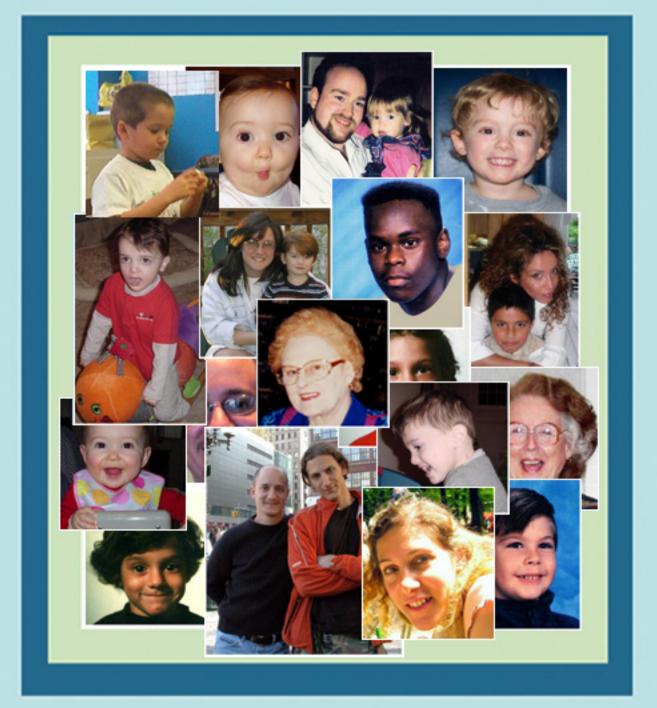
Feingold Bluebook





Behavior, Learning & Health: The Dietary Connection The Feingold[®] Association of the United States, Inc., founded in 1976, is a non-profit organization whose purposes are to generate public awareness of the role of foods and synthetic additives in behavior, learning, and health problems, and to support its members in the implementation of the Feingold Program.

Neither a diagnosis nor a prescription is required to use the Feingold Program as a healthy diet choice for children and adults.

This booklet is for educational purposes only and is not intended to replace competent medical diagnosis and care. The Feingold Association does not endorse, approve or assume responsibility for any product, brand, method or treatment discussed.

a special thanks to our Feingold staff, professional advisors and volunteers whose contributions have made the printing of this book possible

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The Feingold Bluebook

Behavior, Learning, and Health: The Dietary Connection

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Symptoms That May Be Helped by the Feingold Program

A person who may be helped by the Feingold Program displays more of the following symptoms more frequently and to more of an extreme than the average person.

Behavior

MARKED HYPERACTIVITY

Constant motion Running instead of walking Inability to sit still Inappropriate wiggling of legs / hands

IMPULSIVE ACTIONS

Disruptive behavior / disturbs others Unresponsiveness to discipline Poor self-control Destructiveness: throws, breaks things Little or no recognition of danger to self Unpredictable behavior Inappropriate noises Excessive and/or loud talking Interrupts often Abusive behavior to people or pets

COMPULSIVE ACTIONS

Perseveration (repeating an activity) Touching things / people Aggression Workaholic habits Chewing on clothing, other objects Scratching, biting, picking at skin

EMOTIONAL CONCERNS

Low frustration tolerance Sensitivity to touch, pain, sound, lights Depression Frequent crying Demands immediate attention Irritability Panics easily Nervousness Low self-esteem Mood swings Suicidal thoughts

Learning / Developmental

SHORT ATTENTION SPAN

Impatience Distraction Failure to complete projects Inability to listen to whole story Inability to follow directions

NEURO-MUSCULAR INVOLVEMENT

Accident prone Poor muscle coordination Poor eye-hand coordination Dysgraphia / difficulty writing Dyslexia / reading problems Speech difficulties / delays Difficulty with playground activities, sports Eye-muscle disorder (nystagmus, strabismus) Tics Seizures

COGNITIVE & PERCEPTUAL DISTURBANCES

Auditory processing problems Visual processing problems Comprehension & short term memory difficulty Disturbed spatial orientation (up-down; left-right) Reasoning difficulty (math problems or word meaning)

Health / Physical Complaints

POOR SLEEP HABITS

Resistance to bedtime Restless/erratic sleep Inability to fall asleep Nightmares, bad dreams

FREQUENT PHYSICAL COMPLAINTS

Ear infections Bed wetting Day wetting Hives or rashes Asthma Stomachaches Headaches Congestion Leg aches Constipation Diarrhea

The Feingold Program

The Feingold Program addresses additive and salicylate sensitivity. The Feingold Association of the United States (FAUS) provides its members with comprehensive information on brand name foods and nonfood products that are free of the indicated additives.

Stage One - Remove the Following:

• Artificial (synthetic) color

Synthetic food dyes are listed in a number of ways – by name, by FD&C number, by E-number, or not at all. In the US and Canada, the *Feingold Foodlist* will help you be sure you are avoiding them.

• Artificial (synthetic) flavor

The thousands of artificial flavoring chemicals are listed as "flavoring," or "artificial flavoring," except for artificial vanilla (vanillin). Fragrances may be called "fragrance" or "perfume." Most are petrochemicals, but products containing natural fragrance are listed in our *Fragrance Guide*.

• Three petrochemical preservatives

BHA	(Butylated Hydroxyanisole)
BHT	(Butylated Hydroxytoluene)
TBHQ	(Tertiary Butylhydroquinone)

• Artificial (synthetic) sweeteners

Aspartame (Equal, NutraSweet), sucralose (Splenda), and similar brands are not allowed. Products containing saccharin, acesulfame-K, and similar chemicals are also not included in the *Foodlists* although some members do tolerate them.

Real sugars and sugar syrups are allowed.

Alcohol sugars (names end in "-ol") are allowed. Stevia is a natural no-calorie sweetener from an herb. It is allowed, but care should be taken not to choose a brand with questionable additives.

• Salicylates

These are chemical compounds similar to aspirin which occur naturally in some foods, and are added to some medicines and personal care products. They may cross-sensitize to food dyes and are eliminated in the beginning of the Program.

Stage Two – Test the Salicylates:

After observing a favorable response to Stage One, salicylates may be reintroduced and tested for tolerance one at a time. While some people find they need to remain on Stage One, others are able to tolerate some salicylate-containing items occasionally, and still others can eat them freely. The artificial colors, flavors, preservatives, and sweeteners listed above are not reintroduced.

Additional Additives:

Some chemical additives are not routinely eliminated, but products containing them are marked in the *Foodlist* & *Shopping Guide* books so that they can be avoided at the start of the diet or later if necessary. They are Calcium Propionate, Corn Sweetener, Sulfites, Sodium Benzoate, Monosodium Glutamate, Nitrites/Nitrates, and Natural Smoke Flavor.

What the Feingold Association Provides

Program Materials:

- The Handbook indispensable for beginners
 - Explanation of diet and helpful forms to use, including recipes and menu planning
 - Advice for handling various situations, including school, parties, hospitalization, eating out
 - Special needs introduction to dealing with SAS, Gluten, Casein, Benzoates, Sulfur, and more
- The Foodlist specific for your US region or for Canada
- Pure Facts Newsletter 10 electronic issues a year with product updates, articles, and more
- Fast Food Guide what's okay in fast food restaurants
- Mail Order Guide for specialty items and hard-to-find products

Member Services (on website):

- Getting Started information for new members available via a password in their welcome email
- Member Services a resource including form downloads; some of it requires the member password
- Message Support Boards forum for member support, recipe sharing, & helpful information lists
- Free help by phone or email
- **Product Alerts** sent by e-mail as needed

Also Available:

- **Book:** *Why Can't My Child Behave? (fourth ed., 2006)* by Jane Hersey; editor of *Pure Facts.* Based on the experience of thousands of families for several decades, this book provides practical answers.
- Information Packets for teachers and physicians
- **Fragrance Guide** listing of products with natural fragrances.
- Other Books, Audio CDs, a free eNewsletter, an Amazon bookstore ...
- Family Pages a free monthly series of on-line pages of recipes, crafts and tips

How to Become a Member & Get Program Materials:

- WEBSITE: See the home page <u>www.feingold.org</u> and follow directions
- **PHONE:** 1-631-369-9340 (Eastern Time, US, mornings)
- eMAIL: Fausmem@yahoo.com
- **Outside US or Canada:** Register at the International Members Section (under **JOIN** on Home Page). The password you get will let you see all the information as we are able to provide for people outside the US or Canada (free).
- **Caveman Diet:** For those adventurous enough to try the Feingold version of the Paleolithic Diet (free) see website address on inside front cover of this book.

Frequently Asked Questions

1. Who was Dr. Feingold?

Ben F. Feingold, M.D. was both a pediatrician and allergist. He was Chief of Pediatrics at Cedars of Lebanon Hospital in Los Angeles, CA, until 1951, when he became Chief of Allergy at Kaiser-Permanente Medical Center in San Francisco. He continued his work with children and adults with hyperactivity and allergy long after his retirement, until his death at the age of 82, in 1982.

2. What is the Feingold Association?

Founded in 1976, the Feingold Association of the United States (FAUS) is a 501(C)3 non-profit organization made up of parents, professionals and volunteers dedicated to helping children and adults implement the Feingold Program.

3. What is the Feingold Program?

This dietary program was developed at the Kaiser Permanente Medical Center in San Francisco. Called the "K-P Diet," it was an outgrowth of the earlier diet for urticaria (hives) developed by Dr. Stephen Lockey of the Mayo Clinic. The media renamed it the "Feingold Diet." The diet eliminates artificial food colors, artificial flavors, three preservatives, and certain salicylates in food, toiletries, etc.

4. How can this Program help me?

It can help you determine if certain foods or food additives contribute to symptoms. If they do, then the diet itself, adjusted to your individual needs, is also the treatment. It can be part of a multi-modal treatment protocol and is compatible with any other form of treatment, but starting with diet first may save you money and help you determine if other treatment is still needed.

5. How soon can I expect to see results?

It varies with the individual. If the Program is followed carefully, you should see results within one to six weeks. As a rule, young children respond the most quickly, sometimes within a few days. If ADHD medication is being used, a response may take longer. If other allergies or problems are involved, they must also be addressed. The *Foodlist* can also be used with a gluten/casein-free diet or with any allergy diet.

6. Is it hard?

Changing your eating behavior is never easy, but it soon becomes a way of life. Many well-known products are free of problem additives, and you will be able to enjoy most of your favorite foods just by changing some of the brands. Avoiding salicylates is a little harder, but it is an important part of the Program. It is also the only way available to find out whether salicylate-sensitivity is a problem for you or your child.

7. How do I know which foods are OK?

As a member of FAUS, you will receive a book listing the thousands of acceptable brand-name products available in your region of the country.

FAUS began doing this work in 1976, producing a one-page *Foodlist & Shopping Guide*. Today, this unique book is over 300 pages long (*the Canadian Foodlist book is almost 150 pages*) and is organized by category. You can easily take it to the supermarket. It is reprinted frequently and updated through the *Pure Facts* newsletter and email alerts. For an item to be added to this list, the manufacturer must fill out and sign a detailed inquiry form verifying that the product, wrapper, etc. are free of all the undesired additives.

8. Why can't I just read labels?

Regulations governing the labeling requirements of both food and non-food items are inconsistent.

Labels frequently have information that is incomplete or misleading.

Most people think that manufacturers list all the ingredients in a product, but it is not true. They do not have to list additives other companies



put into ingredients they buy, and some products are not required to list ingredients at all. Ingredients such as "flavoring" do not indicate whether they are natural or artificial, or whether they may contain salicylate.

9. Will I have to cook from 'scratch?'

Not unless you want to. The *Foodlist & Shopping Guide* includes a wide selection of prepared foods available in your supermarket. Our product inquiry is ongoing, so new products become available continuously. Moreover, due to consumer demand, manufacturers are responding by providing more products that meet our ingredient guidelines. At the supermarket, you simply choose products from the thousands of acceptable items in your *Foodlist*, including snacks, cakes, ice cream, candy, and prepared foods. Once you are home from the supermarket, you prepare food as you normally would.

10. But what about sugar?

Many people think that sugar causes behavior problems. More likely, the additives in sugary foods are to blame. However, some people are sensitive to corn syrup (or the chemical residues in it), some are sensitive to beet sugar, and a few are unable to tolerate cane sugar.

While items containing corn syrup are marked with a (CS) in the *Foodlist*, sugar is <u>not</u> routinely eliminated on the Feingold Program.

11. Are all additives bad?

There are well over 12,000 food additives in our food supply today, nearly 2/3 of them flavorings, but few have been tested for their effect on the nervous system or the immune system. Furthermore, some additives tested and found to have negative effects are still in use.¹

Meanwhile, scientists working for the food industry² have convinced the FDA to use the De Minimis principle ("a little bit can't hurt") so that new flavoring "chemicals of unknown toxicity" do not even need to be tested on animals before being accepted for use.

As for fragrances – the FDA does not supervise or mandate research on them or control their labeling; they say it is because they do not have any budget for that.

The additives we eliminate appear to be the worst offenders for the majority of children and adults with ADHD and related problems. If improvement is erratic or less than desired, our materials help you consider other additives, such as corn syrup, MSG, sodium benzoate, sulfites, etc.

12. Will I have to take my child off ADHD medication?

You can begin the diet while your child is still on ADHD medication, though it may take longer for the child to respond. Members frequently report that after using diet and medication together for a while, their doctor is able to reduce or discontinue the medication. Other members report that, for their child, medication appears to be more effective when used with diet. For best results, we recommend making the effort to acquire all needed medication in a color-free form. If needed, a compounding pharmacist may be helpful.

When removing a child from behavior modifying medication, the child's symptoms may seem worse. This is a medication "rebound" effect and can last several days to several weeks.

Do not stop any medication without medical guidance.

13. Can other things cause ADHD?

Yes! Many things can trigger such symptoms, including:

- Food allergies
- Medication side effects
- Too little sleep
- Heavy metal exposure acute or chronic
- Buildup of heavy metals if unable to detoxify
- Stress, loss of loved one or pet
- Allergy to pollen, mold, etc.
- Vitamin deficiency or pathway defect
- Zinc or iron deficiency
- Fluoride sensitivity
- Celiac disease
- Manganese buildup (i.e., from soy formula) if unable to detoxify it efficiently
- Being among the youngest in a classroom
- Sensory integration deficits
- Lack of "good fats" such as omega-3
- Exposure to fragrances
- Exposure to gas or oil heat fumes
- Hearing distortion (auditory processing)
- Vision problems
- Parasites such as Toxocara
- Yeast overgrowth in GI tract
- Thyroid dysfunction
- Actual injury to brain from accident or illness
- Poor prenatal nutrition
- Premature birth
- Prenatal exposure to harmful chemicals

^{1.} Aoshima 1997, Bamforth 1993

^{2.} Kroes 2000, 2002, 2004, 2005

Artificial Colors

The synthetic colorings were originally manufactured from coal tar, but today they are made from petroleum.

The seven artificial colors currently certified "FD&C" are permitted by the Food & Drug Administration (FDA) to be added to foods, drugs and cosmetics.

The many colors certified "D&C" may be used only in drugs and cosmetics.

The manufacturer must submit a sample of each batch of dye to the FDA for certification. The FDA is paid a user fee¹ for each pound certified. In 2010, almost **22** *million pounds* of color additives were certified by FDA inspectors.²

FD&C colorings have "Generally Recognized as Safe" (GRAS) status despite studies showing neurological effects,³ DNA damage,⁴ and elevated cholesterol.⁵

Petroleum ... Lead ... Mercury ... Arsenic ... yuck. Knowing these chemicals are in the food dyes is reason enough to avoid them – whether or not you are sensitive to the dyes themselves.

But aren't the FD&C dyes certified to be safe?

No.

They are certified to contain no more than the amount of lead, mercury, arsenic, benzidine, and other contaminants that the Food & Drug Administration (FDA) considers acceptable. They are certified to contain a minimum percent of actual color as specified in the Code of Federal Regulations.⁶

Consider benzidine. Yellow 5 & Yellow 6 are each allowed to have 1 ppb (parts per billion) of benzidine. That is a really tiny amount; benzidine is known to cause cancer,⁷ but it apparently can't be easily removed from the dye, so the FDA decided to allow it at that amount. But how much is *really* in there?



Drs. Peiperl and Prival wanted to see how much benzidine is actually in the Yellow 5 and 6 you buy in the supermarket, so they bought bottles and tested them. They found that half of the 53 Yellow 5 samples they tested contained 7 to 83 ppb of benzidine, and half the 67 samples of Yellow 6 had more than 10 ppb benzidine, with some as high as 104 ppb, and one at 941 ppb. In Canada, Dr. Lancaster did a separate study finding levels of benzidine ranging from less than 5 to 270 ppb.⁸

Consider lead. The FDA, NIH,⁹ and all doctors tell us to avoid lead because it damages the brain of both children and adults. Yet it is an interesting bit of trivia that while the FD&C food colorings are allowed to have no more than 10 ppm (parts per million) of lead, many of the D&C colors permitted in medications and given multiple times a day to sick people are allowed to have <u>twice</u> that amount.⁶

^{1.} Electronic Code of Federal Regulations, Title 21, Part 80 - http://tinyurl.com/FDA-certification

^{2.} FDA Color Certification Reports: http://tinyurl.com/FDA-howmuch

^{3.} Tanaka 1993, 1996, 2001, 2005; Vorhees 1983

^{4.} Rosenkranz 1990; Sweeney 1994; Tsuda 2001; Sasaki 2002

^{5.} Aboel-Zahab 1997

^{6.} Electronic Code of Federal Regulations, Title 21, Part 74 - http://tinyurl.com/chemicals-in-dye

^{7.} Toxicological Profile for Benzidine – <u>http://tinyurl.com/NIH-benzidine</u>

^{8.} Prival 1993; Peiperl 1995; Lancaster 1999

^{9.} NIH: National Institutes of Health, one of the world's foremost research centers, is part of the US Department of Health and Human Services.

Artificial Flavors

Used as low-cost substitutes for natural flavorings, these chemicals are not usually listed individually. You will see them listed as "flavoring" or "artificial flavoring." Although products containing artificial flavorings are required to say so, notice how skinny the letters ae on the package and how easy it is to overlook them.

One flavoring that may be listed separately is vanillin *(imitation vanilla)*, widely used in chocolate as well as in vanilla-flavored items. Some people who believe they are allergic to chocolate may actually be reacting to this artificial flavoring.

One source of imitation vanilla flavoring is the waste product of paper mills; another is petroleum. Therefore, while vanillin is technically identical to one of the chemicals in pure vanilla flavoring, the manufacturing methods result in high levels of sulfites and other contaminants. Moreover, the synthetic version is not bound to other parts of the vanilla bean source in the same way it is found in nature.

A single artificial flavoring can be a combination of hundreds of individual chemicals, many of which are derived from petroleum.

Formula for Raspberry Flavoring:

Vanillin, Ethylvanillin, Alphaionone, Maltol, 1-(p-hydroxy-phenyl)-3-Butanone, Dimethyl Sulphide, 2,5-Dimethyl-N-(2-pyrazinyl) Pyrrole.

Where are the raspberries?



The FDA does

not monitor artificial flavorings nor require that they be tested. Rather, a concept called the "threshold of toxicological concern" has been implemented to set acceptable daily intake levels for chemicals <u>of</u> <u>unknown toxicity</u>, apparently on the theory that a little bit can't hurt. This is also called the "de minimis principle" and was introduced to "save the time, cost, animal use and expertise" usually needed for extensive toxicity testing and safety evaluations.¹

Even when testing is done, however, it may be ignored. Vanillin, for example, continues to be listed as GRAS (*Generally Recognized as Safe*) despite its ability to inhibit the liver enzyme dopamine sulphotransferase by 50%.² Other flavorings affect RNA, thyroid, and various enzymes.³

Most flavorings have simply <u>never</u> been studied for "side effects" relating to human health, and none has <u>ever</u> been studied for neurotoxicity.

Of course, out of the thousands of artificial flavorings commonly used, some are surely perfectly safe; however, nobody knows which ones they are – and even if we knew, we still would not know which ones were in what food products. Therefore, the Feingold Program must simply eliminate all of them.

^{1.} Kroes 2000, 2002, 2005

^{2.} Bamforth 1993

^{3.} El-Saadany 1991

Preservatives

BHA: Butylated Hydroxyanisole BHT: Butylated Hydroxytoluene TBHQ: Tertiary Butylhydroquinone

Preservatives are used primarily to prevent fats from becoming rancid, allowing foods to have a longer "shelf-life." Most preservatives are not believed to be a health hazard, but the above three petroleum-based preservatives have been found to trigger behavior and health problems.

Studies on these chemicals are disturbing. As early as 1974, a study by Stokes & Scudder¹ reported that when pregnant mice were fed BHA and BHT, it affected the brain chemistry of their offspring, reducing their cholinesterase and serotonin to half the normal levels. They reported, "The affected mice weighed less, slept less and fought more than normal controls."

Since BHA, BHT, and TBHQ are included in so many products containing other additives as well, it would be prudent to study their interactions with each other.

One of the few such studies found that BHA can "facilitate the activation of BHT in the lung" and increase its toxicity.² Yet it is common to find both of them in the same meal.

