book to this point, you realize that endogenous estrogens coupled with environmental estrogens make allergy symptoms worse because estrogens have receptors on mast cells and basophils. When estrogens attach to their receptors, they cause histamine and leukotriene release just like pollens and other environmental allergens do. Organophosphate and carbamate insecticides also cause mast cells and basophils to release histamine and leukotrienes. Tina also developed gluten sensitivity and had diarrhea whenever she ingested gluten-containing grains (including beer that has hops). Remember that gluten grains are phytoestrogens and women, who have more estrogen than men, tend to develop more gluten sensitivity and even more celiac disease than men.

5. On physical examination, she was found to have striae on arms, abdomen, thighs, abdominal obesity, and obese thighs and buttocks. This type of body morphology is consistent with too much estrogen and too much cortisol and insulin. Tina's work is demanding and stressful. She is, therefore, converting more of her beginning hormone pregnenolone to cortisol to help her cope with the stress. The increase in cortisol and insulin led to deposition of fat in her abdomen and the abdominal obesity followed. Tina has high cholesterol, and she is on a statin drug (Lipitor) to reduce her cholesterol. The high LDL cholesterol is consistent with her hormone imbalance state. The LDL cholesterol is the starting material to make the hormones. Usually it is made in the liver and then goes to the gallbladder to mix with bile for better digestion of fat. When Tina's hormones became imbalanced, the body wanted to correct them by increasing her LDL cholesterol; however, she had low bile salts in her gallbladder and the cholesterol could not mix with her bile. She, therefore, developed gallstones that led to the cholecystitis (inflammation of the gallbladder) and surgical removal of her gallbladder (cholecystectomy). Her chronic low blood pressure is due to her long-standing low progesterone that leads to low aldosterone that, in turn, leads to the low blood pressure.

Based on my analysis of Tina's case, I was able to categorize her complaints as estrogen-related problems and progesterone-related problems as shown below in the differential diagnosis.

Differential Diagnosis

Estrogen Excess Problems	Progesterone Deficiency Problems
Nasal allergies	Dysmenorrhea (painful menstrual cramps)
Asthma	Menorrhagia (heavy menstrual bleeding)
Multiple chemical sensitivity	Endometriosis
BCP-induced blood clot	Hyperemesis gravidarum (severe nausea and vomiting during pregnancy)
Abnormal weight gain	Postpartum depression
Morbid obesity	Fibromyalgia
Food allergy (gluten sensitivity)	Infertility
Anxiety/depression/panic attacks	
Flushing of the face/chest with or without beer ingestion	
Abdominal bloating/cramping/diarrhea with gluten ingestion	
Premenstrual migraines	
Chronic migraines	

Based on Tina's Complaints and Findings, What Laboratory Tests Should Be Ordered?

The laboratory tests to be ordered should be used to evaluate as many of Tina's complaints as possible.

General Laboratory Workup for Integrative Immunity Evaluation

If you suspect hormone imbalance syndrome, you should ask your physician to order tests for progesterone, DHEA, DHEA-S, androstenedione, estradiol, estrone, four-

point cortisol test (saliva test), and aldosterone (if you have or suspect hypertension or hypotension). Fasting insulin and leptin levels should be added if you are obese. Also, request a lipid panel (LPP test is better than general lipid panel), comprehensive metabolic panel, CBC with differential, total and free testosterone, dihydrotestosterone (DHT), sex hormone binding globulin (SHBG), FSH and LH, homocysteine (included in LPP test), high sensitivity C-Reactive Protein (hsCRP), TSH, free T4, free T3, and reverse T3 (rT3), somatomedin–C (IGF-1), and IGFBP3.

Genova Diagnostics has a hormonal health kit for women and one for men that is reproducible. I have used that test for both women and men with good results. Genova Diagnostics also has a urine test that may be good, but, I prefer the serum test. Many providers go for saliva test because saliva test providers have fought hard to make it the gold standard; however, I usually give sublingual and topical progesterone and the saliva test tend to exaggerate the repeat testing results. The saliva test does not give me any of the estrogen metabolites (2-hydroxyestrone and 16-alpha-hydroxyestrone). The serum test gives me this information, and I can use it to recommend cruciferous vegetables extracts such as DIM and I3C and/or cruciferous vegetables consumption and other recommendations to boost the 2-hydroxyestrone level. Using the hormonal health kit for women and men over the years has been beneficial for the patients and their follow-up. If a patient fails to follow her/his BHRT protocol as prescribed, I can generally tell by looking at the repeat test results.

Test for evaluation of nutritional deficiency and MTHFR gene mutation: New NutrEval (Genova Diagnostics)

Test for evaluation of intestinal malabsorption and gastro-intestinal parasites (GI Effects or CDSA tests by Genova Diagnostics and the Biohealth 401H test; a dual GI Effects and Biohealth 401H tests is often optimal)

Test for heavy metals (serum test or urine test by Genova Diagnostics)

The appropriate test for lipids is the Lipoprotein Particle Testing (LPP); the SpectraCell LPP test kit is a good test. Let your practitioner know that he or she can contact SpectraCell to set up a free account. When you know your LDL particles size, your physician can do a better job at controlling your cholesterol. In many cases, all you need is to balance your hormones by following EFL, using Niacin (vitamin B3), antioxidants, and plant sterols. Blindly lowering total LDL may be detrimental to your health (if your LDL cholesterol decreases too much, guess what else goes out of balance? It is your hormones). Preventing oxidation of LDL by using antioxidants makes more sense.

For Tina, the following laboratory tests were ordered:

Labs: Skin tests for trees, grasses, weeds, molds (including Fusarium), dust mites, and animal dander.

RAST (both ImmunoCap specific IgE and specific IgG): for foods (milk, casein, egg, peanut, wheat, soybean, corn, garlic, gluten, barley, rye, oat, rice, baker's yeast) and candida were ordered.

Additional Tests Ordered: Gluten sensitivity/celiac disease test: antigliadin IgA and IgG, tissue transglutaminase IgA for celiac disease test. CBC with differential, comprehensive metabolic panel (test for liver function, kidney function, and minerals - sodium, potassium, calcium, chloride), ferritin level, hsCRP (high sensitive c-reactive protein, a marker of inflammation but also cardiac risk), vitamin D level, fasting insulin, lipid panel (LPP test by SpectraCell - that fractionates the LDL into particles sizes), leptin level, thyroid function test (TSH, free T4, free T3, reverse T3-rT3, thyroid peroxidase antibody (TPO), thyroglobulin antibody).

Hormone Testing: progesterone, SHBG, DHEA, testosterone, free androgen index, androstenedione, FSH, LH, estradiol, estrone (E1s, E1), and Estriol; estrogen metabolites (2-hydroxyestrone and 16 alpha-hydroxyestrone), pregnenolone, and prolactin.

Initial Assessment

1. Seasonal/perennial allergic rhinitis
2. Perennial nonallergic rhinitis
3. Mild intermittent asthma
4. Perimenopause disorder (even though Tina had a hysterectomy, it was a partial hysterectomy, and she still has her ovaries)
5. Underactive thyroid
6. Metabolic syndrome (morbid obesity, coronary artery disease, hyperlipidemia)
7. Irritable bowel syndrome
8. Depression and anxiety
9. Fibromyalgia

Initial Plan

1. Seasonal/Perennial allergic rhinitis plan: Pathophysiology of SAR/PAR, PNAR discussed. Start on Mometasone Furoate (Nasonex) 2 x 2 x 2 (2 sprays in each nostril, twice daily) and cetirizine (Zyrtec) 10 mg at bedtime. Use buffered isotonic saline nasal spray (Ocean Saline Nasal Spray is one of the best) as a moisturizer for the nose.
2. Perennial nonallergic rhinitis plan: Start on azelastine nasal spray 2 x 2 x 2 for the chemical sensitivity component of her rhinitis symptoms.
3. Mild intermittent asthma plan: Start on Singulair 10 mg every evening (5-6 pm) for control of the asthma, and use albuterol inhaler only as a rescue medication. When nasal symptoms improve, then the mild intermittent asthma symptoms may also subside.
4. Immunotherapy plan: Tina has opted for SLIT for inhalants and risks and benefits discussed. SLIT for inhalants dispensed today.
5. Perimenopausal disorder plan: A hormone test kit provided today, and when the results are available, further management decisions will be made.
6. Underactive thyroid/Fibromyalgia plan: Thyroid function test including TSH, free T3, free T4, reverse T3, antithyroglobulin and thyroid peroxidase antibodies ordered today. When the results are available, further management decisions will be made.
7. Metabolic syndrome plan: I have recommended EFL (Estrogen-Free Lifestyle) and she will avoid some grains (GMO-corn, barley, rye, wheat), dairy, sugars, potatoes, and eat organic foods as much as possible. She will also adhere to a low-glycemic-index diet and avoid xenoestrogens and phytoestrogens (list provided in clinic today) for an optimal weight control and for overall well-being. I have recommended nutritional supplements (Nutrient 950 with NAC, EFA Essentials, Buffered Ascorbic Acid, Magnesium Glycinate, RivitalAge Ultra, Vitamin D3, CoQ10, and Bios Life slim supplement) for her overall well-being and for an optimal weight control.
8. Irritable bowel syndrome plan: The food allergy test is useful to rule out any food allergies that may be contributing to her symptoms. Gi Effect stool analysis kit provided. When the results are available, further management decisions will be made. Tina is already on probiotics and should continue same and should add digestive enzymes and a prebiotic such as (FOS).
9. Depression/anxiety plan: Emotional Wellness (5-HTP, and L-Theanine combination), melatonin, and EFA were recommended to help alleviate some of her symptoms. When her estrogen load decreases, her mood swings may also improve.