These preservatives continue to enjoy GRAS (Generally Recognized As Safe) status despite evidence that they are **toxic** to various cells and organs,³ they are **tumor** promoters,⁴ they **weaken the immune system**,⁵ they impact the **nervous system and behavior**,⁶ and they have a negative effect on **sperm and/or egg production**,⁷ **reproduction and development**.⁸

Sasaki (2002) says that many of the 39 common additives he studied, including BHT and BHA, produced DNA damage at low doses close to the ADI, *(the allowable daily intake)*.⁹

Most additives have *never* been studied in combination with each other -- or with environmental toxins, medications, or vaccines.

adult fish.11

In spite of warning us that fast food workers are exposed to high levels of BHA from the frying oil, this year's Report on Carcinogens concludes, again, that the data on humans is "inadequate." (*Note: To my knowledge, no studies have ever yet been done on humans.*)

BHA has been listed as a cancer-causing chemical since the 6^{th} Annual Report on Carcinogens in 1991.

It has been listed as a carcinogen in every Report since

then. In the year 2011, the 12th Report on Carcinogens

stated - again - that BHA is "reasonably anticipated

to be a human carcinogen based on sufficient

evidence of carcinogeni-city from studies in

experimental animals."¹⁰

When put in their diet,

BHA caused papillomas

and carcinomas (cancers)

in the stomach of rats,

mice, and hamsters. When

fed to fish larvae, BHA caused liver cancer in the

tumors)

(non-cancerous

These preservatives are not always listed on product labels. If the product contains oil or other secondary ingredients, preservatives in those ingredients may not be listed. They can be avoided, however, by using the Feingold Association's *Foodlist & Shopping Guide*.

The acceptable daily intake (ADI) is known to be exceeded in Italy, Lebanon, and the Netherlands^{12,13,14} in spite of knowledge that other preservatives such as pomegranate juice and vitamin E are safer.^{15,16}

- 7. Takami 1999
- 8. Meyer 1980; Vorhees 1981; McFarlane 1997
- 9. Sasaki 2002
- 10. 12th Report on Carcinogens, National Toxicology Program <u>http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf</u>

- 12. LeClercq 2000
- 13. Soubra 2007
- 14. Verhagen 1990
- 15. Naveena 2008
- 16. Kahl 1993

^{1.} Stokes 1974

^{2.} Thompson 1988, 1989

^{3.} Zoccarato 1987; Thompson 1988; Kahl 1983, 1993; Siman 1996; Gudz 1997; Stolze 1999; Safer 1999; Yu 2000; Groten 2000

^{4.} Kahl 1984; Parke 1992; Kahl 1993; Bauer 2001; Sasaki 2002

^{5.} Tryphonas 1999

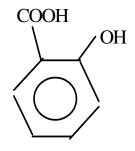
^{6.} Stokes 1974; Tanaka 1993

^{11.} Park 1990

Salicylates

Salicylates [Sa-Lis'-uh-Lates] comprise a group of compounds made by plants as protection against insects and disease. Salicylates are chemically related to aspirin (acetylsalicylic acid). There are several types, such as sodium salicylate, methyl salicylate, ethyl salicylate, aluminum acetyl salicylate, ammonium salicylate, etc.

The salicylates to be eliminated can be found in some fruits and a few vegetables, and are used by manufacturers for flavor, for aroma, or as preservatives in some foods, medications, cosmetics, and other non-food items. While salicylate-containing medicines such as aspirin can offer benefits, and foods containing salicylates can be very nourishing, they are not well-tolerated by everyone. Although salicylate-intolerance is not generally a true





allergy, anyone actually allergic to aspirin may also feel better when eliminating salicylate-containing foods and products.

Many people believe that by measuring the salicylate content of various foods, they can assume that those with the highest levels are the ones that will cause problems. Unfortunately, it is just not that simple.

Here's why:

- There are various kinds of salicylates; we don't know which ones are most likely to cause adverse reactions and even if we did, we don't know which ones are in which fruits and vegetables.
- The amount of salicylate can vary from one variety of a fruit to another, and even the levels in a particular plant can change. For example, organic fruits in an orchard that has been attacked by pests will make more salicylate than other similar fruits.
- Different parts of a plant might have different levels of salicylate. The amounts can vary between the pulp, seeds, and peel of a fruit or vegetable.
- Foods grown in one region might not be the same as foods grown in another region.

- Sensitivity can vary depending on whether the fruit or vegetable is raw or cooked. For example, fresh pineapple may cause a problem for the same person who tolerates canned pineapple or pineapple juice. (Canned pineapple is acceptable on Stage One of the Feingold Diet, but fresh pineapple should not be used at the very beginning.)
- We don't even know that it is only the salicylate in a food that is to blame; there could be other naturally-occurring chemicals that play a part.
- Typically, a salicylate-sensitive person has problems with only some not all salicylates.
- Salicylate sensitivity can change; frequently, a person who avoids them for a year or so can later tolerate moderate amounts of them.

In his allergy clinic at the Kaiser Permanente Medical Center in San Francisco, Dr. Feingold searched for help to identify which salicylates were likely to bother the patients. The only list of foods with salicylates suspected of inducing aspirin-induced asthma was decades old, but he decided to use it as a starting point. Actually, it turned out to be useful, and he did not have to make many changes.

Stage One of the Feingold Program eliminates those salicylates identified as the most troublesome. After a favorable response, salicylate-containing products may be carefully reintroduced, one at a time, to determine if there is a problem with any or all of them.

Feingold Association members report wide variation in salicylate sensitivity, as well as a cumulative effect and a more dramatic reaction when combined with synthetic additives. Some can eat salicylates freely, while others can occasionally tolerate small amounts of a favorite salicylate food if they are otherwise stable on Stage One of the Program.

Environmental Chemicals

Artificially colored, flavored, scented, or preserved non-food items can also cause a reaction when inhaled or absorbed through the skin. The Feingold Program will help you find household and personal care items less likely to cause symptoms.

Although the Program does not address the issue of pesticides directly, some members report symptom improvement when pesticides in food and the environment are avoided. Recent research has found that children with higher levels of organophosphate pesticide chemicals in their urine were more likely to have ADHD symptoms.¹ Worse, prenatal exposure to polychlorinated biphenyl (PCB) chemicals found in pesticides such as DDT and persisting in the environment even after such use was terminated, also cause ADHD-like symptoms in the children.² Nevertheless, food is a primary source of the pesticide chemicals found in children's bodies, and using organic produce can reduce current exposure and lower detectible levels dramatically and immediately.³

Eating organic is not a required part of the Feingold Diet and may not be necessary for symptom improvement - but the more we learn, the more we recommend using organic foods as much as possible.

Pesticides

The National Academy of Science reports "neurotoxic and behavioral effects may result from low-level chronic exposure to some organophosphate and



carbamate pesticides." As long ago as April, 1991, the US government report, Neuroto*xicity: Identifying and* Controlling Poisons for the Nervous System, stated that everyone is at risk of being harmed by these chemicals, but the highest risk groups are fetuses, children, and the elderly.

Pesticides used outside the home are easily tracked inside and are readily inhaled and absorbed through the skin. Children are at high risk of exposure since they are more likely to crawl on the floor and play in the grass and on the school playground.

Nevertheless, a main route of chronic exposure is through the diet, and eating organic foods as often as possible makes a measurable difference.³

Perfumes

Today, fragrances are made primarily from petroleum and can be just as harmful as petroleum-based food additives. When inhaled, they can directly affect the brain, where they can trigger an immediate reaction. Fragrances applied to the skin are also absorbed systemically.

Various chemicals commonly used in perfumes, cleaning supplies – and even children's toys – have been shown to cause adverse effects in



animals, including inhibition of motor activity, respiratory tract irritation, narcotic effects when inhaled, hyperactivity, irritability, liver damage, spasms and death; and, in humans, marked eye, nose, or throat irritation, and numbness of fingers and arms.⁴ **Fragrances are not under FDA regulation and are not required to be tested for safety.** If not tested by the manufacturer, there is supposed to be a note put on the label to that effect. This requirement also is not monitored by anyone.⁵

^{1.} Bouchard 2010; Eskenazi 2007; Raby 1995

^{2.} Sagiv 2010

^{3.} Lu 2006

^{4.} Spencer 1984

^{5.} FDA information via phone calls by this author in 2003, and verified in 2004 and 2007.

A different kind of school lunch

Students in one Midwestern community are enjoying fresh, delicious food plus a big change in their learning environment.

Walk down the hallways of the Appleton, Wisconsin, Central Alternative High School, and you will see students focused on their education, interacting successfully with each other and with their teachers. Notice the calmness and purposefulness that sets these teens apart from others. You will notice that the hallways are different in another respect: They aren't lined with soft drink and junk food machines. Then, check out the cafeteria. Burgers, fries and burritos have been replaced by salads, meats prepared with old fashioned recipes, and whole grain breads. Fresh fruits and vegetables are offered, and the students drink water.

Grades are up, truancy is no longer a problem, arguments are rare, and teachers are able to spend their time teaching. What's going on in Appleton, Wisconsin?



In 1997, Natural Ovens of Manitowoc, WI, initiated a five-year project to bring healthy food into area schools. The goal was to show that fresh, nutritious food can make a real difference in the students' behavior, learning and health.

Just prior to the beginning of the program, Greg Bretthauer had been offered the job of dean of students at the school. What he saw were teens who were "rude, obnoxious, and ill-mannered." Because of problems with discipline and weapons violations, a police officer had to be on staff. Appleton was a school for troubled teens that other schools had given up on, and it was a school out of control.

The story of the Appleton project has been documented on a DVD and videotape called *Impact of Fresh, Healthy Food on Learning*

and Behavior 2004. It is also part of their *Roadmap to Healthy Foods in School*, and both are available from Natural Press, at **1-877-628-8398** or www.naturalpress.info.

Principal LuAnn Coenen is amazed at the change she has seen in her school. Each year, Wisconsin principals are required to file a report on the number of students who have dropped out, been expelled, been found using drugs, been found with weapons, or committed suicide. Since the start of the program, she reported, the number in every category has been "zero." Mary Bruyette, a teacher at the high school, reports that the students are now calm and well-behaved. "I don't have to deal with the daily discipline issues," she said, "that just isn't an issue here." Their biggest problems now are parking and tardiness. "I don't have the disruptions in class or the difficulties with student behavior that I experienced before we started the food program," she said.

Students who previously had been headed for trouble have turned their lives around, according to Dr. Thomas Scullen, Superintendent of the Appleton Area School District. He told the interviewer, "We have kids who have had a lot of problems and got

Today Greg is dean of students in an atmosphere vastly different from what he saw in 1997. through the whole last year without an expulsion. Dropouts dropped to non-existent. Kids came to school. They have learned that with

healthier foods it's going to make them a better person. It keeps them more focused and makes them happier." Dr. Scullen had expected that the healthy diet would improve behavior, but he was pleasantly surprised that it has had such an impact on academic performance.

Mary Bruyette says she can demand more, academically, from the students than she previously could. Now she can use all of her class period for instruction. The high school's counselor, Deb Larson, says, "I don't have the angry outbursts, so instead we get to deal with the real issues that are underlying and causing some of the problems in the kids' lives." Typically, while school dietitians want children to eat healthier food, they are convinced such efforts will be futile, and that if students cannot get their fast food in the cafeteria, they will buy it off campus. This does not appear to be a problem in Appleton, where the food is not only natural, it is prepared with care. Natural Ovens made sure of this by supplying their own cooks to the school.

Like children on the Feingold Program, once these teens have made the connection between food, behavior and learning, they tend to prefer to enjoy the benefits. One student said, "I really like the food. It tastes good, it's hot, it's fresh." One girl commented, "Now that I concentrate, I think it is easier to get along with people." Another student said, "If you're going for a big test, you want to eat great."

The on-campus policeman, Dan Tauber, is able to be a role model now instead of a disciplinarian. Students are interested in how he eats to keep in such good

physical shape and have noticed their athletic abilities have a lot to do with their diet.

"Returning students are now the advocates for the program. The kids encourage each other," according to Mary Bruyette. "They set the example for the new kids. It works great."

Many of the changes are being

phased in to Appleton's middle and elementary schools. Candy machines are gone and pop machines are being replaced with juice machines or water coolers. There is a district-wide commitment to healthier eating and a healthier lifestyle in general.

Even in schools where more modest changes have been made, there are some real differences. Gary Van Lankvelt, principal of the Einstein Middle School, has seen "more calmness and less bouncy activity. Students seem to be more alert and focused."

"We've got to stop using our most precious commodity – our kids – to make extra money." - LuAnn Coenen, Principal

Madison Middle School's principal, Fred Ginnochio, says the students are buying the healthier a la carte

items, and more are using the salad bar. He has found when the kids are in the halls "we have not had one incident all year that I have had to get involved in with shoving, a fight, aggressive behavior." Dr. Scullen sees an eventual switchover in all of Appleton's schools. "It can take several years to make the transition. The program will sell itself on its own merits, given the time. I think instead of looking at the food program as a "break-even" we have to take a look at what do we have to put in to make it really good for the kids."

What about increased cost?

Natural Ovens underwrote the cost for their 5 year study that will eventually impact 200 Wisconsin schools. The price to turn the problem

"One child arrested would cost the schools more." - Dr. Barbara Reed Stitt, Natural Ovens President

around was \$20,000 a year. Natural Ovens President, Dr. Barbara Reed Stitt, noted that "one child arrested would cost the schools more."

Dr. Scullen believes, "If it results in a happier kid,

improved learning, and ultimately a better community, then it's a cost we cannot avoid. It's something we must do." Says Dan Tauber, "Let's invest in the kids now, financially, with food versus invest in them later, financially, with 'how do we correct the problems we have because they are not eating healthy?""

"Nutrition for students should be part of the general operating budget," according to Mary Bruyette. "We're concerned about everything else. We're concerned about new band uniforms. We're concerned about the football team. We're concerned about text books. Why not be concerned about nutrition? That seems to me the basis in many cases for creating a positive learning environment."

LuAnn Coenen says, "I can't buy the argument that it's too costly for schools to provide good nutrition for their students. I found that one cost will reduce another. I don't have the vandalism. I don't have the litter. I don't have the need for high security."



years. I see the kids this year as calmer, easier to talk to. They just seem more rational. I had thought about retiring this year and basically I've decided to teach another year -- I'm having too much fun!"

"I've taught here almost 30

- Dennis Abrahm, Middle School Science Teacher

New York City Public Schools:

Four Years of Success^{1,2}

In the spring of 1979, New York City's public schools ranked in the 39th percentile on standardized California Achievement Test scores given nationwide. That means that 61 percent of the nation's public schools scored higher. They had been in the lower half of the country for years. However, for a few years in the 1980s, these same 803 schools ranked in the upper half of the nation's schools. They went from 11% below the national average to 5% above it. What happened?

In the fall of 1979, the city's Board of Education decided to make some changes in their lunch and breakfast program. They ordered a reduction in sugar (and this would reduce dependence on prepackaged foods), and they banned two artificial food colorings. In the next set of achievement tests, the schools averaged in the 47th percentile – an increase five times larger than any other documented increase. Dr. Elizabeth Cagan, Chief

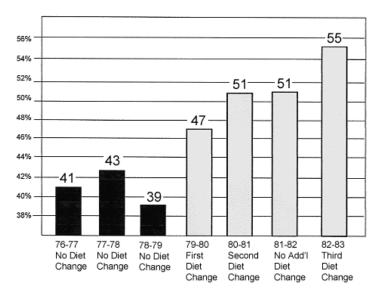
Administrator of the Office of School Food and Nutrition of the New York City Board of Education, and researcher Dr. Stephen Schoenthaler, studied the changes occurring during these years.

As more changes were made, bringing the school lunch and breakfast programs in line with "stage two" of the Feingold Diet – eliminating artificial flavoring and coloring, as well as the preservatives BHA and BHT – school scores rose to the 55^{th} percentile. This was a total rise of almost 16%, in a cohort of over a million children. Moreover, when the changes were analyzed, a dramatic difference was found in the ratio of change to amount of food eaten at school. Before these changes, the more school meals the children ate, the worse their scores. After the changes, this reversed: the more school meals the children ate, the better they did academically.

And that is not all – when Dr. Schoenthaler looked at which children had made such

National Rankings of 803 New York City Public Schools Before and After Diet Changes

Percentile Rankings based on CAT Scores



dramatic changes that the entire school system improved, he found that it was not uniform. Not *all* children made a 16% improvement. Rather, the lowest achievers improved the most. In 1979, before the Board implemented the dietary changes, 12.4% of the one million students in New York City schools were performing two or more grades below the proper level. These were the "learning disabled" and "repeat failure" children. By the end of 1983, only 4.9% of children were in that category. In other words, 7.5% of a million children – 75,000 children – were no longer "learning disabled" low-achievers but had become able to perform at the level normal for their age. These were the children that no other efforts had helped. No other hypothesis fits: all changes were related to the dietary changes.

1. Schoenthaler 1986

^{2.} Schoenthaler 1986a

What about the placebo effect – could that have explained it?

Dr. Schoenthaler analyzed this possibility, in detail, but came to the conclusion that it was not possible. A placebo effect would take place immediately and wear off. This did not happen. A placebo effect cannot explain the reversal in the correlation of children's scores with the amount of food eaten at school. Several other possible explanations were evaluated and rejected as not possible because they, too, simply do not fit the facts. The dietary change explanation, on the other hand, fits every fact observed.

A close look at the graph of student scores reveals two other interesting facts: Looking at the highest black bar, one could wonder if something had happened that year, too. Indeed, it had – the school had attempted to reduce fat in the school food. Again, this would decrease their dependence on prepackaged foods (usually

heavily laced with additives as well as fat), and the effort brought a modest rise in scores. The next year, that effort was abandoned – and the scores again dropped. What about 1981-82? Why does the level remain "stuck" at the 51%? That year, no further dietary changes were introduced. The food available to the children remained the same, and their academic results also remained the

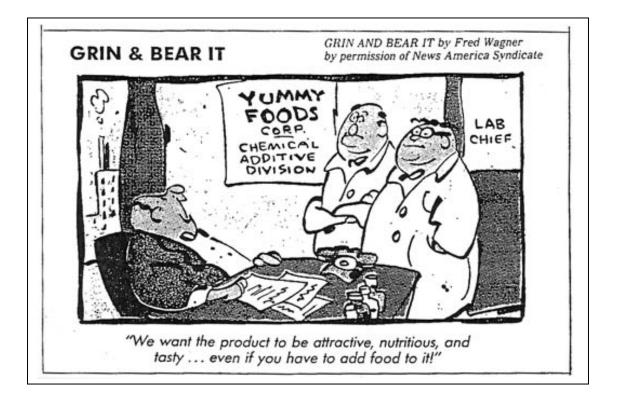
No other school district could be located which reported such a large gain above the rest of the nation so quickly in a large population. - Dr. S. Schoenthaler

same. The following year, when the food was improved by elimination of the petroleum-based preservatives BHA and BHT, average scores rose again -- to well above the national average.

See more about how other schools are helping their students by improving their lunch program – and how you can help your child's school do the same – at the website <u>www.school-lunch.org</u>

See more about recipes recommended for U.S. schools at http://teamnutrition.usda.gov/Resources/usda_recipes.html

See the American Academy of Pediatrics Policy Statement on Soft Drinks in Schools at http://portal.nysed.gov/portal/page/pref/CNKC/IntDocs/152.pdf



ADHD



According to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV), Attention Deficit Hyperactivity Disorder (ADHD) or one of its subtypes can be diagnosed if the child shows certain characteristics for a period of six months or more, with at least some of the symptoms beginning before age 7.

The symptoms are subjective, are generally described by a parent, and require:

1) Six or more symptoms of lack of attention, as paraphrased below:

- Fails to pay attention, makes mistakes...
- Has difficulty staying on tasks
- Does not seem to listen
- Fails to finish things
- Does not like homework or schoolwork
- Has trouble organizing things
- Loses things
- Is easily distracted
- Is forgetful

- OR -

2) Six or more symptoms of <u>hyperactivity-impulsivity</u>, as paraphrased below:

- Fidgets
- Leaves seat in class
- Runs around, is restless
- Has difficulty playing quietly
- Acts like "driven by a motor"

- Talks too much
- Blurts out answers
- Can't wait his turn
- Interrupts others

Depending on which symptoms above are reported, the child will be diagnosed as **ADHD-attentional**, **ADHD-hyperactive**, or **ADHD-combined**. For those who don't fit neatly into one of these three categories or who don't have quite enough symptoms for diagnosis, there is another diagnosis called "**ADHD**, **not otherwise specified**." Diagnoses such as ODD (Oppositional Defiant Disorder), Conduct Disorder and Explosive Disorder are descriptive of their major problem symptoms; medical treatment offered is often the same as for ADHD.

Many of the symptoms listed above overlap. For example, how would you separate the symptoms: <u>loses things</u>, is <u>forgetful</u>, and <u>has trouble organizing</u>? Are they really three separate symptoms?

The famous Multimodal Treatment Study of 1999¹ found that medication worked better than talk therapy and better than no therapy (they did not consider diet or any other "alternatives"). However, they



continued to follow the children in the study and eventually found² that although stimulant medications have dramatic short-term effect, they have little long-term benefit. A principal scientist in the study, psychologist William Pelham, told the *Washington Post*³ that the most obvious interpretation of the data is that the medications are useful in the short-term but ineffective over longer periods. His colleagues, he said, had repeatedly sought to explain away any evidence that challenged the long-term usefulness of

^{1.} MTA 1999

^{2.} MTA 2009

^{3.} Washington Post, March 27, 2009 <u>http://tinyurl.com/MTA-study</u>

medication, reaching for new explanations when each one failed to hold up. "The stance the group took in the first paper," he said, "was so strong that the people are embarrassed to say they were wrong and we led the whole field astray." He added, "If five percent of families in the country are giving a medication to their children, and they don't realize it does not have long-term benefits but might have long-term risks, why should they not be told?"

Another researcher, Peter Jensen, told the *Post* that the researchers had not misled the public, insisting

that some children do indeed do well on drugs over the long term. He claimed Pelham was the only member of the research team with the "silly message" that the study had raised questions about the long-term utility of drugs, but the Post said that Pelham was not alone.

While much attention is focused on the symptoms of ADHD, many of

these children are not just problems for their parents and teachers – they are physically sick.

It is now generally recognized that bed wetting and ADHD "go together;" ear infections and ADHD "go together;" sleep disturbances and ADHD "go together;" etc. These children have headaches, they have poor appetites, they can't sleep, they get ear infections, they have rings under their eyes, their skin seems dry, pale, or rough – they often simply appear to be unwell.

There is research that links each of these symptoms to diet,¹ and when the Feingold Program works for children like this, most or all of the symptoms seem to improve. It has also long been noted by a number of researchers that people with



^{2.} Brenner 1979; Ålberti 1999; Čarrie 2002; Gomez 2006; Hamazak 2002; McFadden 1996; Oades 1998



Something to consider is that the amount of food dyes our children are exposed to has risen 500% since 1955. Side effects of these drug-like chemicals were once a small problem. Now it is an epidemic.

ADHD may have abnormal levels of zinc, copper, manganese, lead, cadmium, essential fatty acids, electrolytes, sulfate metabolism, etc.² These things also may need to be addressed before the children will really be well.

Zinc is interesting, in particular, because two studies by a chemist in England³ showed that children with ADHD lose zinc when exposed to Yellow 5 and 6, but children without ADHD do not. Symptoms of zinc deficiency include behavioral effects as well as various physical effects.

Perhaps this is one reason for the dramatic response many of these children make to a change in diet – not just in attention, but in a multitude of symptoms, both major and minor.

Research on several hundred children in the UK^4 has shown that 20 to 62.4 mg of food dyes and preservative pushes ordinary children about 10% closer to an ADHD diagnosis.

Even if additives are

not the only cause of a child's problems, the researchers said, avoiding them may help. The Feingold Association agrees. We believe that children with learning or behavior problems deserve careful evaluation. Any underlying physical illnesses, vitamin deficiencies, heavy metal toxicity, and allergies should all be ruled out or addressed during diagnosis.

A brief trial of the inexpensive Feingold elimination diet can rule out - or identify - sensitivity to additives and/or salicylates.

If that is not sufficient alone, there are other possibilities to explore, including supplements⁵ which have been shown to be as effective as stimulants for some children.

^{3.} Ward 1990, 1997

^{4.} McCann 2007

^{5.} Harding 2003



Asthma

By using the Feingold Foodlist and Program information, you will learn how to eliminate the bronchoconstricting food additives, salicylates, sulfites, and many of the environmental chemicals likely to cause problems. Our Product Information Center and our more than 30 years of experience will make this process much easier. If you are a health care provider, we can be a resource for your asthmatic

patients. Patients with both ADHD and asthma will have a double benefit, as both symptoms may improve.

Then ...

In the mid-twentieth century, asthma was less common and was considered psychoso-

matic, caused by emotional conflict. Often, parents seeking help for their children's asthma problems found themselves blamed for it. Dr. M. Murray Peshkin,¹ medical director of the Children's Asthma Research Institute and Hospital in Denver from 1940 to 1959, coined the term "parentectomy," claiming that children developed asthma in response to an overbearing, rejecting mother. Seriously ill children sent to his clinic high in Colorado's clean mountain air did indeed improve quickly, often with no medication. Called "rapid responders," they lived there while their parents received psychotherapy back home. By 1958, however, the Institute's 98% rapid responders dwindled down to 28% and from there to zero.² Rather than looking to see what had changed in the Colorado environment (pollution? diet?), the experts decided the early "rapid responders" never really had asthma to begin with.²

and now ...

Today, according to WebMD, more than 22 *million people* in the U.S. have asthma, making

it the leading chronic disease in this country. As long ago as 1985, the American Academy of Pediatrics³ recognized the bronchoconstricting nature of some food dyes, as well as sulfiting agents such as potassium metabisulfite, potassium and sodium bisulfite, sodium sulfite, and sulfur dioxide. The Food and Drug Administration requires that Yellow 5 be listed by name on ingredient labels, due, in part, to their recognition of the danger this dye can pose for asthmatics. And yet – even today – in spite of this knowledge, parents are rarely advised to avoid foods containing these additives, and some asthma medications actually contain colorings and sulfites.

In recent studies,⁴ almost 2000 children were studied from before birth to 5 years. It was shown that when vitamin E and zinc are low in a mother's diet during pregnancy, her children are more likely to suffer wheezing and asthma. Since Yellow 5 & 6 cause loss of zinc in some people,⁵ perhaps pregnant women should be told to avoid the food dyes – or at least be tested for deficiency – to protect their children.

Some studies try hard to absolve the food dyes. One ⁶ began by saying that 84 of 90 studies on Yellow 5 (tartrazine) had "problems," and the other six didn't prove anything. This researcher went on to study 26 people, only 11 with asthma, and none with any history of reactions to tartrazine although they had other documented allergies (grass, dust mites, etc.). Giving these 26 people – ON their asthma-preventing drugs and NOT on a dye-free diet -- divided small doses of Yellow 5 totaling 35 mg, the researchers concluded that tartrazine is nothing to worry about.

We may not have much control over some asthma triggers, such as small-particulate pollution in the outside air, but we can pay attention to those over which we do have control: food additives, scented toys, children's vitamins, toothpaste, classroom disinfectants, markers, and other controllable sources of environmental toxins.

You can find relevant studies and information on the Feingold web site.⁷

^{1.} National Library of Science Breath of Life. http://tinyurl.com/Peshkin

^{2.} Childhood Asthma: Pathophysiology and Treatment by David G. Tinkelman, MD, publ.1993 by Marcel Dekker, pp.556-558

^{3.} AAP: Inactive Ingredients in Pharmaceutical Products, Pediatrics, 1985;76;635 http://tinyurl.com/Inactive-Ingredients

^{4.} Litonjua 2006, Devereux 2006b

^{5.} Ward 1990, 1997

^{6.} Pestana 2010

^{7.} Feingold Website pages: www.feingold.org/asthma.php, www.feingold.org/Research/asthma.html

Aggression and Violence

Disruptive Behavior, ODD, CD



We all know that violence has increased dramatically. Efforts at gun control, more prisons and severe punishments – all have failed to reduce our standing as the most violence-prone of all industrialized nations.¹

Experts have been calling for research on the causes, and the National Research Council recommends finding genetic and biological factors of "violence-prone" children.²

However, genetic factors do not change in one generation and cannot explain the whole story. It is time to begin paying attention to some research being done in another direction.

As long ago as the 1980's, studies in both schools and jails dramatically showed that a diet that removes additives and enhances nutrition brings significant improvement in behavior and academic performance. In 1985, Dr. Stephen Schoenthaler published a series of studies on 12 juvenile correctional facilities, housing 8,076 young offenders.³ Just as he showed in the school studies (*page 13*), not *all* the children improved, but 20% of them made such a dramatic recovery that the total "deviant behaviors" for all the children fell by 47%.

At a Tidewater, VA detention facility, behavior problems fell 48% following dietary changes: Violence declined 33%, theft dropped 77%, etc.⁴

A controlled study of 1,382 youths at three Los Angeles County probation detention centers found a 44% reduction in bad behavior,⁵ and a northern California probation department facility making similar dietary changes⁶ found that violence fell 25% and "horseplay" declined 42%. In both these California institutions, suicide attempts fell 44%.

Animal studies⁷ on the petroleum-based antioxidant preservatives BHA, BHT, and TBHQ have long shown them to cause decreased learning and grooming, increased activity, developmental delays and <u>aggression</u>.

Other studies⁸ have shown these preservatives to be carcinogenic as well. Isn't it time to simply replace these three petrochemical preservatives with others equally available but less harmful? Instead, in efforts to reduce transfats, we have greatly *increased* the use of these chemicals in food oils.

We will all have to pay the piper in the form of increased cancer, increased violence, increased school problems, and decreased academic performance.

^{1.} NY Times, Nov.13, 1992, Study Cites Role of Biological and Genetic Factors in Violence: <u>http://tinyurl.com/genetic-role</u>

^{2.} Ibid.

^{3.} Schoenthaler 1985

^{4.} Schoenthaler 1983, 1983a

^{5.} Schoenthaler 1986, 1991; See the experience of several schools at www.school-lunch.org

^{6.} Schoenthaler 1983b

^{7.} Meyer 1980; Stokes 1974; Tanaka 1993; Zoccarato 1987

^{8.} Bauer 2001, 2005; Kahl 1984, 1993; NIH 11th Report on Carcinogens; Sarafian 2002; Sasaki 2002; Thompson 1988, 1989

Again, studies¹ have shown that violence-prone males have abnormal copper-to-zinc ratios and that 75% of young criminals have allergy and nutritional problems.

In 1997 and 1998, Bennett¹ showed that when treated appropriately by diet and nutritional intervention, most young offenders improve and never re-offend. Even earlier, a chemist in the UK had found that children with ADHD lost zinc through their urine when exposed to Yellow 5 and 6, resulting in a variety of symptoms, including aggression and violence.²

Monkeys fed soy formula³ (which has much more manganese than breast milk) develop neurological and behavior problems. Some violent adolescents have been found to have high levels of manganese in their hair. Whether these children are the ones who simply cannot excrete excess manganese, or whether

they have actually been poisoned by soy formula as babies and perhaps other sources of manganese, is not yet known.

Since manganese has been proposed as a gasoline additive to replace lead, it is important to know what chronic exposure can do. A number of studies in recent years show that this chemical, although an essential nutrient, can be toxic at high concentrations. Chronic exposure to dust containing manganese in industrial settings can cause symptoms similar to Parkinson's disease. Low level exposure appears to cause neurologic changes, decreased learning ability, and increased violence.⁴ Appallingly, after discussing these implications, the author concludes that the research literature to date is not "strong enough" to justify concern about the effect

- 4. Finley 2004
- 5. Khan 2011; Schrauzer 1992



This is not an appropriate future for our troubled children. We may not be able to provide every child with two loving parents, but we really can improve nutrition, reduce toxins, and even test for metabolic abnormalities through our schools and medical services.

of manganese on "healthy adults" in the North American population.

Some other recent studies⁵ document the effect of manganese and arsenic in water on the intelligence, behavior, and aggression of children, the interaction of lithium and vitamin B12, etc.

In 1989, the Kellogg Report⁶ said, "Nutrition, lifestyle choices and the state of our environment hold solutions to many of the crises which beset society." They go on to say, "Many who readily accept the link between diet and heart disease or other chronic physical conditions, find it hard to imagine that nutrition could have a direct and determining effect on human behavior and personality dysfunctions."

A 2005 review of the literature on violence discusses the effects of cholesterol and hormone levels, nutritional deficits, prenatal/postnatal exposure to metals, smoking and other toxins, iron, zinc, neurotoxins, brain injury, and family environment.⁷

Organizations and families dealing with violent children (and adults) must begin to consider the role of foods, additives, heavy metal exposure, essential fatty acid levels, vitamins, and other dietary factors. Diet change alone may help some, but if it doesn't appear to "work," then these other environmental chemicals as well as vitamins and enzymes which effect neurotransmitters must also be considered.

^{1.} Walsh 1997; Bennett 1997, 1998

^{2.} Ward 1990, 1997

^{3.} Cockell 2004; Golub 2005

^{6.} The Kellogg Report 1989

^{7.} Liu 2005

Enuresis (Bedwetting)

As if the child with learning and behavior problems doesn't have enough to deal with, bedwetting (*nocturnal enuresis*) and daytime wetting (*diurnal enuresis*) may be another part of their daily struggle. ADHD children at age 6 are **2.7 times more likely** than controls to have nocturnal enuresis and **4.5 times more likely** to have diurnal enuresis.¹ It has been known since at least 1976 that an improvement in diet can cure enuresis in many children.² In 1992, Egger et al. reported on 21 children with enuresis who had been successfully treated by diet for either hyperactive behavior or migraines. For 12 of them, the enuresis stopped, and for another 4 it improved. They confirmed this by a double blind follow-up study.³

Although the Feingold Program has never been promoted as a bedwetting "cure," over the years, parents have frequently reported that one of the benefits they have seen with the Feingold Program is the disappearance of bedwetting.

Seizures, Headaches, other Physical Problems

HEALTH

BEHAVIOR

LEARNING

Other symptoms also often improve on the Feingold Program. When implementing the diet for behavior problems, parents are more often than not surprised that the child's (or their own) headaches, sleep difficulties, GI problems, skin problems, etc. are suddenly gone, as well.

There are many symptoms that "travel with" the symptoms of ADHD but are often either treated as

separate illnesses or ignored altogether. Besides the asthma and bedwetting already discussed, some people suffer from chronic headaches or migraine, frequent earaches, stomach aches, trouble sleeping, chronic dehydration, dry or "allergic" skin conditions, seizures, etc. Not all people have all these symptoms, of course, but all the people who respond

to dietary intervention "fit" somewhere in the profile of symptoms on page 1, represented here by three interlocking circles. It is astonishing how many parents report that their children have "all" the symptoms listed – and yet by simply changing their diet, all or most of their problems improve or disappear.

In research, this has been shown repeatedly by studies on migraine, seizures and enuresis. Egger found that in 45 children with epilepsy as well as various physical or behavioral problems listed in "our" symptoms list, 80% of them improved on his elimination diet. However, of the 18 children with epilepsy alone and no other symptoms, none improved.⁴

- 1. Robson 1997
- 2. Salzman 1976
- 3. Egger 1992
- 4. Egger 1989

Egger 1983, 1985
 Ward 1990, 1997
 Oades 1998
 Megson 2000
www.feingold.org/megson.html

In other studies,⁵ Egger found that 93% of 88 children with frequent migraine, and 81.6% of 76 overactive children, recovered on an additive-free diet. Again, other symptoms these children had, and which also improved, included abdominal pain, behavior problems, seizures, asthma, and eczema.

Ward,⁶ a British chemist, found that ADHD children lost zinc in response to exposure to Yellow 5 and 6.

Symptoms exhibited included asthma, speech problems, behavioral deterioration, eczema, and aggression. And in 1998, Oades found that children with ADHD drank four times as much water as "normal" children, yet tended to remain dehydrated, had twice the normal level of neuropeptide Y, and excreted more norepinephrine and a serotonin metabolite, but less sodium, phosphate

and calcium, than normal children.⁷

Could these findings indicate a genetic difference? Possibly. Or could it indicate metabolic damage by vaccination or other chemical exposure, as suggested in a Congressional Committee hearing?⁸ Might the damage itself involve sulfur metabolism, explaining the phenol sulfotransferase link? Also possible. Or perhaps the implicated additives are akin to drugs, and what we see as symptoms are actually "side effects?" Again possible. What is clear to us, at least, is that the difference in these children is at a level basic to many bodily functions. Treatment at the specific receptor level, as is done with stimulant medication, may relieve some symptoms, but is never a cure. A better choice, often with better results and no side effects, is appropriate dietary change. Certainly, diet is worth trying first - and worth continuing even if medication must be added in individual cases for maximum relief.

Autism Spectrum Disorders

Autism, High Functioning Autism, Pervasive Developmental Delay (PDD), Asperger's Syndrome

Web sites worth visiting:

www.autismNDI.com - Autism Network for Dietary Intervention

www.autism.com/pdf/providers/ParentRatings2009.pdf

www.autism.com - Autism Research Institute

- Parent Ratings of Biomedical Interventions

- at Congressional Committee, April 2000

www.gfcfdiet.com - Gluten-Free, Casein-Free Diet

www.feingold.org/megson.html - Mary Megson, MD,

www.nvic.org - National Vaccine Information Center

Not so many years ago, autism was a rare disorder, affecting **3 in 10,000** children. In 1997, the Autism Society of America reported that autism occurred in approximately **1 of every 500** births, the symptoms usually becoming apparent during the first three years of life. This was already, at that time, considered an epidemic.

In 2006, however – less than 10 years later – the Centers for Disease Control acknowledged that **1 in 150** children in the U.S. is autistic. In 2007, British researchers confirmed that the rate of autism in Great Britain had risen to **1 in 58**, and in 2011 a study

Research studies underway in England, Norway, and the United States are investigating biochemical processes and genetic errors in patients with autism. These include gluten and casein intolerance, phenolsulfotransferase (PST) deficiency, and others. Hopefully, research will soon reveal methods to detect those children at risk, to protect them before they become autistic.

Many families have reported that symptoms of autism improve by using the Feingold Program alone or in combination with a gluten-free, casein-free (GFCF) diet. Some Feingold members find that their

carried out in South Korea revealed the startling rate of 1 in 38.¹

Autism is the fastestgrowing developmental disability today.

Much of the on-going research is devoted to discovering a genetic predisposition for it. Worth keeping in

mind, however, is that it is simply impossible to have an epidemic – and this IS an epidemic – of a genetic disorder unless gene variations that were harmless for centuries have now suddenly been impacted by some change in the environment.^{2, 3}

A growing body of evidence suggests that observable symptoms of autism are linked to biochemical intolerances, allergies, or metabolic errors. It is believed that foods and environmental factors play a major role. One of the environmental changes causing much controversy is the increase in vaccine exposure. According to the National Vaccine Information Center, safety research on these vaccines is deficient, and the vaccines themselves certainly contain enough chemical toxins to pose a problem for any babies who may (genetically) have difficulty dealing with them.⁴ children benefit from the GFCF diet, and some doctors now put ADHD at the mild end of what they are calling ASD, or Autism Spectrum Disorders.

ADHD

One impressive

nonprofit organi-zation is the Autism Research Institute (ARI) which was founded by the late Dr. Bernard Rimland, the first to recognize autism as a biological disorder rather than a mental illness. Since 1967, the ARI has collected data from more than 27,000 parents of autistic children, rating the interventions they have used for their children. More than 40 drugs have been rated, from Adderall to Zoloft, as well as 26 supplements and 9 dietary variations.

Overall, the supplements and diets show higher success rates than the medications. The rate of negative responses (getting worse), moreover, is much higher for medications than for the other treatments.

The most successful diet is the Specific Carbohydrate Diet (71%), followed by the GFCF Diet (69%), the Feingold Diet (58%) and the Candida Diet (58%).

^{1.} Kim 2011

Hunter-gatherer tribes rarely suffered from diabetes until they adopted the Western diet and culture which they were not genetically able to tolerate. Almost immediately, they developed an "epidemic" of diabetes afflicting up to 50% of their population.
 Pima Indians, Genetic Research

www.diabetes.niddk.nih.gov/dm/pubs/pima/genetic/genetic.htm

^{4.} National Vaccine Center - www.nvic.org

The PST Connection

Some people have too little of an important enzyme called phenol sulfotransferase (PST).¹ It is made in the intestines, which need PST to metabolize (detoxify) the phenolic compounds in many foods, including salicylates and the high-phenolic petroleum-based additives. However, the brain also requires PST for "housekeeping" duties involving neuro-transmitters – those chemicals which jump the tiny space (synapse) between brain cells (neurons). Each time a neuron "fires" and the neurotransmitter "jumps" that space, PST must prepare the space to "fire" again. This is measured in *nanoseconds*, occurs millions of times a second all over the brain and must be perfectly synchronized.

If a person is marginal or low in PST and eats lots of high-phenolic foods and additives, there may not be enough PST left to do the "clean up" work in the brain, thus preventing neurons from firing effectively.² Moreover, it seems that salicylates (which are also phenolic compounds) not only need PST but actually suppress its production,³ making PST levels even lower. This may help explain why the avoidance of



salicylates at the start of the Feingold Program is important. Once suppression is stopped, there may be some recovery, leading to improved tolerance of salicylates later on. Surely, this is only part of a larger and complex picture, but in this area the circumstantial evidence is mounting. More at page 41.

In practice, the Feingold Program guides parents in choosing a low-phenolic diet, taking stress off a fragile sulfation system.⁴ This may be especially important for people with autism, who have been shown to have extremely low PST levels. Other interventions that may help include avoiding sources of sulfite (SO₃), while increasing sources of the sulfate (SO₄) which is needed for PST production. Some people increase sulfate through the skin (Epsom salt baths) or by drinking Evian water.

Unfortunately, many children with autism crave the

casein and gluten that hurts them - like little drug

addicts, they need their "fix." Parents of such children

report that their child's whole diet consists of macaroni

& cheese, cereal & milk, bread & butter, pizza, cheese

puffs, cheese sandwiches, puddings, etc. Moreover,

these children may have sensory problems related to

diet - some tolerate only soft foods, while others can't

stand the feel of soft foods and require crunchiness.