10. Fibromyalgia Plan: Magnesium Glycinate (or Threonate) at bedtime and balancing the hormones may help.

Laboratory Results and Interpretation

Skin testing today was positive for trees, grasses, weeds, molds, cat dander, dog dander, and dust mites. In addition to above medication therapy, SLIT (Sublingual Immunotherapy for the inhalant allergens) was started as indicated in the plan under immunotherapy.

The RAST for foods came back two weeks later and was mildly positive for egg white, casein, wheat, garlic, and strongly positive for candida by IgG testing. It was recommended that Tina follow the EFL (Estrogen-Free Lifestyle) strictly.

CBC with differential showed low MCV, high RDW, but hemoglobin was within normal limits. To understand this lab, her ferritin result was checked and it was very low (7) indicating iron deficiency (low MCV, high RDW, and low ferritin, all point to iron deficiency.) The comprehensive metabolic panel showed high fasting blood glucose, low alkaline phosphatase, high fasting insulin; and leptin level was high. This is consistent with insulin and leptin resistance. The low alkaline phosphatase is consistent with hypothyroidism, and magnesium, and zinc deficiency (These are more plausible in this case.) The LPP test showed high LDL, high homocysteine (she was started on Mineral 650 with copper and iron to boost her minerals, iron stores, and thyroid function. She was also started on additional B-complex vitamins to reduce her homocysteine level); but the LDL particles were mostly large particles. Since her LDL particles were large particles, and given her aches and pains, she was advised to stop her statin drug and to start taking CoQ10 250 mg daily in addition to EFA Essentials. The hsCRP was high (8), and she was to continue on her EFA Essentials, 4 grams daily. Vitamin D level was very low (18). She was started on vitamin D3 5,000 IU daily.

Gluten sensitivity/celiac disease test: antigliadin IgA, IgG, and tissue transglutaminase IgA were normal.
The GI Effects stool test was negative for parasites and other bacteria overgrowth and digestion seemed effective. The test revealed normal E. coli, but low Bifidobacterium and Lactobacillus. Her probiotic was changed to Probiotic-50B and she was to continue on the prebiotic FOS to boost both her Bifidobacterium and Lactobacillus colonies.
Thyroid function test showed (TSH=3.5, free T4=0.7, free T3=2.2, reverse T3 = 27, thyroid peroxidase antibody (TPO) =1213, thyroglobulin antibody=889). These results show that Tina has Hashimoto's Thyroiditis with suboptimal thyroid function. She was started on Armour Thyroid 60 mg daily x 8 weeks and then will be reassessed (depending on the patient, you may start Armour thyroid as low as 15

mg daily and progressively increase to an optimal dose). Tina's high thyroid antibodies are consistent with estrogen sensitivity. I, therefore, explained again the importance of strictly following the EFL (Estrogen-Free Lifestyle).

Hormone testing: Progesterone was low (<0.27), 17-OH pregnenolone was low (0.2), SHBG was high (135), DHEA was low (30), testosterone was low (0.12), free androgen index was low (0.96), FSH was normal (4), LH was high (16) and LH/FSH ratio (4 is consistent with PCOS) and, in this case, her androgens were low because they are converting to estrogens faster. Estradiol was low normal, estrone (E1s, E1) were high, Estriol was normal, prolactin level was high (27), and consistent with her high estrogenic state. Estrogen metabolites (2-hydroxyestrone was very low (72), and 16-alpha-hydroxyestrone was very high (779), and the 2/16 ratio was NR=non-readable).