Removing casein and gluten quickly from such a

child's diet may be a Mission Impossible task. We

recommend a slower approach, beginning with the

much easier "regular" Feingold Diet - which alone

The Gluten/Casein Connection

A baby nurses, falling asleep when full. This is due partly to endorphins made by the baby when tasting milk and partly to the milk protein itself which enters the baby's blood in a morphine-like form.⁵ This "leaky" gut is normal in babies and is one reason that babies may develop allergies if given solid foods too early – because when other partially digested proteins get through the gut wall and into the blood, where they don't belong, they may be treated as invaders by the infant's developing immune system. Toward the end of his first year, the baby's intestinal wall becomes less permeable, allowing tolerance for new foods. However, if anything has happened to prevent this, damaging the delicate intestinal system, the growing child may experience symptoms of digestive distress, allergies, or cognitive problems. It has long been known that incompletely digested proteins can cause allergies. Less well known is that the incompletely digested casein protein (casomorphin) and gluten protein (gluteomorphin or gliadorphin) both act as morphines, possibly causing symptoms of autism, ADHD, or even schizophrenia.

may decrease some symptoms and improve appetite. Meanwhile, several tests provided by the Great Plains Laboratory⁶ can help determine whether the child actually needs a gluten/casein free diet, whether he may be deficient in zinc or other minerals, etc. If necessary, casein and gluten items can be replaced by substitutes very slowly, a tablespoon per day, for example. Remember that this is an addiction condition, and the child may have serious withdrawal symptoms, including behavioral deterioration, if changes are made too quickly.

^{1.} Alberti 1999; Scadding 1988; Sinaiko 1996

^{2.} Bamforth 1993; Harris 1996; Sinaiko 1996

^{3.} Harris 1998 **4.** McFadden 1996 **5.** Blass 1996 **6.** Great Plains Lab: 1-913-341-8949

<u>GPL4U@aol.com</u> <u>www.greatplainslaboratory.com</u>

A Call for Better Research

Animal studies on food dyes traditionally focus on whether they cause cancer, damage reproduction, or distort physical development. Very few studies have considered the effect(s) on cognitive function in either animals or people – and when they do, they use dye levels far below "real world" amounts.

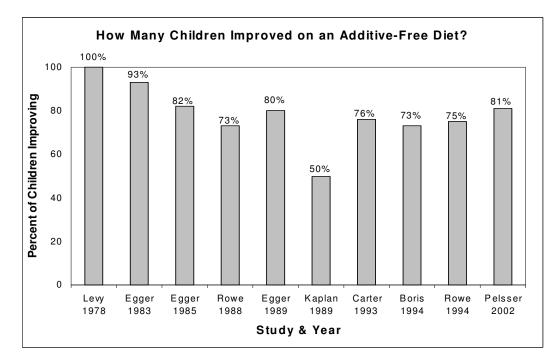
In the 1970s, a food industry group calling itself the Nutrition Foundation recommended **27 mg per day** of artificial food colors in human studies on food additives and behavior. Meanwhile, a National Academy of Sciences study on 12,000 people showed that the top 1% of them ate **335 mg** of dye per day with an average of **86 mg** for all "dye eaters" over two years old (in 1977). Even if you discount the "lakes" which are dye mixed with aluminum or calcium salts, you still get far more than the Nutrition Foundation's fairy tale number of 27 mg. And none of this takes into account today's blue soda, colored applesauce, fluorescent cereals, striped toothpaste, and other such questionable inventions. One must assume today's rate is higher, especially for children, to whom many of these "fun foods" are marketed. In fact, the average consumption of artificial food dyes today is <u>five</u> times higher than it was in 1955.

For the 2011 Hearing on Food Dyes, the FDA published a list of what "high users" might eat – calculated as if only 10% of the population ate all the dye. The FDA's point was that each dye amount was still "below the Allowable Daily Intake (ADI)." This is true – but where is **any study** showing that a total dye amount of 450 mg is safe for children? Nor has **any study** ever been done on the ADI amounts themselves.

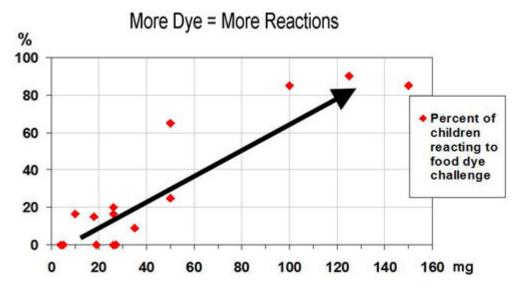
Food Coloring	Mg/Day per National Academy of Science 1977 Survey 2+ yr old "eaters"		Mg/Day for "High User" as calculated by FDA for 2011	Mg/Day Nutrition Foundation Recommended for Research on
	Mean	Top 1%	Hearing	ADHD & Diet
Red 3 + Red 3 Lake	10.3	39.0	6.1	1.6
Yellow 5 + Yellow 5 Lake	17.4	65.6	120.6	7.3
Green 3	1.0	4.3	0.38	0.1
Blue 1 + Blue 1 Lake	4.95	21.6	17.2	0.8
Yellow 6 + Yellow 6 Lake	12.9	51.0	107.4	6.1
Blue 2 + Blue 2 Lake	1.6	10.9	19.5	0.5
Red 40 + Red 40 Lake	35.6	135.0	179.1	10.5
Orange B	2.2	7.8		0.1
TOTAL	85.9	335.2	450.3	27.0

The Feingold Association would like to see:

- 1. Research measuring the amount of food dye actually in common foods children eat.
- Research using a more realistic amount of food dye a likely maximum, not an average or minimum amount. How much do they really get at a birthday party, for example? No studies have ever been done testing the neurological effect of 300 mg or more of food coloring on children.
- 3. Research investigating combinations of food dye plus other additives to test for interactions.
- 4. Research on the effect of artificial food dye on cognitive function animals and humans.
- 5. Research on the effect of food dye on mood, irritability, sleep, and other symptoms beyond ADHD.
- 6. Research on the Feingold Program as it is really used in the real world.
- 7. Studies of the prevalence and relative toxicities of the different natural salicylate compounds.



In the studies above, the researchers put the children on an additive-free diet similar to the Feingold Diet. As you can see, a high percentage of children improved. Later, each researcher gave the children an additive or group of additives in a double-blind test. The results varied, depending upon the type and amount of challenge material.



The above graph shows the number of children reacting to dye depending on amount of dye used. And this is for dye alone. There are **more than 12,000 other food additives now in use but never tested for behavioral or neurological effects.** Safety studies usually test each additive alone, although we eat lots of them together, often combined in a single product. In 2006, a landmark study¹ found that an additive plus a dye together inhibited developing neurites far more than they were expected to do, based on the damage each caused alone.

^{1.} Lau 2006

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Studies are listed alphabetically in each section by last name of primary author.

 \star = Double-Blind Placebo-Controlled Study

The Nutrition Foundation

It is important to understand that much of the early research was designed and funded by the Nutrition Foundation. What was this foundation? It was a trade industry organization, now called the International Life Sciences Institute (ILSI). In 1977, Nutrition Foundation members included representatives from:

- ➢ Hoffmann-LaRoche, Inc. − pharmaceuticals
- Fritzsche-D & O, Inc. artificial flavors
- Stange Company *artificial colors & flavors*
- Florasynth, Inc. *artificial flavors*
- ➢ Kohnstamm & Co. − artificial colors & flavors
- ➢ PFW, Inc. − artificial flavors
- Monell Chemical Senses Ctr. artificial flavors
- ICI Americas, Inc. dyes, pesticides, petrochemicals
- ➢ Ajinomoto CO., Inc. − MSG
- Griffith Laboratories *nitrites*
- The Coca-Cola Company
- CPC North America corn syrup
- Amstar Corporation *sugar*
- Revere Sugar Corporation *sugar*

The Role of Diet in Behaviour Ben F. Feingold, MD

Written just before his death & published posthumously in Ecology of Disease. 1982. 1(2-3) pp.153-65.

"The increase in behavioural disorders accompanied by a persistent drop in scholastic performance coupled with the continuing rise in the prevalence of delinquency is undoubtedly one of the most important expressions of the disruption of nature by the rising concentration of pollutants in the ecosystem ... Public recognition and participation in the problem are mandatory to correct the insidious downgrading of the human race, which is already evident."

www.feingold.org/Research/PDFstudies/Feingold82.pdf

Concerns About the Research on Coloring

- **Double-blind "challenge" studies:** Usually colorings alone are used as challenges, often only a small amount of a single color. The other additives eliminated by the Feingold Program are usually ignored.
- The increasing use of synthetic colorings without adequate testing: One study (Husain 2006) revealed that children in Kuwait eat <u>2 to 8 times more than the acceptable daily intake</u> (ADI) of food coloring. Others found that several colors exceeded the ADI in Brazil (Machinski 1998) and India (Tripathi 2010). How much food coloring are American children eating? Nobody appears to be looking.
- Since companies refuse to reveal how much coloring is in their products, how do you know how much you are actually eating? In cooperation with the Center for Science in the Public Interest, we have sent several products to a laboratory for analysis. Look at some of our results, just for Red 40:

 - > 1 cupcake with red frosting 58 mg Red 40
 - ➢ 5-ounce bag of Twizzlers 10 mg Red 40

NO STUDIES approaching the ADI have ever been done on any children, with or without ADHD!

Concerns About Medications

Stimulant medications appear to work for the majority of children - often dramatically. Unfortunately, one of the worst side effects of these drugs – <u>small vessel disease</u> – can be diagnosed only by viewing the heart at autopsy. People using both the Feingold Program and stimulant medication report needing much less medication. As far as we know, no research has yet been done to explain this.

- **Bailly** 2006 Since the introduction of **Selective Serotonin Reuptake Inhibitors** (*SSRIs, e.g. Prozac*) in the 1990s, reported side effects include excitation, restlessness, disinhibition (*acting out*), and self-injurious thinking and behavior. The authors warn that side effects must be monitored frequently.
- **Brown** 1989 ★ 11 black male children with ADHD were given placebo and **Ritalin** for two weeks each. They had a significant increase in blood pressure on Ritalin and should be monitored carefully.
- **Castner** 2003 Primates given **amphetamine** develop monoamine dysregulation and hallucinations. Symptoms include looking at and reaching for things not there, and hypervigilance.
- **El-Zein** 2005 12 children were tested before and 3 months after starting on **Ritalin**. <u>In all of them</u>, chromosome abnormalities were tripled. The relationship between chromosome abnormalities and cancer is well-documented.
- **Findling** 2011 There is essentially no research addressing stimulant medication-related emotional responses such as irritability (or aggression, crankiness) sadness, moodiness, mood swings, flattened affect, or depression.
- **Food & Drug Administration** 2005 Manufacturers were ordered to add a **"Black Box" warning** to the labeling of all **antidepressant** medications because they can cause suicidal thoughts and behavior.
- **Food & Drug Administration** 2007 Manufacturers were ordered to add a "**Black Box**" warning to the labeling of all drug products approved for the treatment of Attention Deficit Hyperactivity Disorder (ADHD) to alert patients to the risk of increased blood pressure, stroke, heart attack, worse behavior, bipolar illness, increased aggression, and psychotic or manic symptoms, as well as sudden death in patients with (unrecognized but theoretically pre-existing) heart defects.
- **Henderson** 1995 Small lesions (*damaged areas*) were found in the myocardium (*heart wall muscle*) of a patient treated with **Ritalin**. Rats and mice were injected with various doses of Ritalin, and their hearts examined. Heart damage was found in all rats, even with the smallest dose given for the shortest time.

- Kelly 1988 ★ In 47 children with ADHD, doses of **Ritalin** were linearly related to increasing heart rate, depending upon both the initial rate and the length of time on medication.
- Markowitz 1999 Ethylphenidate was found in the blood and liver of people who died after taking Ritalin (*methylphenidate*) and alcohol (*ethyl alcohol*). The authors do not know what this chemical does or if it is toxic. Note: Taking Ritalin in order to drink more alcohol without passing out is a new "party" activity.
- MTA 2009 After 8 years of treatment and followup, children still taking medication fared no better than their nonmedicated counterparts (except for math) despite a 41% increase in the average total daily dose, failing to support continued medication treatment as salutary.
- Negrao 2009 Methylphenidate causes an increase in heart rate, and in systolic and diastolic blood pressure.
- **Olfson** 2006 Antidepressant drug treatment in children under 19 was significantly associated with suicide attempts and deaths. Antidepressants are sometimes used with ADHD treatment.
- Wang 1994 Ritalin decreased blood flow in all regions of the brain in 5 healthy men, up to 30% in some regions. The authors recommend that this effect on blood vessels be considered when prescribing.

Research on Colorings and Flavorings

NOTE: The U.S. Food & Drug Administration (FDA) certifies all batches of synthetic coloring. They are not certified to be *safe*. They are certified to have "acceptable" levels of certain contaminants such as:

- See Lancaster 1999)
- Lead, not more than 10 parts per million. (Colorings in drugs & cosmetics can have 20 ppm.)
- ✤ <u>Arsenic</u>, not more than 3 parts per million.
- ★ <u>Mercury</u>, not more than 1 part per million.
- Abd El-Wahab 2012 In 10 groups of rats, 3 food dyes and 3 synthetic flavorings were tested singly at the ADI level and in combinations of a dye plus a flavoring. All treated groups ate more than controls but lost weight. They had reduced hemoglobin and red blood cell counts, and enzyme changes indicated liver and kidney damage.
- **Abdel-Aziz** 1997 In mice, Red 3 reduced sperm count by 50%, reduced the number of moving sperms by 57%, and increased the number of sperm abnormalities.
- Aboel-Zahab 1997 A combination of food colorings were fed to healthy adult rats. Results included:
 - Decreased body weight, hemoglobin, and red blood cells
 - Increased thyroid hormone, cholesterol, triglycerides
 - Increased liver enzymes
 - Brown pigment deposit in liver and kidney tubular cells
 - ✤ Areas of hemorrhage in both liver and kidneys
 - ✤ Abnormal balance between types of white blood cells

Allen 1984 – Food intolerance symptoms can be caused by small molecules in the food or additives. These reactions are pharmacological (*like drug side effects*) and do not show up on IgE allergy testing.

In summary, certified food dyes can:

- Make you hyperactive
- Give you cancer
- Damage your sperm
- Damage your liver
- Lower your immunity
- Raise your cholesterol
- Decrease your brain size
- Trigger an asthma attack
- ✤ Give you hives
- Make you cranky
- ✤ Damage your nerves
- Amin 2010 Yellow 5 and Carmoisine (E-122, not used in US) alter biochemical markers in the liver and kidneys at low doses with a dose-effect.

- Aoshima 1997 The effects of certain chemicals and additives on GABA (*inhibitory neuron*) receptors were measured. Results indicated that food additives can measurably modulate the neural transmission in the brain, which "changes the frame of the human mind, as alcohol or tobacco does."
- Ashida 2000 Artificial food colors may impair hepatic (liver) function.
- Augustine 1980 In frog nerves, Red 3 produced a dose-dependent increase in neurotransmitter release.
- **Bamforth** 1993 Yellow 5 and the artificial flavoring *vanillin* inhibit the enzyme dopamine sulfotransferase. Vanillin also inhibits by 50% the metabolism of a birth control medication which is sulfated in the liver.
- Ceserani 1978 Yellow 5 causes bronchoconstriction in some aspirin-sensitive people, just like aspirin.
- **Dalal** 2009 Testing Red 3 on neurochemicals involved in hyperactivity, results suggest Red 3 may reduce serotonergic activity with modulation of central dopaminergic activity depending on the brain region.
- **D'Souza** 1987 Aspirin, Indomethacin and Yellow 5 (0.1-2.0 mg/kg) induced dose-dependent increases in carotidsinus nerve activity, accompanied by increases in mean arterial blood pressure.
- **el-Saadany** 1991 Synthetic colorings and flavoring were given to adult rats. Serum protein, RNA and T4 (*thyroid*) hormone were increased. Nucleic acid enzymes were stimulated in all the organs studied. G-6-PD and 6-PGD activity increased. Coloring and flavoring together resulted in the highest increases.
- **Food & Drug Administration Public Health Advisory** 2003 Doctors must stop using Blue 1 in enteral (*tube feeding*) solutions because it causes blue skin, urine, feces, and colon, and sometimes metabolic acidosis and death. Blue 1 is a mitochondrial toxin, used for 30 years in tube feedings without any safety studies.
- **Gao** 2011 Mice and rats on tartrazine (Yellow 5) were more active but took longer to escape a maze and had memory loss. Their brains showed oxidative damage.
- **Groten** 2000 The author claims that combining unrelated additives is not a health concern because of the low doses involved. *Note: The journal which published this is owned by ILSI a food industry organization.*
- Hashem 2011 Red 2 and Yellow 6 can impair liver function. When these additives were fed to pregnant rats, many of the pups were growth-retarded and/or had skeletal and visceral abnormalities. Only 3% of pups of the rats fed high doses of curcumin (a natural coloring) were growth-retarded, and they had no other abnormalities.
- Hassan 2010 The colorings Tartrazine (Yellow 5) and Chocolate Brown (not used in US) caused DNA liver and kidney damage detected by the comet assay.
- Hedman 1981 Tiny amounts of Yellow 5 cause contractions in the trachea smooth muscle of guinea pigs.
- **Helal** 2001 Rats fed sodium nitrite with Yellow 6 had a decrease in body weight and fewer red and white blood cells. A number of tests for glucose, thyroid, calcium, cholesterol, and enzymes were abnormal, leading to the conclusion that "even the permitted doses of colourants and food preservatives may be harmful."
- **Ibrahim** 2008 Upon exposure to sunlight, Yellow 6 in bottled drinks is degraded to benzene sulfonic acid sodium salt which caused liver damage and an increase in tumor markers in rats. Treatment with either ginger or green tea prevented damage and promoted recovery.
- **Kamel** 2011 This study in rats concluded there is sufficient scientific evidence of a true causal link between tartrazine (Yellow 5) and hyperactivity, anxiety and depression-like behavior. The authors warn of the hazardous impact of tartrazine on public health.

Koutsogeorgopoulou 1998 – Results showed clear immuno-suppressive effects of Red 2 and Yellow 5.

Kroes 2000 – The Threshold of Toxicological Concern and **de minimis** concepts (*"a little bit can't hurt"*) can be used to evaluate additives, saving the cost and time needed to actually test additives for safety.

- Kroes 2002 Using the Threshold of Toxicological Concern, a de minimis value can be set for chemicals of unknown toxicity. This method of "safety evaluation" is approved by ILSI and is now used by the FDA and WHO to evaluate flavoring substances. ILSI (International Life Sciences Institute) is composed of companies that make food additives, pesticides, snack foods, etc.
- Lancaster 1999 FDA allows only 1 part per billion (1 ng/g = <u>one</u> nanogram per gram) of benzidine in food dyes because it is so highly carcinogenic. Testing commercially available food colors, Lancaster found levels up to 270 ng/g – MUCH higher than the amount allowed by the FDA's own regulations.
- Lau 2006 Inhibition of neuron growth indicates neurotoxicity during development. Testing the amount of additives often found in snack foods, Lau combined <u>Blue 1 + MSG</u>, and <u>Yellow 10 + Aspartame</u>. The combinations were synergistic, far more toxic than expected by adding up the effect of each one tested alone. <u>Blue 1 + MSG</u> was 4 times as toxic, and <u>Yellow 10 + Aspartame</u> was 7 times as toxic. Note: Yellow 10 is not used in snacks in the U.S., but is used in medications, cosmetics, etc.
- Mehedi 2009 Mice fed Yellow 5 in their water have fewer sperm and more sperm abnormalities.
- **Mizutani** 2009 Red 2, Red 3, and Yellows 5 & 6 attack drug metabolizing enzymes, but hopefully won't interfere with drug metabolism "under normal conditions." Patients with ulcers should avoid them. Since the dyes react to light, causing rough skin, people with facial inflammation should avoid cosmetics, say the authors.
- **Mpountoukas** 2010 Red 2, Red 3, and Yellow 5 tested on human blood cells in vitro bind directly to DNA, causing its degradation, and indicating a toxic potential to human lymphocytes.
- National Academy of Sciences 1979 From 2 weeks of data on 12,000 people, the NAS determined that people may eat up to an average of 335 mg food dye per person per day (the top 1% of 2-week averages analyzed). For reasons unknown, they divided this number by 5 for their final report.
- Peng 2009 Blue 1 can prevent paralysis when used on spinal cord injury. Note: We always said it's a drug!
- **Reyes** 1996 All food dyes tested inhibited mitochondrial respiration. Percentage of inhibition varied per color and was dose related. *Note: Mitochondria control the energy in your cells. Inhibition is not good.*
- **Rosenkranz** 1990 In chemical studies, one of the aromatic amines obtained upon reduction (*a part of digestion*) of Red 40 was unexpectedly mutagenic (*making mutations or changes in DNA*).
- Sasaki 2002 ★ Low levels of each food dye tested on mice caused DNA damage in the stomach, colon, and bladder.
- Shimada 2010 Azo dyes caused colon-specific DNA damage in mice but not rats, which may be insensitive.
- **Sweeney** 1994 Intestinal bacteria "reduce" the azo bond in azo dyes, producing superoxide free radicals, thus confirming that azo dyes are a source of genotoxic agents (*resulting in mutations or cancer*).
- Tanaka 1993 ★ Red 2 was fed to two generations of mice. The pups weighed more and had trouble turning over and finding a source of an odor. Movement was affected, and more pups died.
- Tanaka 1996 ★ Yellow 6 was fed to two generations of mice. There were unspecified adverse effects on litter size, weight, and sex ratio. Pups had trouble with surface righting (*turning over*), negative geotaxis (*crawling upwards*), swimming direction, and swimming head angle. The effects were dose-related.
- Tanaka 2001 ★ Red 3 was fed to two generations of mice. Movement and other changes were dose-related.
- Tanaka 2006 ★ When mice ate Yellow 5, activity and body weight increased, and some developmental milestones changed. "Nevertheless," says Tanaka, "the actual dietary intake of tartrazine (*Yellow 5*) is presumed to be much lower." *Note: This is a presumption of safety, apparently based on nothing.*
- **Tsuda** 2001 ★ Very low doses of three azo food dyes fed to mice caused DNA damage in the colon, lung, bladder, etc. Damage was observable as early as three hours after they ate it. Tsuda says, "More extensive assessment of azo additives is warranted."

- Vorhees 1983 ★ Red 40 produced physical and behavioral toxicity in rat pups at high doses (10%). When rats ate Red 40, their reproductive success, parent and pup weight, brain weight, survival, and female vaginal development were reduced. Running wheel activity decreased, and open-field rearing activity increased.
- Ward 1990 ★ Yellow 5 reduced zinc in blood and saliva, and increased urinary zinc in children with ADHD but not in the controls. Zinc loss corresponded to deterioration in behavior and emotional responses.
- Ward 1997 ★ In hyperactive children, Yellows 5 and 6 significantly lowered zinc levels, causing one or more of the following symptoms: overactivity, aggression, violence, poor speech, poor coordination, asthma, eczema. Hyperactive children were low in zinc and iron, but high in aluminum, cadmium and lead.
- **Worm** 2001 In people with atopic dermatitis and food intolerance, additives (*Yellow 5, benzoate, nitrite, etc.*) cause white blood cells to make more leukotriene, a chemical contributing to allergic reactions and asthma.

Research on the Three Preservatives

Note: No studies have been found on the effects of BHA, BHT, or TBHQ on human behavior, and the only one on animals is the one by Stokes in 1974 (see below).

Bauer 2001 – Butylated hydroxytoluene (BHT) increases lung tumors in certain kinds of mice. Thus, BHT is used with other tumor and inflammation promoters to increase tumor production for research.

- **Bauer** 2005 In a study of chronic pulmonary disease and how it causes lung cancers, the researchers used BHT together with other promoters to maximize the available mouse tumors to study.
- **Dengate** 2002 ★ Calcium propionate (a bread preservative) caused irritability, restlessness, inattention and sleep disturbance in children on an additive-free diet. *Note: Products with this preservative are marked with a* "(*CP*)" in Feingold Foodlist books.
- Engin 2011 ★ Before giving rats a known endotoxin, they were pre-treated with either BHT, L-Arginine (*an amino acid*), or saline (*placebo*). BHT pre-treatment caused "remarkable liver injury" at all time points.
- **Fisherman** 1973 ★ 250 mg BHT in food caused an asthma attack in 75 minutes in some asthmatic patients.
- Kahl 1983 Feeding rats BHT increases some chemicals but decreases others in hepatic (liver) microsomes.
- Kahl 1984 BHA and BHT do protect against radiation and have anti-tumor actions, but their use in the prevention of human cancer is "unlikely" in light of their ability to promote tumors themselves.
- Kahl 1993 BHT is toxic to the lungs and causes liver tumors. BHA causes forestomach tumors. Vitamin E (*alpha tocopherol, a natural antioxidant*) is not carcinogenic and is safe to use in higher doses.
- LeClercq 2000 The theoretical maximum daily intake of BHT is above the current ADI in Italy.
- McFarlane 1997 ★ Pregnant rats fed a "nominal" dose of BHT (500 mg/kg) had liver enlargement and abnormality. Pups were born at normal weight but lost weight while nursing and did not gain it back.
- Meyer 1980 ★ When pregnant rats received 500 mg/kg BHT, it significantly affected body weight in both generations. There were developmental problems in the pups, starting during the lactation period.
- **Meyer** 2006 Injected BHT causes lung damage in mice, in which one sort of cell dies and another type replaces them. In this study, the authors show that this injury elicits an inflammatory response with elevated expression of enzymes in the eicosanoid pathway.
- Naveena 2008 Pomegranate juice or pomegranate rind powder extract can protect cooked chicken patties against oxidative rancidity longer than BHT.
- NIH Twelfth Report on Carcinogens 2011 BHA is "reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity from studies in experimental animals." Note: The NIH has said this in every report for many years already, but it is still used freely in food.

- **Park** 1990 Fish larvae fed a diet with BHA for 12 days had hepatic (*liver*) tumors 6 months later. Results were clearly dose-dependent and show that BHA is carcinogenic in fish even at the low 0.01% dose.
- Safer 1999 Rats fed BHT had increased liver weight. Under an electron microscope, the liver cells showed gradual vacuolization (*holes*) and disintegration, a "moth-eaten" appearance, withered mitochondria (*mitochondria control cell energy*), and necrosis (*death*).
- Sarafian 2002 Marijuana smoke and BHA together are far more harmful to the lungs than either one alone.
- Sasaki 2002 Both BHA and BHT cause DNA damage in the stomach, colon and bladder.
- Siman 1996 Like tobacco and many drugs, BHT is metabolized by *cytochrome P450* in the liver, where it becomes a harmful pro-oxidative compound instead of the anti-oxidant it is supposed to be.
- Soubra 2007 Children in Lebanon may be exceeding the acceptable daily intake for BHT and sulfites.
- Stokes 1974 ★ 0.5% BHA or BHT was fed to pregnant mice and their offspring. Compared to controls, BHAtreated pups explored more, and slept, groomed, and learned less. BHT-treated pups slept less, learned less, and were more aggressive.
- Stolze 1999 BHA and TBHQ together cause harm to erythrocyte (red blood cell) membrane structures.
- Takami 1999 ★ BHA, BHT and 3 other preservatives were shown to damage oocyte (*egg*) maturation in female rats. Antioxidants with no harm to oocyte maturation included ascorbic acid and vitamin E.
- Tanaka 1993 ★ BHT was fed to mice for three generations. Body weight of the pups was higher at birth and during lactation for each generation. Turning over and crawling uphill were affected.
- **Thompson** 1988 BHA interacts with BHT in the lungs of mice by stimulating formation of hydrogen peroxide, increasing BHT binding to protein and directly injuring the lung tissues.
- Thompson 1988 ★ BHT produces an increase in mouse lung weight by the necrosis (*death*) of cells in the lung walls. BHA alone has no effect on lung weight until added to a small amount of BHT.
- **Thompson** 1988 In rat liver mitochondria, BHA and BHT inhibit respiratory control of cells by stimulating state 4 respiration. They also affect the mitochondrial membrane, causing calcium release and mitochondrial swelling. There is a rapid decrease in ATP (*energy source*) levels and then cell death.
- **Thompson** 1989 Both BHA and phenolic compounds in medicine and foods stimulate BHT to become the more toxic BHT-quinone methide. *Note:* Salicylates, food dyes even neurotransmitters are phenolic.
- **Thompson** 1989 The addition of BHA enhanced the covalent binding of BHT by 400%, increased the formation of the polar and aqueous metabolites of BHT, and created two additional metabolites of BHT.
- **Tryphonas** 1999 0.5% BHT treatment resulted in a significant reduction in natural killer cell activity of splenocytes (cells in spleen that kill invaders). Note: This means BHT affects the immune system.
- **Umemura** 2006 BHT was successful as a **tumor promoter** with each of six test lung carcinogens. Thus, BHT is now a dependable way to test new chemicals to see if they are lung carcinogens.
- Verhagen 1990 The ADI (allowable daily intake) of BHT is exceeded by all age groups in the Netherlands.
- Yu 2000 The proposed use of BHA in cancer prevention is challenged by the observation that BHA has a toxic effect in animals, causing apoptosis (*cell death*) in freshly isolated rat hepatocytes (*liver cells*).
- **Zoccarato** 1987 In guinea pig cerebral cortex neurons, BHA and BHT strongly inhibit certain processes important to calcium ion depolarization of GABA and glutamate neuron transmission.

Research on Sweeteners

CORN SYRUP

Corn syrup and high fructose corn syrup are not eliminated on the Feingold Program. However, products containing them are marked in the Feingold Foodlist for those who wish to avoid them. Our experience has been that about 20% of our members are intolerant of corn syrup, although most of them can tolerate ordinary cane sugar with no problem. Those sensitive to sulfites should avoid corn syrup.

Bocarsly 2010 ★ HFCS accounts for 40% of caloric sweeteners in the US. Rats given access to HFCS ate the same number of total calories as rats given access to sucrose, but they gained significantly more body weight - especially abdominal fat - and had elevated triglyceride levels. The authors say HFCS may contribute to obesity.

Children who eat a lot of "sugar" are probably eating a lot of corn syrup. Some Feingold members report sensitivity to corn syrup.

Other names for corn syrup Dextrose, Glucose, are High Fructose Corn Syrup, Corn Sweetener, Maltodextrin, and Corn Syrup Solids. Efforts are being made to rename it "corn sugar."

- **Dufault** 2009 Samples of corn syrup were found to contain mercury from manufacturing plants at levels up to 0.57 micrograms mercury per gram of corn syrup. The authors say that mercury exposure alters neuronal function and increases oxidative stress. Some food color additives have also been shown to lead to deficiency in zinc, required for mercury elimination.
- Gaby 2005 Consumption of high-fructose corn syrup (HFCS) may now exceed that of sucrose. Although it does not hurt blood-sugar regulation in the short-term, HFCS has other effects on metabolism. It promotes the formation of toxic chemicals involved in aging, in diabetes complications, and in hardening of the arteries. It may be partly responsible for the increase in obesity, diabetes mellitus, and non-alcoholic fatty liver disease. The authors say that the evidence suggests it is more harmful than generally recognized.
- Hallfrisch 1990 When introduced in 1967, high fructose corn syrup (HFCS) was recommended for diabetic and obese people. Although it causes a smaller increase in blood glucose and insulin than sugar does, there are undesirable effects that show up later. Fructose, when consumed in excess of glucose, may be malabsorbed. HFCS turns into fat more easily and raises triglycerides and cholesterol more than ordinary sugar or other carbohydrates. It increases blood pressure, uric acid, and lactic acid.
 - Sugar is allowed on the Feingold Program. Sugar not labeled "cane sugar" is usually beet sugar. SUGAR Some people who seem to be sugar-intolerant may be reacting to chemicals used in the manufacture of refined sugar. Honey is usually well tolerated.
- Blunden 2011 Children with ADHD and sleep disturbance ate more carbohydrates, fats, and (especially) sugar. Note: It is unclear if additives in the sugary foods were considered, or if the "sugar" was sucrose or HFCS.

Inam 2006 **★** Serotonin is a neurotransmitter important in mood, stress, and attention. One group of rats was fed a standard diet, while another group was fed a standard diet with 25% table sugar for 5 weeks. The sugar induced a change in the serotonin receptor's ability to receive messages both before and after the synapse (space between neurons). Note: Don't eat that much sugar!

- Johnson 2011 Recent studies suggest ADHD is associated with a disruption in dopamine signaling in reward-related brain regions, as seen in food or drug addiction. Chronic excessive sugar intake may lead to alterations in mesolimbic dopamine signaling, contributing to ADHD symptoms.
- Wolraich 1994 ★ Children whose parents said they were "sugar-sensitive" were tested with a series of three diets - one with sugar, one with aspartame, and one with saccharin. Wolraich concluded that the three sweeteners could not all be "equally bad" because the children had improved continuously during the nine weeks of the study. Note: Since all three diets were without

The Wolraich study is often guoted to "prove" that parents are poor judges and that sugar is not harmful to behavior. However, the study did not test the synthetic dyes, artificial flavorings or preservatives found in foods like candies and soda. It did not even use the same kind of sweetener found in most of these foods, so the results are of little value.

artificial food colorings, flavorings, and preservatives, this improvement is not surprising. Also, most candy and soda contains CORN SYRUP - not table sugar - and this study did not test corn syrup.

ASPARTAME

Aspartame (Equal, NutraSweet, etc.) and the related chemicals Neotame, Alitame, and Advantame, were specifically excluded from the Feingold Program in 2004.

Butchko 2002 - "It is clear that aspartame is safe." Note: This study was done by the NutraSweet Company.

- Christian 2004 Rats getting aspartame in their drinking water took longer to run a T-Maze, indicating an effect on memory. Changes were seen in receptor densities in various parts of their brains, as well as enzymes.
- Kim 2011 Zebrafish were fed aspartame or saccharin with a high cholesterol diet. Those fed saccharin got high cholesterol; 30% of those fed aspartame died with "swimming defects" and inflamed brain and liver cells.
- Lau 2006 Food coloring + aspartame was found to be synergistic, i.e., far more toxic to developing neurites than expected by just adding up the effect of each additive given alone.
- Lowen 2011 Results from several large population studies suggest regular consumption of artificial sweeteners like aspartame and sucralose may actually contribute to rather than combat weight gain and diabetes.
- Maher 1987 If mice are given enough aspartame to elevate plasma phenylalanine levels more than tyrosine levels (which happens in humans too), seizures are more easily induced.
- Nakao 2003 Formaldehyde is a breakdown product of aspartame. In rat cells, 100 *microM* of it significantly increased the number of shrunken cells and cells with damaged DNA. Much higher concentrations have been measured in humans.
- Roberts 2001- Aspartame-induced disorders include headache, confusion, convulsions, irritability, depression, intellectual deterioration, antisocial behavior, rashes, asthma and unstable diabetes, as well as actual addiction to aspartame-containing products.

A senior FDA toxicologist, the late Dr. Adrian Gross, tried to prevent the approval of aspartame. He told Congress that they were violating the Delaney Amendment since aspartame can trigger brain tumors. He said, "If the FDA violates its own laws, who is left to protect the public?"

OTHER ARTIFICIAL **SWEETENERS**

As "artificial flavorings," artificial sweeteners are not acceptable on the Feingold Program. Specifically, no products containing sucralose (Splenda) or saccharine (Sweet 'N Low) are listed in the Feingold Foodlist books.

Sucralose (Splenda) pretends to be natural. It is made from sugar by replacing certain parts of the sugar molecule with chlorine. It thus becomes a chlorocarbon whose chemical structure is closer to a pesticide than a sugar. It is often bulked up with puffed corn syrup solids, with *almost* 5 calories per teaspoon (legally called "zero").

Feingold members who had used Splenda were asked for feedback, and reported racing heart, stomach ache, head banging, crying, asthma attack, depression, yeast infection, and memory loss. It is not known whether these effects were caused by the sucralose itself or the corn syrup component.

Other noncaloric sweeteners include Tagatose (from milk), Trehalose (from starch), Acesulfame potassium, Neohesperidine DC (from oranges) and others from plants. Their safety for people on the Feingold Program is not known, and no products containing them are in the *Feingold Foodlists*.

AGAVE

Agave syrup is made from the agave plant and is a natural sweetener. It is acceptable on the Feingold Program.

STEVIA

Stevia is an herb that has been used as a no-calorie sweetener in Japan and Brazil for over 20 years. Studies show it can also lower blood pressure, improve blood sugar control, and increase insulin sensitivity (Chang 2005, Hsieh 2003). Stevia is acceptable on the Feingold Program. Some brands contain unacceptable additives, however.

ALCOHOL SUGARS

Alcohol sugars are allowed on the Feingold Program. Care should be taken not to overdo them, since they have a laxative effect. When a sugar name ends in "ol" that means it is an alcohol sugar. Commonly used alcohol sugars are Sorbitol, Mannitol, <u>*Xylitol, Polyols (from hydrogenated starch hydrolysates).*</u>

ADHD and Autism Research

- Bateman 2004 ★ In a large group of normal toddlers, 20 mg of coloring with benzoate caused adverse effects detectable by parents. The authors suggest removing these additives from the diet of all children.
- Bennett 1997 A survey determined that 75% of young criminals, but only 18% of non-offenders, are physically ill with allergy and nutritional problems.
- **Bennett** 1998 When treated for food intolerance, allergy, and mineral imbalance, 9 child criminals improved physically and psychologically. 7 of them continued the diet and continued to improve.
- Boris 1994 ★ 73% of 26 children with ADHD improved on an elimination diet. In a double-blind test with 100 mg dye and suspect foods, ALL reacted. "Dietary factors may play a significant role in the etiology of the majority of children with ADHD."
- Brenner 1977 Intending to prove Dr. Feingold wrong, Brenner offered the diet to 32 families whose children had <u>not</u> improved on medication. On the diet, 11 (34%) "were markedly improved … the startling changes seen in patients who had been followed for years with other forms of therapy suggest strongly that this improvement was genuine."

Risk-Benefit Analysis

By Philip Handler, President* National Academy of Sciences

"A sensible guide would surely be to reduce exposure to hazard whenever possible, to accept substantial hazard only for great benefit, minor hazard for modest benefit, and no hazard at all when the benefit seems relatively trivial."

The manufacturer benefits from the use of inexpensive synthetic coloring; the consumer bears all the risk, with no benefit whatso-ever.

*Handler served two terms, 1969-81 and received the National Medal of Science.

- **Brenner** 1979 20 children who responded to the Feingold Diet, and 14 who did not, were tested for zinc and copper levels in their blood. Children who responded to the diet had high copper levels in their blood. (*See the Ward studies, page 37.*)
- Cade 2000 High IgG antibodies to gluten were found in 87% of autistic and 86% of schizophrenic patients. IgG antibodies to casein were found in 90% of autistic and 93% of schizophrenics. A gluten-casein free diet was accompanied by improvement in 81% of autistic children. This supports the proposal that both disorders are due to absorption of morphine-like chemicals formed in the intestine from digestion of gluten and casein.
- Carter 1993 ★ 75.6% of 78 hyperactive children improved on an open trial of an elimination diet. This was verified in a placebo-controlled double-blind challenge protocol.
- **Conners** 1976 ★ Using a Feingold Diet and a control diet on 15 hyperactive children, both parents and teachers reported improvement on the K-P (*Feingold*) Diet.
- **Dengate** 2002 ★ Calcium propionate (*a preservative used in bread*) caused irritability, restlessness, inattention and sleep disturbance in children who had improved on an additive-free diet. *Note: Products containing this preservative are marked in the Feingold Foodlists*.
- **Dumbrell** 1978 The nutritional quality of the Feingold Diet is superior to that of the normal diet.
- Egger 1983 ★ 93% of 88 children with frequent migraine recovered on a "few foods" additive-free diet. Other symptoms which improved: abdominal pain, behavior disorder, seizures, asthma, and eczema.
- Egger 1985 81.6% of 76 overactive children improved on a "few-foods" additive-free diet. Headache, abdominal pain, and seizures improved, too.

The oligoantigenic *(few foods)* diet eliminates all additives and many foods. It is useful for a short trial for diagnosis but is not a satisfactory long-term diet.

Egger 1989 \star 80% of 45 children with epilepsy and physical or behavioral problems

recovered or improved on a "few foods" diet. Headache, abdominal pain, and hyperactivity stopped in all those whose seizures stopped, and in some whose seizures did not. Symptoms returned in **94**% of children challenged with the foods and additives.

- Egger 1992 ★ On a diet avoiding additives, 76% of 21 children whose migraine or hyperactive behavior had improved also stopped bed wetting.
- Fitzsimon 1978 ★ Children 6-14 years old who had improved on the Feingold Diet were given 40 mg of acetylsalicylic acid or placebo. Significance was reached in tests of general cognitive capacity, line walking and the "finger-to-nose" tests, as well as increased disturbance in sleep patterns in the children when given the salicylate.

Note: 40 mg is only half of a baby aspirin.

- Goyette 1978 ★ Performance on a visual-motor tracking task was impaired after a challenge of artificial colors. The author said, "Artificial food dyes do indeed impair and disrupt the behavior of the children."
- **Gross** 1987 36 children at a summer camp were put on a Feingold-type diet for **one week** and then on an additivecontaining diet for one week. Gross concluded the Feingold Diet has no merit, conceding that the camp director and teachers all felt the children were noisier during the additive-rich week.

Note: All but one of the hyperactive children remained on their (colored) behavior-modifying medication during the study. Most children need more than one week to respond to a diet change. Two children were sent home during the "additive-rich" week; one was the only ADHD child not on medication, and the other child's ADHD medication was suddenly "not strong enough" when additives were present. See more about this study at <u>www.feingold.org/how-to-design-a-really-bad-study.html</u>

- Harding 2003 ★ Food supplement treatment of ADHD was of equal efficacy to Ritalin treatment. The author suggests 8 risk factors for ADHD: food and additive allergies, heavy metal toxicity and other environmental toxins, low-protein/high-carbohydrate diets, mineral imbalances, essential fatty acid and phospholipid deficiencies, amino acid deficiencies, thyroid disorders, and B-vitamin deficiencies.
- Hamazak 2002 ★ DHA (*in fish oil*) controls aggression in young people under stress, and this study was designed to see if it is useful for elderly people. The ordinary DHA intake in food (150 mg per day) was not enough, but getting an extra 1500 mg of DHA a day significantly decreased aggressiveness in older university employees, while the placebo did not. *Note: Fish oil is not part of the basic Feingold Program, but much research has shown it to be a helpful addition to everyone's diet. 1500 mg DHA is 1.5 grams or 10 to 15 capsules of fish oil, depending on the brand.*
- Harley 1978 ★ 10 hyperactive preschool children were tested with two diets, not knowing which was the Feingold Diet. 100% of them improved on the Feingold Diet. Harley admits he was "not in a position to refute his [*Feingold's*] claims regarding the possible causative effect played by artificial

food colors in preschool children." *Note: This study was funded by the food and additive industry organization called the "Nutrition Foundation.*"

Harley 1978 ★ 36 school-age boys were tested with two diets after stopping medication. Only 22 of them had normal EEGs, while 14 had various neurological problems besides ADHD. 13 children improved on the Feingold Diet, 12 of them (92%) in the group with the control (additive-containing) diet first. In this study, Harley trusted product ingredient labels, not Feingold materials, and did not eliminate preservatives.