Based on this hormone profile, Tina was started on the following:

1. Progesterone cream—and an aromatase inhibitor was added to the cream to prevent her progesterone from converting to estrogen.
2. Progesterone sublingual was prescribed (to be titrated up for mood swings).
3. DHEA + 7-Keto-DHEA recommended to boost her DHEA so as to promote an increase in testosterone and male androgen index, and to help lose weight.
4. Zinc was added to this regimen as additional aromatase inhibitor to prevent the testosterone from converting to estrogens.
5. Since DHEA was very low and Tina has ventral obesity, it is clear that she has high cortisol. She was started on Cortisol Calm to help alleviate her cortisol load and, hence, boost her DHEA level (her pregnenolone is converting to cortisol by a mechanism known as pregnenolone steal and that is the reason for the low DHEA).
6. Since her progesterone, DHEA and pregnenolone are low, she was started on pregnenolone to boost both the progesterone and DHEA.
7. She was started on DIM Detox and was told to eat plenty of cruciferous vegetables to help boost her 2-hydroxyestrone level.
8. Again, she was advised to strictly adhere to the EFL while she is on this BHRT (Bioidentical Hormone Therapy).

Tina was scheduled to follow-up in eight weeks for repeat thyroid evaluation and in 4 months for hormone evaluation; she will have a hormone test repeated at the end of her third month of therapy. She was told to call any time during this therapy with any questions or concerns.

Eight Weeks Follow-Up

Tina reported losing 15 pounds during these 8 weeks by strictly following the EFL and taking her thyroid supplementation and other nutritional supplementation.

Repeat thyroid function test showed (TSH=1.5, free T4=1.2, free T3=3.5, reverse T3 = 15, thyroid peroxidase antibody (TPO) =712, thyroglobulin antibody=586). These results show that the Hashimoto's Thyroiditis and suboptimal thyroid function are

improving. She will continue on Armour Thyroid 60 mg daily for 6 months and then will be reassessed. She will continue to strictly follow the EFL (Estrogen-Free Lifestyle).

Four Months Follow-Up

Tina returned to the clinic in 4 months, after getting a repeat hormone test. She followed all the recommendations as prescribed and had no problems with her BHRT. She has lost 35 pounds and was happy about her weight loss and overall progress.

Repeat hormone testing: progesterone was low normal (4.5), SHBG has decreased (83), DHEA has increased (215), testosterone has also increased (0.35), free androgen index has increased (2.6), FSH was normal (3), LH was (8.5) and estradiol was within the normal range. Estrone (E1s, E1) were within the normal range, Estriol was normal, and prolactin has decreased (15).
Estrogen metabolites (2-hydroxyestrone was low normal 235, 16 alpha-hydroxyestrone was still high 587, and the 2/16 ratio was low normal).

Since this repeat hormone test results were encouraging, Tina was told to continue on her current BHRT protocol for six months. At the end of the 6 months, her hormone test and thyroid function test will be repeated and both her thyroid supplementation and BHRT will be adjusted at that time, if her laboratory results indicate a change.

Laboratory normal ranges

TSH	(0.45-4.50 uIU/ml)
Free T4	(0.8-1.8 ng/ml)
Free T3	(2.3-4.2 pg/ml)
Reverse T3	(8-25 ng/dL)
TPO	<33.4
TG antibody	<244
Progesterone	(0.95-21 ng/ml)
17-OH Pregnenolone	(1-3 ng/ml)
SHBG	(18-144 nmo/L)
Estradiol	(27-246 pg/ml)
Estrone (E1s)	(0.75-4.28 ng/ml)
Estrone (E1)	(28-163 pg/ml)
Estriol	≤80
DHEA	(35-430 mcg/dL)
Free Testosterone	(0.10-0.75 ng/ml)
Free Androgen Index	(0.43-8.48)
2-hydroxyestrone	(112-656 pg/ml)
16-alpha-hydroxyestrone	(213-680 ng/ml)
2: 16 Ratio	(0.40-1.40)
FSH	(1.2-9.0 miU/ml)
LH	(≤14.7 miU/ml

LH/FSH	< 3
Prolactin	(1.9-25ng/ml)
hsCRP	< 3
5-HydroxyVitamin D	(30-100 ng/ml)
Ferritin	(13-150 ng/ml)
Alkaline Phosphatase	(35-104 U/L

Appendix B shows nutritional supplements recommendations for various conditions. Many patients have obtained satisfactory results with EFL and these supplements. These recommendations are not intended to replace your Integrative Immunity provider or Functional Medicine provider's evaluation and recommendations. You should always work with your providers for proper evaluation and guidance.

I wish you well in health and happiness.

APPENDIX B: DR. TANO'S RECOMMENDATIONS

Your Health Is Your Wealth! Protect It by Following These Suggestions:

1. **Avoid these grains**
    - **Corn (GMO)**
    - **Barley**
    - **Rye**
    - **Wheat**
    - **Spelt**
    - **Kamut**
    - **Millet**

You Can Eat Other Grains. Try these: oats, rice, quinoa, amaranth (beware amaranth has a high glycemic index).