100% of the preschoolers improved on the Feingold Diet in this early double-blind study.

The "control" diet itself had few additives (less than 27 mg/day). Since children who are recently off stimulant meds may take longer to respond to diet change, it is not surprising that 92% of the responding children were in the group trying the Feingold Diet *after* the control diet.

But Harley did not "get" it, writing, "Whatever the reason, the fact that the experimental diet seems to 'work' only when a control diet is given first would appear to attenuate the claimed efficacy of the experimental diet." Thus, he concluded the diet "doesn't work" in spite of his own results.

- Harper 1978 On the "hyperkinesis diet," nutrient intakes of 54 hyperactive children "compared favorably with the Recommended Dietary Allowances."
- **Howard** 2010 Following 2,868 children from birth through 14 years old, the researchers determined that those on a "Western" diet were more likely to have an ADHD diagnosis.
- **Husain** 2006 A dietary record of 3,141 children in Kuwait indicated that they exceeded the ADI (*acceptable daily intake*) of 4 of the 9 permitted colors by factors of 2 to 8. The authors call for studies into potential adverse health effects associated with the high intake of these artificial colors.

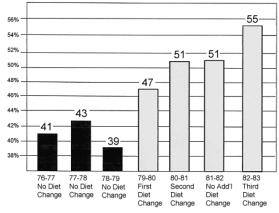
- **Kamel** 2011 This study in rats concluded there is sufficient scientific evidence of a true causal link between tartrazine (Yellow 5) and hyperactivity, anxiety and depression-like behaviors. The authors warn of the hazardous impact of tartrazine on public health.
- Kaplan 1989 Children with ADHD do not eat differently; therefore, their nutrition-behavior interactions are more likely to be a function of idiosyncratic sensitivities.
- Kaplan 1989a ★ In a 10-week diet study, more than half the children improved in behavior, with negligible placebo effects. Other symptoms improved: halitosis, night awakenings, and inability to sleep.
- Levy 1978 ★ Some mothers reported more symptoms after the challenge, but objective tests did not show significant deterioration. Tests were given the DAY AFTER the children ate cookies containing only ONE mg of Yellow 5.

A "challenge" of only 1 mg of food dye is absurdly small, and if any reaction had occurred, it would surely be gone by the next day.

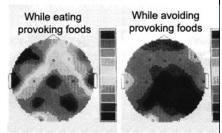
- Lien 2006 Children drinking 4 or more glasses of sugar-containing soft drinks per day had mental health problems and hyperactivity. *Note: Soda usually contains corn syrup, synthetic coloring, flavoring, and preservatives. This study shows a correlation between soda and mental health, but cannot implicate sugar alone.*
- Mattes 1981 ★ Calling it a "high dose," Mattes gave cookies with only 13 mg of food dye to children on the Feingold Diet. He concluded there was "no evidence of a food coloring effect." Note: Children on the diet for years can handle an occasional small exposure to food additives without obvious effect.
- McCann 2007 ★ "Artificial colours or a sodium benzoate preservative (or both) in the diet result in increased hyperactivity in 3-year-old and 8/9-year-old children in the general population."
- Niederhofer 2006 132 patients with celiac disease were assessed for ADHD symptoms before and 6 months after starting a gluten-free diet. Their Conners' scores and most of their ADHD symptoms improved significantly on the diet. *Note:* Processed food products often contain gluten. Whether the improvement was from elimination of gluten or reduction of synthetic additives was not determined.
- Niederhofer 2011 10 of 67 patients with ADHD also had celiac disease and their ADHD symptoms improved on a gluten-free diet. The author suggests that celiac disease should be included in the ADHD symptom checklist.
- **Pelsser** 2002 **80.6%** of 31 children with ADHD who completed a 2-week trial on the "few foods" diet improved by at least 50% on both the Conners' Scale and the ADHD Rating Scale. The authors say, "In young children with ADHD, an elimination diet can lead to a statistically significant decrease in symptoms."
- Pelsser 2009 73% of children in the diet group decreased their symptom scores by 50% or more, vs. NONE of the control group. Oppositional defiant disorder (ODD) symptoms also improved significantly in the diet group. Note: The diet eliminated milk, wheat, eggs, chicken, and beef as well as additives.
- **Pelsser** 2011 The difference in ADHD ratings between the diet group and control group was significant. The author says an elimination diet "is a valuable instrument to assess whether ADHD is induced by food." IgG blood test scores, however, did not predict which foods would trigger relapse.
- **Pollock** 1990 ★ Artificial food colors had "an adverse effect" on the Conners' behavior rating of 19 children.
- **Rowe** 1988 **★** 72.7% of 55 children on a 6-week trial of the Feingold Diet demonstrated improved behavior.
- Rowe 1994 75% of 200 children put on the Feingold Diet measurably improved.
- **Rowe** 1994 ★ Following the previous study, 54 children were put on an additive-free diet and then challenged with 6 different doses of Yellow 5. 82.6% of 23 "suspected reactors" and even 10% of the 20 "control" children reacted. Length and severity of reactions were dose-dependent.

- Salamy 1982 ★ When given drinks with food additives, all the children showed changes in EEG and heart rate. Hyperactive children were more affected than normal children.
- Salzman 1976 93% of 15 children put on the Feingold Diet improved in overactivity, distractibility, impulsiveness and excitability. Sleep and bed-wetting problems were partly or completely resolved.
- Schmidt 1997 ★ The children with conduct disorder who responded to dietary treatment did just as well as those who responded to medication.
- Schoenthaler 1986 Over 4 years, a school breakfast and lunch program with less sucrose and fewer additives was implemented in 803 NYC public schools (a MILLION children). As changes approached the Feingold Diet, test score averages on national tests rose. From start to finish, there was a 15.7% increase in mean academic percentile rating.

Moreover, 12.4% of the one million children were more than two years below grade level before the change. Afterwards, only 4.9% of them were more than two years below grade level.



- Schoenthaler 1991 Improving the diet in 813 state facilities (*jails*) resulted in "significantly improved conduct, intelligence, and/or academic performance..."
- Swanson 1980 ★ After a few days on an additive-free diet, 40 children were given 100-150 mg of mixed food dyes or placebo. The learning ability of the 20 hyperactive children (but NOT the 20 normal controls) was worse after the food dyes. *Note:* Swanson was criticized for using "too much" dye. Critics said it was a "toxic reaction." However, 100 mg of dye is easily reached by anybody eating a few pieces of colored candy, a cupcake with bright colored frosting, some Jello, or a few cups of red soda. If 100 mg is "toxic," then why is it so freely allowed in the supermarket, while its use by researchers is restricted? While eating While eating
- Uhlig 1997 ★ This is the first study to show an association between brain electrical activity and the intake of provoking foods in children with food-induced ADHD. *Beta*-1 activity in the fronto-temporal areas of the brain was increased (in the one on the left).
- Ward 1990 ★ Yellow 5 caused a reduction in serum and saliva zinc and an increase in urinary zinc with a corresponding deterioration in behavior and emotional responses in ADHD children but not in the normal children.
- Ward 1997 ★ In hyperactive children, Yellow 5 and 6 caused a reduction in zinc, resulting in one or more of the following symptoms: overactivity, aggression, violence, poor speech, poor coordination, asthma, eczema. Compared to controls, these children were low in zinc and iron but high in aluminum, cadmium and lead.
- Weiss 1980 ★ Using 35.26 mg dye on children who were *not* hyperactive, he concluded: "Modest doses of synthetic colors, and perhaps other agents excluded by elimination diets, can provoke disturbed behavior in children."
- Williams 1978 ★ Although both placebo and medication pills contained coloring, and his "modified" diet did not exclude salicylates, Williams nevertheless showed that drugs-plus-diet works better than drugs alone, by both parent and teacher ratings. In fact, 7 of the 26 children were diagnosed as "no longer hyperactive."



Bio-Markers - Biochemical Differences in ADHD

It has often been said that ADHD is a disease with no biological marker, i.e., there is no blood or urine or other physical test to identify it. Some even claim it does not exist because it cannot be measured. This is like saying that a headache does not exist – after all, a headache, too, is subjective and cannot be measured.

However, research has revealed there **ARE** biochemical differences in children with ADHD. We wonder why these differences continue to be ignored. To develop accurate medical testing, and to better understand the condition, we need more studies like these:

- Alberti 1999 ★ Autistic children showed a low level of sulfation, indicating difficulty in detoxifying or metabolizing certain compounds.
- Brenner 1979 ★ 20 children who responded to the Feingold Diet, and 14 who did not, were tested for zinc and copper levels in their blood. Responders had a higher level of copper.
- Oades 1998 ★ Over two days, children with ADHD drank four times as much water and showed twice the levels of neuropeptide Y (NPY) as healthy children. Urinary excretion of norepinephrine and a serotonin metabolite were markedly increased in children with ADHD, while excretion rates for sodium, phosphate and calcium were decreased. In spite of drinking more water, children in the ADHD group produced less urine. Oades writes, "Increases of drinking and circulating NPY in ADHD children and decreased electrolyte excretion may reflect a common disturbance in metabolic homeostasis."
- **Kiddie** 2010 66% of children with ADHD were found to be deficient in zinc and 23% were deficient in copper, even though they were eating adequate amounts of both.
- Walsh 1997 An independent laboratory compared the blood copper/zinc ratio of assaultive males with other male patients with no history of violence, showing clearly a statistically abnormal zinc/copper ratio in violence-prone individuals. Note: This is not necessarily a biochemical marker, but more important it is something that can be diagnosed ... and remedied.
- Ward 1990 ★ Yellow 5 reduced zinc in blood and saliva, and increased it in urine of the ADHD children but not the controls. The zinc loss corresponded to deterioration in behavior and emotional responses.
- Ward 1997 ★ In hyperactive children, Yellows 5 and 6 significantly reduced zinc levels, causing one or more of the following symptoms: overactivity, aggression, violence, poor speech, poor coordination, asthma, eczema. Compared to controls, hyperactive children were low in zinc and iron but high in aluminum, cadmium and lead.
- Warrington 1986 In patients with chronic additive-induced urticaria, or aspirin-sensitive asthma, Yellow 5 caused significant LIF (*T cell-derived lymphokine leucocyte inhibitory factor*) release from mononuclear cells. These results "suggest a potential diagnostic test for this condition." Note: Asthma and skin problems frequently plague children with ADHD. When diet helps the ADHD, it usually helps the other conditions as well. It would be interesting to measure LIF release in ADHD.

Allergy: Asthma, Eczema / Urticaria

- Arai 1998 60% of 20 adult asthmatics reacted to metabisulfite with airway obstruction, urticaria, skin problems and nasal congestion. *Note: Products containing sulfite are noted in the Feingold Foodlists.*
- **Barnes** 1998 Asthma cannot be controlled even by high dose corticosteroids in 5% of patients. The author recommends looking for unrecognized allergens, occupational sensitizers, dietary additives, etc.
- Cant 1986 Changing the mother's diet helped 37 breast-fed infants with eczema.
- **Ceserani** 1978 Yellow 5 induces bronchoconstriction similar to that caused by aspirin and other nonsteroidal antiinflammatory drugs in some aspirin-sensitive people.

- **Devereux** 2006 Since 1960, asthma and allergic disease have become a major public-health concern. The mother's diet during pregnancy might be connected to her child's development of asthma.
- **Devereux** 2006 1,861 children were followed from conception to five years old. When a mother received enough vitamin E and zinc while pregnant, her child had a lower risk of getting asthma by age five.
- **Dodig** 2008 Complete or partial remission of urticaria was obtained with antihistamines and a **low salicylate**, **low preservative diet**. Three of the children also needed high-dose intravenous immunoglobulin.
- Egger 1983 \star 93% of 88 children with frequent migraine recovered on the "few foods" additive-free diet. Abdominal pain, behavior disorder, seizure, asthma, and eczema also improved.
- **Genton** 1985 In 20 of 34 patients with asthma or urticaria, a diet without additives or aspirin resulted in a "marked improvement of symptoms" within five days.
- **Gomez** 2006 Zinc deficiency affects enzymes, causing major changes in the lipid (*fats such as cholesterol*) composition of the lung. Therefore, zinc supplementation must be included in public health interventions and therapies for high-risk subjects or those with certain diseases, such as **asthma**.

Note: Brenner found that children with ADHD have zinc deficiency; Ward found that synthetic colorings cause children with ADHD to lose zinc; and Arnold found that children given zinc respond better to stimulant medications. Since both ADHD and asthma may be linked to zinc deficiency, it is not surprising that many children have this combination of disorders.

- Hong 1989 ★ In 42.7% of 36 patients on medication given blind provocation tests, aspirin and food additives overcame their medications, causing bronchoconstriction, angioedema, or urticaria.
- **Huang** 1998 62 children who developed a rash after being given antibiotic were given the same antibiotic but without dyes at the next infection. Only 8 of them developed a skin rash, and it was mild.
- Jimenez-Aranda 1996 Yellow 5 was the most reactive additive tested in 40 patients with chronic urticaria (hives).
- Juhlin 1981 ★ In 330 patients with recurrent urticaria (*hives*), a questionnaire revealed a common history of allergy, asthma, and abdominal problems. Provocation tests with various food additives revealed one or more positive reactions in one-third of the patients.
- **Juhlin** 1987 In chronic urticaria (*hives*), the author suggests looking for adverse reactions to food additives.
- Kalinke 1999 ★ A 58-year-old patient had progressive pigmented purpura, a brown pigmentation of the skin caused by leaky blood vessels near the surface of the skin, from the legs upward. Controlled oral provocation testing showed that Yellow 5 triggered flareups. This patient was followed for over 20 years.
- Litonjua 2006 In 1,290 pregnant women, higher intake of vitamin E and zinc was related to less wheezing in their children at two years. The authors recommend more antioxidants during pregnancy to decrease wheezing risk for the baby.
- Lockey 1977 He created an allergy diet for urticaria and asthma at the Mayo Clinic. *This diet was used and further refined by Dr. Feingold as the K-P Diet. It was later commonly called the Feingold Diet.*
- Longo 1987 In 87.8% of 82 patients with asthma who were put on an additive-free "few-foods" diet, their eosinophil count went down, and improvement of symptoms followed.
- **Magerl** 2010 "Pseudoallergens" include food additives and natural substances in fruits, vegetables and spices that induce reactions similar to true allergic reactions. A pseudoallergen-free diet is beneficial for one in three patients with urticaria (*hives*).
- **Pachor** 1989 An adult with Melkersson-Rosenthal syndrome experienced intolerance to the food additives sodium benzoate and Yellow 5, with swelling of the face, hypertrophy of the gums, etc. All symptoms went into remission once the food additives were excluded from the diet.

- **Ring** 2001 "Pseudoallergic" reactions can be caused by low-molecular-mass chemicals (i.e., preservatives, colorings, etc.). Allergic contact eczema can be caused by artificial flavorings such as vanillin.
- Sakakibara 1995 Aspirin-induced asthma affects 9.8% of asthmatic adults. They may also have chronic sinusitis, nasal polyps, and inability to smell. Some medications make it worse, and some patients are sensitive to Yellow 5, sodium benzoate, parabens etc. *Note: Some asthma drugs actually contain these additives*.
- Sloper 1991 ★ 74% of 66 patients with eczema improved on a diet eliminating colors, preservatives, milk, eggs, and tomatoes. The authors say, "This diet may be considered in all children with moderate or severe eczema."
- Van Bever 1989 ★ After testing 25 children with severe atopic dermatitis, the authors found that some foods, food additives, tyramine, and acetylsalicylic acid can cause skin, intestinal, and respiratory reactions.
- Veien 1991 A patient suffered severe leukocytoclastic vasculitis (*blood vessel inflammation*) after eating 50 mg of Red 4 (*E124 in Europe*). It faded after 2 months on a diet without food additives.
- Ward 1997 ★ In hyperactive children, Yellows 5 and 6 significantly lowered zinc levels, causing one or more of the following: overactivity, aggression, violence, poor speech, poor coordination, asthma, eczema.
- **Warrington** 1986 In chronic additive-induced urticaria or aspirin-sensitive asthma, Yellow 5, sodium benzoate or salicylates cause a measurable cell-mediated immune response.
- **Worm** 2001 In people with atopic dermatitis and food intolerance, additives (*Yellow 5, benzoate, nitrite, etc.*) cause white blood cells to make more leukotriene, contributing to allergic reactions and asthma.
- Wuthrich 1981 ★ In oral tests using aspirin and additives, 26.6% of 620 patients with urticaria, asthma, or chronic rhinitis were intolerant, and 2/3 of them improved on an elimination diet.
- **Yoneyama** 2000 On a Japanese island, researchers were able to study an entire population of children under 4 years old, of which only half had been vaccinated with the DPT vaccine. Vaccinated children had **10 times** more asthma than those who were not vaccinated. Very few studies compare vaccinated with unvaccinated children.

	Vaccinated Children	Unvaccinated Children
Asthma	25.6%	2.3%
Atopic Dermatitis	18.0%	2.3%
Asthma, Rhinitis, or Dermatitis	56.4%	9.3%

Note: The DPT vaccine is no longer being used, having been replaced by the acellular DaPT vaccine which is theoretically safer. Since parents of children who become more prone to allergic, autoimmune, or behavioral disorders after vaccination often report benefit from the low-phenolic Feingold Diet, we suspect that the damage may be somewhere in the sulfation system. Research along these lines, and research into identifying those children at risk, is needed. To our knowledge, no such research is being done.

Physical Problems: Migraine, Seizures, Earache, Etc.

- Antico 1989 **IBS:** Comparing diet and other therapies, the authors conclude that food additive intolerance may be a major factor in the pathogenesis of Irritable Bowel Syndrome.
- Egger 1983 ★ MIGRAINE, GI PAIN, SEIZURES: 93% of 88 children with frequent migraine recovered on the "few foods" diet. Abdominal pain, behavior disorder, seizures, asthma, and eczema also improved.
- Egger 1985 HEADACHE, GI PAIN, SEIZURES: 81.6% of 76 overactive children improved on a few-foods diet without additives. Their headaches, abdominal pain, and seizures also improved.

- Egger 1989 ★ HEADACHE, GI PAIN, SEIZURES: 80% of 45 children with epilepsy plus headaches, abdominal symptoms, or hyperactivity improved on an elimination diet. Symptoms improved even in some whose seizures did not stop. In double-blind challenge, symptoms returned in 15 of 16 children.
- Egger 1992 ★ MIGRAINE, ENURESIS: On a diet without additives, 76% of 21 children whose migraine or hyperactive behavior had improved also stopped bedwetting.
- **Faulkner-Hogg** 1999 **CELIAC:** 22 patients switched from *almost* to *totally* gluten-free; 5 became well and 10 improved. Diarrhea, headache, nausea, and flatulence were provoked by amine, salicylate and soy, as well as gluten. *Note: Amines are addressed by the Failsafe Diet. See* <u>www.fedup.com.au</u>
- **Feingold** 1979 **EYE PROBLEMS:** Eye muscle disorders may respond to the Feingold Diet. Benzoates should also be eliminated. Dr. Feingold said a variety of neurologic and neuromuscular disturbances "may be induced by identical chemicals, depending upon the individual's genetic profile and the interaction with other environmental factors."
- McFadden 1996 NEUROLOGICAL DISORDERS: People with chronic degenerative neurological and immunological disease, including ADHD, autism, Alzheimer's, Parkinson's, and rheumatoid arthritis may have impaired sulfation. He concludes that this "may be a factor in the success of the Feingold Diet."
- Murphy 2006 ★ SEIZURES: The Ketogenic Diet used for epilepsy improves symptoms of ADHD in people with both disorders. Hyperactive rats put on the Ketogenic Diet improved in 24 hours. Note: Any diet removing most "processed" foods approaches the Feingold Diet by eliminating the many additives used in processed foods. Research on whether the nerve-protecting effect of the high-fat Ketogenic Diet counteracts the neuron-damaging effect of additives should be done. See Lau 2006.
- Neuman 1978 ★ ALLERGY: This was a randomized, controlled, clinical trial. 122 patients with allergies ate 50 mg Yellow 5 or placebo. Their reactions included general weakness, heatwaves, heart palpitations, blurred vision, rhinorrhoea (*runny nose*), feeling of suffocation, pruritus (*itching*), and urticaria (*hives*). There was activation of the fibrinolytic pathway.
- Newman 2001 MIGRAINE: Two patients with aspartame-triggered migraine were given migraine medication containing aspartame, and it made them worse.
- Nsouli 1994 ★ EARACHE: An additive-free diet prevented recurrence of earache in 70 (86%) of 81 patients. A challenge diet with the suspected food(s) produced earache in 66 of the 70 patients (94%). Nsouli said, "Food allergy should be considered in all pediatric patients with recurrent serous otitis media …"
- **Pelsser** 2010 **SLEEP:** A group of 27 children with ADHD were assigned to either a diet or a control group. The following symptoms were significantly reduced in the diet group: headaches, stomach aches, thirst, perspiration, and sleep problems.
- Petitpierre 1985 ★ IBS: 14 patients with irritable bowel syndrome got better on an elimination diet. Doubleblind challenges with foods or additives caused IBS symptoms. Elevated yeasts (Candida *albicans*, Geotrichum *candidum*) were also important, favoring the development of allergic and pseudo-allergic reactions.
- **Robson** 1997 ★ **ENURESIS:** Children with ADHD were 2.7 times more likely to wet their bed, and 4.5 times more likely to wet their pants, than children without ADHD. *Note: Feingold members find that when ADHD improves on the diet, the enuresis usually does, too.*
- Salzman 1976 SLEEP & ENURESIS: 93% of 15 children given the Feingold Diet improved in overactivity, distractibility, impulsiveness and excitability. Sleep and enuresis (*bedwetting*) problems were resolved partially or completely.

PST / Sulfation Pathways

- Alberti 1999 ★ Autistic children showed a low level of sulfation, indicating difficulty in detoxifying or metabolizing certain compounds.
- Bamforth 1993 ★ In test-tube studies, Yellow 5 and the flavoring vanillin can inhibit the enzyme dopamine sulfotransferase. Vanillin also inhibits the metabolism of a birth control medication which is sulfated in the liver.
- Harris 1996 Dietary factors play an important part in the sulfation detoxification pathway.
- **Gibb** 1987 Several synthetic food dyes were found to be potent inhibitors of the enzyme phenol sulfotransferase P (PST). If this occurred *in vivo*, potentially toxic concentrations of some phenolic compounds might result.
- Harris 1998 Low dose salicylic acid (*aspirin*) consistently and selectively inhibits the enzyme phenol sulfotransferase (PST). *Note: Thus, if a child is low in this enzyme, salicylate would make it worse.*
- McFadden 1996 People with environmental intolerance or chronic disease may have impaired sulfation. He concludes that this "may be a factor in the success of the Feingold Diet."
- Scadding 1988 78% of 74 people with food sensitivity were "poor sulfoxidisers," having trouble with sulfur and carbon oxidation reactions. A metabolic defect is suspected.
- **Waring** 2008 Phthalates and phenols are dietary and environmental endocrine disruptors, inhibiting sulfotransferase pathways.

Animal Research – Additives, Behavior and Neurology

- Abd El-Wahab 2012 In 10 groups of rats, 3 food dyes and 3 synthetic flavorings were tested singly and in combinations of a dye plus a flavoring. All treated groups ate more than controls but lost weight. They had reduced hemoglobin and red blood cell counts, and enzyme changes indicated liver and kidney damage.
- Amin 2010 Yellow 5 and carmoisine (*E-122, not used in US*) alter biochemical markers in rat livers and kidneys at low doses with a dose-effect.
- **Dalal** 2009 Red 3 may reduce serotonergic activity with modulation of central dopaminergic activity depending on the brain region.
- **Gao** 2011 Mice and rats on tartrazine (Yellow 5) were more active than those in the control group, but took longer to escape a maze and had memory loss. Their brains showed oxidative damage.
- **Kamel** 2011 The authors say this study in rats shows that a causal link truly exists between Yellow 5 and hyperactivity, anxiety and depression-like behaviors in rats and points to the hazardous impact of tartrazine on public health.
- Mehedi 2009 Mice fed Yellow 5 in their water had fewer sperm and more sperm abnormalities.
- **Meyer** 1980 \star When BHT was given to pregnant rats, pups had developmental problems.
- Mpountoukas 2010 Red 2, Red 3, and Yellow 5 bind directly to DNA, causing its degradation.
- Peng 2009 Blue 1 prevents paralysis when used on spinal cord injury. Note: Dr. Feingold called it a drug in 1973!
- **Ruppert** 1985 ★ A single exposure to the metals cadmium or tin produced hyperactivity in the rat pups. The authors conclude these metals are neurotoxic to the developing nervous system.
- Shimada 2010 Azo dyes cause colon-specific DNA damage in mice but not in rats, which seem insensitive.

- Stokes 1974 ★ BHA and BHT were fed to pregnant mice and their pups. BHA-fed pups explored more, but slept, groomed, and learned less. BHT-fed pups were aggressive, with learning difficulties.
- Tanaka 1993 ★ Red 2 was fed to mice. The pups weighed more, had trouble turning over, had trouble finding the source of an odor, and more died. Movement activity of male pups was affected.
- Tanaka 1993 ★ BHT was fed to mice for three generations. Body weight increased. Turning over and crawling uphill were abnormal in all treatment groups, but Tanaka concluded there is "little effect" on the mice.
- Tanaka 1996 ★ When Yellow 6 was fed to mice for two generations, the pups weighed more and had dose-related difficulty with surface righting (*turning over*), negative geotaxis (*crawling upwards*), and swimming.
- Tanaka 2001 ★ When Red 3 was fed to two generations of mice, movement and other changes in the pups were dose-related.
- Tanaka 2006 ★ When Yellow 5 was fed to mice, activity and body weight increased, and the timing of some developmental milestones changed. "Nevertheless," says Tanaka, "the actual dietary intake of tartrazine is unlikely to produce any adverse effects in humans." Note: He provides no supporting facts for this conclusion.
- Vorhees 1983 ★ When Red 40 was fed to rats for two generations, reproductive success, brain weight, female vaginal development, and survival were all reduced. Running wheel activity decreased, and rearing *(standing up)* increased. The authors say "Red 40 produced physical and behavioral toxicity in developing rats."

How to Design a Really Bad Study

All research studies have flaws, and those who don't like the results of a study can always pick holes in its design or methodology somewhere. However, some research papers are simply beyond belief BAD - yet they manage to get published in peer review journals and are read and quoted by otherwise intelligent people. So, if it is your ambition to create a *Really Bad Study*, some of the following pointers should be helpful.

1. Use subjects without the disorder you are studying.

Gross (1987) used 36 children and averaged all their responses – but only 18 of them had ADHD.

2. Add your numbers up wrong.

This is useful when you don't really want to use the amounts of challenge material you promised to use. Adams (1981) added up this list of additive-containing foods for his challenge, totaling 26.3 mg. The real total is only 18.8 mg.

1 glass lemonade w/ Yellow 5	0.3 mg
1 cupcake with Red 3	3.0 mg
Additional Red 40	14.7 mg
White Frosting w/ Yellow 5	0.8 mg

3. Use too little "challenge" material.

Notice how much yellow coloring was added to the white frosting in the cupcake in #2 above? The placebo cupcake had white frosting, and they looked

alike. In other words, not enough coloring was used to actually change the color of the frosting – is this reasonable? The Levy (1978) study used only ONE mg of dye per cookie, for a total of only FIVE mg per day; Mattes (1981) used such a small amount of dye that the cookies with the dye were the same color as those that did not contain any dye; Rose (1978) used just ONE cookie per day containing 1.2 mg of Yellow 5 – not enough to change the color of the oatmeal cookie used for challenge and placebo. Astonishingly, Rose actually did get results, using two subjects who had been on the Feingold diet for a while.

4. Expose subjects to the eliminated additives during their "experimental diet" phase.

Gross (1987) did it by restricting the children's diet while turning a blind eye to additives in medications or toiletries; Williams (1978) gave the children medications (with coloring) and placebo medications (also with coloring) while "modifying" the Feingold diet by not bothering to remove salicylates. Pestana (2010) never took the subjects off food dyes at all, except for the one half-day each week they were tested with Yellow 5 or placebo.

Another way is to simply minimize the importance of diet infractions (Harley 1978 and others). Since each infraction may take a few days to overcome, two a week (depending on the child and the infraction) may totally negate any benefit of the diet.

5. Give subjects meds to interfere with the diet study; drop those whose meds don't interfere.

In the Gross (1987) study, all but one of the 18 children with ADHD were on stimulant meds the whole time. The one not on meds was sent home for bad behavior when dyes were introduced, and one other child also was sent home when his medication was suddenly "not strong enough" to prevent bad behavior when given food dyes in his food.

In the Pestana (2010) study, the subjects continued to take their bronchodilators and other asthma or urticaria medication which would have prevented any actual asthmatic or skin reaction to Yellow 5 from showing up. Devlin (1992) also studied urticaria reactions in children using meds, giving them 50 mg tartrazine but averaging their skin reactions over the next 7 days following each trial a good way to wipe out any short-term reaction.

Use challenge cookies so large that the kids can't 6. finish them.

Some parents complained that cookies with 13 mg of food dyes provided by the Nutrition Foundation were too big for the kids to finish. This also meant, of course, that the children never got any dye on an empty stomach, like they would have with candy, and they sometimes never got the total expected dose.

7. Don't let the food dye touch the subject's skin, mouth, tongue, or esophagus.

Kleinman (2011), in his design for future studies, suggests giving the food dye tests in capsules – a common practice in the past, too. Thus the dye is never in touch with the child's tongue, lips, mouth, or esophagus, where it would be absorbed directly into the blood. A capsule would not dissolve until it reaches the stomach, where it dissolves within minutes to hours, depending on its coating.

8. Conflicts of interest can be a useful impetus in designing a Really Bad Study.

Select doctors paid by a food dye company (e.g., Clydesdale) or by Coca-Cola (e.g., Kleinman) to design a study of food additives - then just relax. See http://feingold.org/enews/09-2011.html

9. Aspirin will cure a headache right now, or it doesn't work. Treat diet the same way.

Gross (1987) put the children on a Feingold-type diet for one week before deciding that diet didn't work. As all Feingold members know, it usually takes at least one week to *begin* to respond to the diet change.

Swanson (1980) put the children on a diet for only three days before giving them either food dye or placebo and a learning test. He also rated their behavior using the Conners scale. Not surprisingly, he saw no differences between the placebo and the dve group in behavior, in spite of a dramatic drop in scores of the children with ADHD on the learning test when exposed to food dyes.



Timing is crucial: if you try a fitness program for only two days, for example, you will have sore aching muscles and fatigue, thus "proving" that exercise is harmful. If you test the Feingold diet for one week, or three

days, or - as Pestana (2010) did - for half a day, you may see no results at all.

10. Consider food dye ONLY; ignore everything else.

There are thousands of chemicals - most of them under the heading of "artificial flavorings" which are also eliminated from the Feingold diet. But forget about all that. Forget the preservatives and the salicylates, too - if the one food dye chemical you choose to test doesn't happen to make the child hyper, claim the diet doesn't work, and you're done.

11.	0		Rimland.		absolutely	ignore
results of animal or <i>in-vitro</i> studies.		Some animal & lab studies since then:				

In 1983, Dr. Bernard Rimland published a paper in the Journal of Learning Disabili-Here is what he had to say about animal studies:

"Unlike school children. laboratory rats cannot trade a tuna sandwich for a Twinkie, or drink an illicit Kool-Aid on the way home from school. Thus animal studies can be quite revealing. Nevertheless, the reviews pay scant, if any, attention to animal studies, and to in vitro studies, of

additives. The study by Goldenring, Wool, Shaywitz, Batter, Cohen, Young and Teicher (1980) reported 163% more activity and 128% greater failures in avoidance learning in rat pups given small amounts of food dyes, as compared to controls. The dosage level was equated to the average US per capita intake for humans. Similarly, the many testtube studies showing food colorings to damage nerve tissue, such as those by Augustine and Levitan

Shimada 2010 ties, in which he commented Amin 2010 on the main reviews of the • Hashem 2010 Feingold Diet at that time. Moutinho 2007

- Lau 2006
- Murphy 2006

• Kamel 2011

Gao 2011

- Ashida 2000
- Aboel-Zahab 1997
- Aoshima 1997
- Dees 1997
- Reyes 1996
- Voorhees 1983
- Tanaka 1993. 1996, 2001, 2006

(1980) and Lafferman and Silbergeld (1979) are all but ignored by the reviewers.

"Neurons and neurotransmitters are the very stuff that brains and therefore learning and behavior are made of. Does anyone believe that the adverse effects of food dyes on neurotransmitters are irrelevant to a sensible evaluation of the Feingold diet? I hardly think so! Since our measures of children's impairment, consisting primarily of parent and teacher subjective ratings, are so notoriously weak and insensitive, we should emphasize, not ignore, laboratory studies of animals and nerve tissue."

Reviews of Research

Anthony 1999 – An elimination diet is effective in most cases; medication should be reserved for those who fail.

- Arnold 1999 In a report under contract for the 1998 NIH Conference on ADHD, Arnold identified 23 non-stimulant treatments. He said only dietary treatment has convincing double-blind evidence of efficacy.
- **Banergee** 2007 Although ADHD is genetic, several biological and environmental risk factors interact with the genes, including food additives, diet, lead, maternal smoking or alcohol, and low birth weight.
- Baumgaertel 1999 The science supports dietary management and trace element supplementation for ADHD.
- **Berdonces** 2001 Psychiatric medications have major risks. Additives, preservatives, dyes, etc. can make ADHD worse. He also discusses omega-3 oils, vitamins, minerals, and herbs.
- Breakey 1997 After reviewing the research literature, she concludes that "diet definitely affects some children."
- Curtis 2008 Nutritional and environmental factors play major roles in autism and ADHD.
- **Ghuman** 2008 Evidence based treatment for ADHD: Level A for methylphenidate and Level B for parent behavior training, child training, and additive-free diet. (Level B: "At least fair scientific evidence suggests that the benefits of the clinical service outweighs the potential risks. Clinicians should discuss the service with eligible patients.")
- **Jacobson** 1999 with 2008 update The Center for Science in the Public Interest (CSPI) recommends NIHsponsored research on additives, and an FDA ban on the use of synthetic dye in products for children.
- **Kavale** 1983 In a meta analysis of early studies in the 1970's on artificial colors only, they concluded that the diet has no significant benefit. *Unfortunately, this old analysis continues to be quoted by ADHD authorities.*
- Kellogg Report 1989 The authors say the brain abnormalities associated with learning and behavioral problems appear related to neurotransmitter precursor imbalances, vitamin and mineral deficiencies, and "the consumption of refined carbohydrates, toxic elements, additives, colorings, caffeine, and allergens."
- **Kidd** 2000 Major contributors to ADHD include food additives and foods, environmental chemicals, molds, and fungi, and exposure to neurodevelopmental toxins such as heavy metals and organohalide pollutants.
- Liu 2005 This paper reviews early biological risk factors for violence, including pregnancy/birth complications, fetal exposure to nicotine, alcohol, and drugs, low cholesterol, malnutrition, lead and manganese exposure, head injuries and brain dysfunction, low arousal, low serotonin, low cortisol, and high testosterone.
- Millichap 2012 This review promises twice, in the abstract, to emphasize recent research. However, almost all the recent research is omitted, including the most significant study on the topic the <u>McCann study</u> published in 2007. Referring mostly to 30-years old studies and information, he admits that a "healthy diet" is important for children with ADHD but specifically excludes the Feingold diet.
- Nigg 2012 In a meta analysis of studies on food dyes, the authors conclude 8% of children with ADHD may have symptoms related to synthetic food colors, and that a "restriction diet" benefits some children with ADHD.
- **Pellow** 2011 Highlights the latest research regarding complementary and alternative medicine (CAM) for ADHD: dietary modifications, nutritional supplementation, herbal medicine, and homeopathy.
- **Rimland** 1983 Invited by publishers to comment on the Mattes, Kavale & Forness reviews of the early Feingold diet studies, Rimland concludes: "GIGO = garbage in / garbage out." He specified:
 - 1) Most of the studies were nearly irrelevant because they studied only 10 dyes but the diet excluded over 3,000 other compounds used at that time (*over 12,000 today*).
 - 2) The dosage levels of the colorings tested were ridiculously small.
 - 3) They failed to consider the role of the subject's nutritional status.
 - 4) They failed to recognize and control relevant variables (*e.g., copper levels or fluorescent lights*)
 - 5) They came to arbitrary negative conclusions not supported by the actual data (e.g. Harley study)
 - 6) They paid inadequate attention to animal and *in vitro* studies.

- Schab 2004 In a meta-analysis of studies on artificial food dyes, the authors concluded the studies are consistent with evidence that "neurobehavioral toxicity may characterize a variety of widely distributed chemicals."
- Schnoll 2003 Food additives, refined sugars, food sensitivities/allergies, and fatty acid deficiencies are all linked to ADHD, and diet modification should be part of the treatment protocol.
- Schnyder 1999 Adverse reactions to foods may be caused by toxic, enzymatic, pharmacological, "pseudoallergic" or allergic mechanisms. Diagnosis can usually be based on the history and results of a diet.
- Sinn 2008 Current evidence supports indications of nutritional and dietary influences on behavior and learning, with the strongest support to date reported for omega-3s and behavioral food reactions.
- **Stevens** 2011 A trial elimination diet is appropriate for children who have not responded satisfactorily to conventional treatment or whose parents wish to pursue a dietary investigation.
- Weiss 1982 As a toxicologist, Dr. Weiss re-analyzed several of the early "negative" studies and concluded, "The Feingold hypothesis, in principle, is supported by experiments that meet scientific criteria of validity..." Note: In other words, even the early studies funded by the additive industry did actually support the Feingold Diet when analyzed properly.

Weiss 2012 – Examination of the FDA's decision on food dyes, and why this is an environmental health issue.

LIST OF CITATIONS

- Abd El-Wahab HM, Moram GS. 2012. Toxic Effects of Some Synthetic Food Colorants and/or Flavor Additives on Male Rats. *Toxicol. Ind. Health.* 2012 Feb. 8 epub ahead of print.
- 2. Abdel Aziz AH, et al. 1997. A Study on the Reproductive Toxicity of Erythrosine in Male Mice. Pharmacol Res. 1997 May 35(5):457-62.
- Aboel-Zahab H, et al. 1997. Physiological Effects of Some Synthetic Food Colouring Additives on Rats. Bollettino Chimico Farmaceutico. 1997 Nov;136(10):615-27.
- 4. Adams W, 1981. Lack of Behavioral Effects from Feingold Diet Violations. *Perceptual & Motor Skills*. 1981 Feb;52(1):307-13.
- 5. Alberti A, et al. 1999. Sulphation Deficit in "Low-Functioning" Autistic Children: A Pilot Study. *Biological Psychiatry* 1999 Aug 1;46(3):420-4.
- 6. Allen DH, et al. 1984. Adverse Reactions to Foods. *Medical Journal of Australia* 1984, Sep 1; 141 (5 Suppl): S37-42.
- Amin KA et al. 2010. Effect of Food Azo Dyes Tartrazine and Carmoisine on Biochemical Parameters Related to Renal, Hepatic Function and Oxidative Stress Biomarkers in Young Male Rats. Food and Chemical Toxicology 2010 Oct;48(10):2994-9.
- Anthony HM, Maberly DJ, Birtwistle S. 1999. Attention Deficit Hyperactivity Disorder, Archives of Disease in Childhood 1999;81:189 (August).
- Antico A, Soana R, Clivio L, Baioni R., 1989. Irritable Colon Syndrome in Intolerance to Food Additives, Minerva Dietologica e Gastroenterologica. 1989 Oct-Dec;35(4):219-24.
- Aoshima H, Tenpaku Y, 1997. Modulation of GABA Receptors Expressed in Xenopus Oocytes by 13-L-Hydroxylinoleic Acid and Food Additives, *Bioscience, Biotechnology, & Biochemistry*. 1997 Dec;61(12):2051-7.
- Arai Y, et al. 1998. Food and Food Additives Hypersensitivity in Adult Asthmatics. III. Adverse Reaction to Sulfites in Adult Asthmatics, Arerugi 1998 Nov; 47 (11); pp.1163-7.
- 12. Arnold LE, 1999. Treatment Alternatives for Attention-Deficit/Hyperactivity Disorder (ADHD), Journal of Attention Disorders, Vol. 3 No. 1 (April 1999), 30-48.
- 13. Arnold LE et al., 2011. Zinc for Attention-Deficit/Hyperactivity Disorder: Placebo-Controlled Double-Blind Pilot Trial Alone and Combined with Amphetamine. *J Child Adolesc Psychopharmacol*. 2011 Feb;21(1):1-19.
- 14. Ashida H, et al. 2000. Synergistic Effects of Food Colors on the Toxicity of 3-amino-1,4-dimethyl-5H-pyrido[4,3-b]indole (Trp-P-1) in Primary Cultured Rat Hepatocytes, *Journal of Nutritional Science & Vitaminology* (Tokyo) 2000 Jun;46(3):130-6.
- 15. Augustine G, Levitan H, 1980. Neurotransmitter Release from a Vertebrate Neuromuscular Synapse Affected by a Food Dye, *Science Magazine*, March 28, 1980, Vol. 207, pp. 1489-90.
- Bailly D, 2006. Safety of Selective Serotonin Reuptake Inhibitor Antidepressants in Children and Adolescents, Presse Medicale, 2006, Oct;35(10 Pt 2):1507-15. Review. French.
- Bamforth KJ, et al. 1993. Common Food Additives are Potent Inhibitors of Human Liver 17 Alpha-ethinyloestradiol and Dopamine Sulphotransferases, *Biochemical Pharmacology*. 1993 Nov 17;46(10):1713-20.
- 18. Banergee TD et al. 2007. Environmental Risk Factors for Attention Deficit Hyperactivity Disorder. Acta Paediatr. 2007 Sep;96(9):1269-74
- 19. Barnes PJ & Woolcock AJ, 1998, Difficult Asthma, European Respiratory Journal. 1998 Nov; 12(5); pp.1209-18.
- Bateman B, et al., 2004. The Effects of a Double-Blind Placebo-Controlled Artificial Food Colourings and Benzoate Preservatives Challenge on Hyperactivity in a General Population Sample of Preschool Children, Arch of Disease in Childhood, 2004 Jun;89(6):506-11.
- 21. Baumgaertel A, 1999. Alternative and Controversial Treatments for Attention-Deficit/Hyperactivity Disorder, *Pediatric Clinics of North America.* 1999 Oct;46(5):977-92.
- 22. Bauer AK, et al., 2001. Butylated Hydroxytoluene (BHT) Induction of Pulmonary Inflammation: A Role in Tumor Promotion, *Experimental Lung Research.* 2001 Apr-May;27(3):197-216.
- 23. Bauer AK, et al. 2005. Toll-like Receptor 4 in Butylated Hydroxyltoluene-Induced Mouse Pulmonary Inflammation and Tumorigenesis, Journal of the National Cancer Institute. 2005 Dec 7;97(23):1778-81.
- 24. **Bennett** CPW, Brostoff J., 1997. The Health of Criminals Related to Behaviour, Food, Allergy and Nutrition: A Controlled Study of 100 Persistent Young Offenders, *Journal of Nutritional & Environmental Medicine*, Vol.7, No.4 Dec 1997 pp.359-366.
- 25. Bennett CPW, et al. 1998. The Shipley Project: Treating Food Allergy to Prevent Criminal Behaviour in Community Settings, *Journal of Nutritional & Environmental Medicine*, Vol.8, No.1,Mar.1998, pp.77-83.