2. **Avoid potatoes (sweet potatoes are fine)**
3. **Avoid dairy (drink almond, cashew, coconut, or rice milk)**
4. **Avoid refined sugars (half of all refined sugars are from sugar beets, number 1 GMO)**
5. **Avoid the dirty dozen foods unless organic (see list) and processed foods in general - eat whole foods, organic as much as possible**
6. **Avoid xenoestrogens and phytoestrogens (see list in table 17)**

If you are allergic to any foods, do not eat them!!!

In addition to strictly following the Estrogen-Free Lifestyle, take these nutritional supplements for overall health

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **CoQ10 or Ubiquinol**
- **Magnesium Citrate, if constipated, and Glycinate or Cognimag otherwise**

Nutritional Supplements for Irritable Bowel Syndrome

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid for constipation**
- **Magnesium Citrate for constipation**
- **Probiotics (50 B) + the prebiotic Fructooligosaccharides (FOS)**
- **Digestive Enzymes Ultra (or Digestive Enzymes Ultra with Betaine HCL, if you have GERD)**

Balance the hormones

Nutritional Supplements for Headaches and Migraine Headaches:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (or Glycinate)**
- **Glutathione**
- **RevitalAge Ultra**
- **COQ10**

Balance the hormones

Nutritional Supplements for Depression and Anxiety:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate**
- **Probiotics (50 B) + FOS**
- **Digestive Enzymes Ultra**
- **Emotional Wellness**
- **Melatonin, 3 mg**

Balance the hormones

Nutritional Supplements for Fibromyalgia:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate or Glycinate or Threonate (CogniMag by Pure Encapsulations)**
- **Emotional Wellness**
- **Melatonin, 3 mg**
- **RevitalAge Ultra**
- **Mental Sharp**
- **COQ10**
- **PS (phosphatidylserine) Plus**

Balance the hormones

Nutritional Supplements for Chronic Fatigue:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate or Threonate)**
- **Emotional Wellness**
- **Melatonin, 3 mg**
- **RevitalAge Ultra**
- **Mental Sharp (or PS Plus)**
- **COQ10**
- **Glutathione**
- **Asparagine**
- **Glutamine**

Balance the hormones, including thyroid hormones

Nutritional Supplements for Allergies and Asthma:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate)**
- **Emotional Wellness (many asthmatics have anxiety and L-Theanine and 5-HTP may help)**
- **Melatonin**
- **RevitalAge Ultra**
- **COQ10**
- **Choline**
- **Aller-Essentials**
- **D-Hist**

Balance the hormones, including thyroid hormones

Nutritional Supplements for Metabolic Syndrome:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate or Threonate)**
- **Probiotics (50 B) + FOS**
- **Digestive Enzymes Ultra with Betaine HCL**
- **COQ10**
- **Chromium**
- **Cinnamon**
- **Benfomax (benfothiamine)**
- **RevitalAge Ultra (or RevitalAge Nerve, if you have a neuropathy)**

Balance the hormones, including thyroid hormones

Nutritional Supplements for Hypertension:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate or Threonate)**
- **Probiotics (50 B) + FOS**
- **Digestive Enzymes Ultra with Betaine HCL**
- **COQ10**
- **Chromium**
- **Nitric Oxide Ultra**
- **RevitalAge Ultra**

Balance the hormones, including thyroid hormones

Nutritional Supplements for Obesity:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate or Threonate)**
- **Probiotics (50 B) + FOS**
- **Digestive Enzymes Ultra with Betaine HCL**
- **COQ10**
- **Chromium**
- **Garcinia + Caralluma Fimbriata + CLA +Bios Life Slim**
- **Benfomax**
- **RevitalAge Ultra**
- **Growth Hormone Support (nutritional supplement)**
- **Thyroid Support Complex**

Balance the hormones, including thyroid hormones

Nutritional Supplements for Atopic Dermatitis in Adults:

- **Nutrient 950 with NAC**
- **EFA Essentials**
- **Vitamin D3**
- **Buffered Ascorbic Acid**
- **Magnesium Citrate (Glycinate)**
- **Probiotics (50 B) + FOS**
- **Digestive Enzymes Ultra**
- **COQ10**
- **Silymarin (milk thistle extract)**
- **RevitalAge Ultra**

Balance the hormones

Nutritional Supplements for Atopic Dermatitis in Children:

- **Junior Nutrients (or PurePals for the very young)**
- **Liquid EPA/DHA**
- **Vitamin D3 (35 IU/lb)**
- **Probiotics 123 by Pure Encapsulations (for children)**