- 26. Berdonces JL, 2001. Attention Deficit and Infantile Hyperactivity, Revista de Enfermeria 2001 Jan; 24 (1): 11-4.
- 27. Blass EM, 1996. Mothers and Their Infants: Peptide-Mediated Physiological, Behavioral and Affective Changes During Suckling. *Regulatory Peptides*, 1996 Oct 8;66(1-2):109-12.
- 28. Blunden SL, et al. 2011. Diet and Sleep in Children with Attention Deficit Hyperactivity Disorder: Preliminary Data in Australian Children. J. Child Health Care. 2011. Mar; 15(1):14-24.
- Bocarsly ME, et al. 2010. High-Fructose Corn Syrup Causes Characteristics of Obesity in Rats: Increased Body Weight, Body Fat and Triglyceride Levels. *Pharmacology, Biochemistry, and Behavior*. 2010 Nov; 97(1):101-6.
- Boris M, Mandel F. 1994. Foods and Additives are Common Causes of the Attention Deficit Hyperactive Disorder in Children, Annals of Allergy, May 1994, Vol. 72, pp. 462-8.
- Bouchard MF et al. 2010. Attention-Deficit/Hyperactivity Disorder and Urinary Metabolites of Organophosphate Pesticides, *Pediatrics*. 2010 Jun;125(6):e1270-7.
- 32. Breakey J, 1997. Review: The Role of Diet and Behaviour in Childhood, J of Paediatrics and Child Health, 1997, Jun; 33(3) pp.190-194.
- Brenner, A, 1977. A Study of the Efficacy of the Feingold Diet on Hyperkinetic Children. Some Favorable Personal Observations, *Clinical Pediatrics*, 1977, Jul; 16(7) pp.652-656.
- 34. **Brenner** A, 1979. Trace Mineral Levels in Hyperactive Children Responding to the Feingold Diet, *Journal of Pediatrics* 1979 Jun;94(6):944-5.
- 35. Brown RT, Sexson SB, 1989. Effects of Methylphenidate on Cardiovascular Responses in Attention Deficit Hyperactivity Disordered Adolescents, *Journal of Adolescent Health Care*. 1989 May;10(3):179-83.
- Butchko et al., 2002. Aspartame: Review of Safety, NutraSweet Company, *Regulatory Toxicology and Pharmacology* 2002 Apr;35(2 Pt 2):S1-93.
- 37. Cade R et al. 2000. Autism and Schizophrenia: Intestinal Disorders. *Nutritional Neuroscience*, March 2000.
- Cant AJ, Bailes JA, Marsden RA, Hewitt D, 1986. Effect of Maternal Dietary Exclusion on Breast Fed Infants with Eczema: Two Controlled Studies, *British Medical Journal* (Clin Res Ed) 1986 Jul 26; 293 (6541):231-3.
- 39. Carter CM, et al. 1993. Effects of a Few Foods Diet in Attention Deficit Disorder, Archives of Disease in Childhood, Nov. 1993; Vol.69(5): 564-8.
- Castner SA, Goldman-Rakic PS, 2003. Amphetamine Sensitization of Hallucinatory-Like Behaviors is Dependent on Prefrontal Cortex in Nonhuman Primates, *Biological Psychiatry*. 2003 Jul 15;54(2):105-10.
- 41. Ceserani R, et al. 1978. Tartrazine and Prostaglandin-System, Prostaglandins and Medicine. 1978 Dec;1(6):499-505.
- 42. Chang JC, 2005. Increase of Insulin Sensitivity by Stevioside in Fructose-Rich Chow-Fed Rats, *Hormone & Metabolic Research*, 2005 Oct;37(10):610-6.
- 43. Christian B, et al. 2004. Chronic Aspartame Affects T-Maze Performance, Brain Cholinergic Receptors and Na+, K+-ATPase in Rats. *Pharmacology, Biochemistry and Behavior.* 78(2004): 121-127.
- 44. **Cockell** KA, et al. 2004. Manganese Content of Soy or Rice Beverages is High in Comparison to Infant Formulas. *J of the American College of Nutrition*, 2004 Apr;23(2):124-30.
- 45. Conners CK, et al. 1976. Food Additives and Hyperkinesis: A Controlled Double-Blind Experiment. Pediatrics 1976 Aug;58(2):154-66.

46. Curtis LT, Patel K. 2008. Nutritional and Environmental Approaches to Preventing and Treating Autism and ADHD. *J.Altern.Complement.Med.* 2008. Jan-Feb; 14(1):79-85.

- Dalal A, Poddar MK 2009. Short-Term Erythrosine B-Inducted Inhibition of the Brain Regional Serotonergic Activity Suppresses Motor Activity (Exploratory Behavior) of Young Adult Mammals. *Biochemistry, and Behavior*, 2009 Jun;92(4):574-82.
- Dees C, et al. 1997. Estrogenic and DNA-Damaging Activity of Red No. 3 in Human Breast Cancer Cells. *Environ Health Perspect* 1997 Apr;105 Suppl 3:625-32
- Dengate S, Ruben A, 2002. Controlled Trial of Cumulative Behavioural Effects of a Common Bread Preservative, Journal of Paediatrics and Child Health. 2002 Aug;38(4):373-6.
- 50. Devereux G, 2006a. The Increase in the Prevalence of Asthma and Allergy: Food for Thought. *Nature Reviews, Immunology*, 2006 Nov;6(11):869-74.
- 51. Devereux G, et al, 2006b. Low Maternal Vitamin E Intake During Pregnancy is Associated With Asthma in 5-Year-Old Children, American Journal of Respiratory and Critical Care Medicine, 2006 Sep 1;174(5):499-507.
- 52. Devlin J, David TJ 1992. Tartrazine in Atopic Eczema. Archives of Disease in Childhood 1992. 67: 709-711.
- 53. Dodig S, Richter D 2008. Chronic Autoimmune Urticaria in Children. Acta Dermatovenerologica Croatica. 2008;16(2):65-71.
- D'Souza SJ, Biggs DF, 1987. Aspirin, Indomethacin, and Tartrazine Increase Carotid-Sinus-Nerve Activity and Arterial Blood Pressure in Guinea Pigs. *Pharmacology* 1987;34(2-3):96-103.
- Dufault R, et al. 2009. Mercury From Chlor-Alkali Plants: Measured Concentrations in Food Product Sugar. Environmental Health, 2009 Jan 26;8:2
- Dufault R, et al. 2009. Mercury Exposure, Nutritional Deficiencies and Metabolic Disruptions May Affect Learning in Children. Behavioral & Brain Functions, 2009 Oct 27;5:44
- 57. Dumbrell S, 1978. Is the Australian Version of the Feingold Diet Safe? Medical Journal of Australia. 1978 Dec 2;2(12):548, 569-70.
- 58. Egger J, et al. 1983. Is Migraine Food Allergy? A Double-Blind Controlled Trial of Oligoantigenic Diet Treatment. *The Lancet* 1983 Oct 15; 2(8355): 865-9.
- 59. Egger J, et al. 1985. Controlled Trial of Oligoantigenic Treatment in the Hyperkinetic Syndrome. The Lancet, March 9, 1985.
- 60. Egger J, et al. 1989. Oligoantigenic Diet Treatment of Children with Epilepsy and Migraine. Journal of Pediatrics 1989 Jan; 114(1): 51-8.
- 61. Egger J, et al. 1992. Effect of Diet Treatment on Enuresis in Children with Migraine or Hyperkinetic Behavior. *Clinical Pediatrics* (*Phila*) 1992 May;31(5):302-7.
- El-Saadany SS, 1991. Biochemical Effect of Chocolate Colouring and Flavouring Like Substances on Thyroid Function and Protein Biosynthesis. Nahrung 1991;35(4):335-43.
- 63. El-Zein RA, et al. 2005. Cytogenic Effects in Children Treated with Methylphenidate. Cancer Letters, 2005 Dec 18; 230(2):284-91.
- 64. Engin AB, et al. 2011. Effect of Butylated Hydroxytoluene (E321) Pretreatment Versus L-arginine on Liver Injury after Sub-Lethal Dose of Endotoxin Administration. *Environ. Toxicol. Pharmacol.* 2011 Nov; 32(3):457-64.
- 65. Eskenazi B, et al. 2007. Organophosphate Pesticide Exposure and Neurodevelopment in Young Mexican-American Children. Environmental Health Perspectives, 2007 May; 115(5): 792-8.
- 66. **Faulkner-Hogg** KB, et al. 1999. Dietary Analysis in Symptomatic Patients with Coeliac Disease on a Gluten-free Diet: The Role of Trace Amounts of Gluten and Non-Gluten Food Intolerances. *Scandinavian Journal of Gastroenterology*. 1999 Aug; 34(8):784-9.
- 67. Feingold BF, 1979. Dietary Management of Nystagmus. Journal of Neural Transmission, 1979, 45(2): 107-115.
- 68. Feingold BF, 1982. The Role of Diet in Behaviour. Ecology of Disease. 1982. 1(2-3) pp.153-65.
- 69. Findling RL, et al., 2011. Changes in Emotions Related to Medication Used to Treat ADHD. Part II: Clinical Approaches, *J of Attention Disorders*, Feb; 15(2):101-12.

- 70. Finley JW, 2004. Does Environmental Exposure to Manganese Pose a Health Risk to Healthy Adults? Nutr. Rev. 2004 Apr; 62(4): 148-53.
- Fisherman EW, Cohen G, 1973. Chemical Intolerance to BHA and BHT and Vascular Response as an Indicator and Monitor of Drug Intolerance, *Annals of Allergy*, 1973, Vol. 31, No. 3, pp. 126-133.
- 72. Fitzsimon M, et al. 1978. Salicylate Sensitivity in Children Reported to Respond to Salicylate Exclusion. *Medical Journal of Australia*, 1978. Dec. 2: 2(12); pp.570-572.
- 73. Food & Drug Administration (U.S.) Color Additives Fact Sheet. www.fda.gov/Food/Food/IngredientsPackaging/ucm094211.htm
- 74. Food & Drug Administration (U.S.) Report on the Certification of Color Additives. http://tinyurl.com/FDA-howmuch
- 75. Food & Drug Administration Public Health Advisory 2003, <u>http://tinyurl.com/fda-blue</u> Reports of Blue Discoloration and Death in Patients Receiving Enteral Feedings Tinted with the Dye, FD&C Blue No. 1.
- 76. Food & Drug Administration October 2004 Press Release about new Black Box Warning mandated for antidepressants. http://feingold.org/DOCS/fda-antidepressant-blackbox.pdf
- 77. Food & Drug Administration February 2007 FDA Directs ADHD Drug Manufacturers to Notify Patients about Cardiovascular Adverse Events and Psychiatric Adverse Events. <u>http://tinyurl.com/fda-blackbox</u>
- 78. Food & Drug Administration, Title 21 Part 74 Food & Drug Administration Listing of Color Additives Subject to Certification. http://tinyurl.com/dye-certification
- 79. Gaby AR, 2005. Adverse Effects of Dietary Fructose. Alternative Medicine Review, 2005 Dec;10(4):294-306.
- Gao Y, et al. 2011. Effect of Food Azo Dye Tartrazine on Learning and Memory Functions in Mice and Rats, and the Possible Mechanisms Involved. *Journal of Food Science*, Volume 76, Issue 6, pages T125–T129, August 2011.
- Genton C, et al. 1985. Value of Oral Provocation Tests to Aspirin and Food Additives in the Routine Investigation of Asthma and Chronic Urticaria. Journal of Allergy and Clinical Immunology 1985, Jul;76(1); p.40-5.
- Ghuman JK, et al. 2008. Psychopharmacological and Other Treatments in Preschool Children with ADHD: Current Evidence and Practice. J Child Adolesc Psychopharmacol. 2008 Oct; 18(5):413-47.
- Gibb C, et al. 1987. In Vitro Inhibition of Phenolsulphotransferase by Food and Drink Constituents. *Biochemical Pharmacology*, 1987. Jul 15;36(14):2325-30.
- 84. Goldenring et al. 190. Effects Of Continuous Gastric Infusion Of Food Dyes On Developing Rat Pups. *Life Sciences*, 1980 Nov 17;27(20):1897-904.
- Golub MS, et al. 2005. Neurobehavioral Evaluation of Rhesus Monkey Infants Fed Cow's Milk Formula, Soy Formula, or Soy Formula with Added Manganese. *Neurotoxicology & Teratology*, 2005 Jul-Aug;27(4):615-27
- Gomez NN, et al. 2006. Zn-limited Diet Modifies the Expression of the Rate-Regulatory Enzymes Involved in Phosphatidylcholine and Cholesterol Synthesis. *The British Journal of Nutrition*, 2006 Dec;96(6):1038-46.
- Goyette GH, et al. 1978. Effects of Artificial Colors on Hyperkinetic Children: A Double-Blind Challenge Study. *Psychopharmacology Bulletin.* 1978 Apr;14(2):39-40.
- Gross MD, et al. 1987. The Effect of Diets Rich in and Free From Additives on the Behavior of Children with Hyperkinetic and Learning Disorders. J of the American Academy of Child and Adolescent Psychiatry. 1987 Jan;26(1):53-5.
- Groten JP, 2000. An Analysis of the Possibility for Health Implications of Joint Actions and Interactions Between Food Additives. *Regulatory Toxicology and Pharmacology*. 2000 Feb;31(1):77-91.
- Hallfrisch J, 1990. Metabolic Effects of Dietary Fructose. Gerontology Research Center, National Institute on Aging, Baltimore, Maryland 21224. *The FASEB Journal*, 1990 Jun;4(9):2652-60.
- Hamazak T, et al. 2002. The Effect of Docosahexaenoic Acid on Aggression in Elderly Thai Subjects--A Placebo-Controlled Double-Blind Study. Nutritional Neuroscience. 2002 Feb;5(1):37-41.
- Harding KL, Judah RD, Gant C., 2003. Outcome-Based Comparison of Ritalin versus Food-Supplement Treated Children with AD/HD. Alternative Medicine Review. 2003 Aug; 8(3): 319-30.
- 93. Harley, JP et al. 1978. Hyperkinesis and Food Additives: Testing the Feingold Hypothesis. Pediatrics, 1978. June Vol. 61 (6) p. 818-827.
- 94. Harper PH, Goyette CH, Conners CK, 1978. Nutrient Intakes of Children on the Hyperkinesis Diet. Journal of the American Dietetic Association. 1978 Nov;73(5):515-9.
- 95. Harris RM, Waring RH, 1996. Dietary Modulation of Human Platelet Phenol-sulphotransferase Activity. *Xenobiotica*. 1996, Dec; 26 (12): 1241-7.
- 96. Harris RM, 1998. Inhibition of Phenolsulphotransferase by Salicylic Acid: A Possible Mechanism by Which Aspirin May Reduce Carcinogenesis. *Gut.* 1998 Feb; 42 (2):272-5.
- 97. Hashem MM, et al. 2010. Food and Chemical Toxicology. Food and Chemical Toxicology, 48(2010): 1581-1586.
- Hashem MM, et al. 2011. Toxicological Impact of Amaranth, Sunset Yellow and Curcumin as Food Coloring Agents in Albino Rats. J. Pak. Med Stus., 1(2): 43-51 July-September 2011.
- Hassan GM, 2010. Effects of Some Synthetic Coloring Additives on DNA and Chromosomal Aberrations of Rats. Arab J. Biotech., 13(1): 13-24.
- 100. Hedman SE, Andersson RG, 1981. Effects of Tartrazine on Different Contractile Stimuli in Guinea Pig Tracheal Muscle. Acta Pharmacologica et Toxicologica (Copenh) 1981 Feb;48(2):101-7.
- 101. Helal EGE 2001. Progressive Effects of the Interaction of Sodium Nitrite and Sunset Yellow (Yellow 6) on Different Physiological Parameters in Albino Rats, *The Egyptian J. Hospital Medicine*, Vol. 2: 23-46.
- 102. Henderson TA, Fischer VW, 1995. Effects of Methylphenidate (Ritalin) on Mammalian Myocardial Ultrastructure. American Journal of Cardiovascular Pathology. 1995;5(1):68-78.
- 103. Hong SP et al. 1989. Oral Provocation Tests with Aspirin and Food Additives in Asthmatic Patients. *Yonsei Medical Journal*, 1989. Dec.30(4): 339-45.
- 104. Howard AL, et al. 2010. ADHD is Associated with a "Western" Dietary Pattern in Adolescents. Attention Disorders 2010 Jul;15(5):403-11
- 105. **Hsieh** MH, et al. 2003. Efficacy and Tolerability of Oral Stevioside in Patients with Mild Essential Hypertension: A Two-Year, Randomized, Placebo-Controlled Study. *Clinical Therapeutics*. 2003 Nov;25(11):2797-808.
- 106. Huang SW, Borum PR 1998. Study of Skin Rashes After Antibiotic Use in Young Children. Clin Pediatr (Phila) 1998 Oct;37(10):601-7.
- 107. Husain A, et al. 2006. Estimates of Dietary Exposure of Children to Artificial Food Colours in Kuwait. *Food Additives & Contaminants* 2006 Mar;23(3):245-51.
- 108. Ibrahim AAE, et al. 2008. The Role of Ginger or Green Tea in Counteracting the Deleterious Effects of Benzene Sulfonic Acid in Weanling Male Rats. Egyptian J of Natural Toxins, 5(1,2): 56-99, Dec. 2008.
- 109. Inam QU, et al. 2006. Effects of Long Term Consumption of Sugar as Part of Meal on Serotonin 1-a receptor Dependent Responses. Pakistan Journal of Pharmaceutical Sciences. 2006 Apr;19(2):94-8.
- 110. Jacobson MF, Schardt D, 1999. Diet, ADHD & Behavior: A Quarter-Century Review, by Center for Science in the Public Interest, Washington, DC.

- 111. Jimenez-Aranda GS et al. 1996. Prevalence of Chronic Urticaria Following the Ingestion of Food Additives in a Third Tier Hospital. *Revista Alergia Mexico*, 1996 Nov-Dec; 43(6); p.152-6.
- 112. Johnson RJ, et al. 2011. Attention-Deficit/Hyperactivity Disorder: Is it Time to Reappraise the Role of Sugar Consumption? *Postgraduate Medicine*. 2011. Sep; 123(5):39-49.
- 113. Juhlin L, 1981. Recurrent Urticaria: Clinical Investigation of 330 Patients. British Journal of Dermatology, 1981 Apr;104(4):369-81.
- 114. Juhlin L, 1987. Additives and Chronic Urticaria, Annals of Allergy 1987 Nov;59(5 Pt 2):119-23.
- 115. Kahl R, Kahl GF, 1983. Effect of Dietary Antioxidants on Benzo[a]pyrene Metabolism in Rat Liver Microsomes. *Toxicology* 1983;28(3):229-33.
- 116. Kahl R, 1984. Synthetic Antioxidants: Biochemical Actions and Interference with Radiation, Toxic Compounds, Chemical Mutagens and Chemical Carcinogens. *Toxicology* 1984 Dec;33(3-4):185-228.
- 117. Kahl R, Kappus H, 1993. Toxicology of the Synthetic Antioxidants BHA and BHT in Comparison with the Natural Antioxidant Vitamin E. Z Lebensm Unters Forsch 1993 Apr;196(4):329-38.
- 118. Kalinke DU, Wuthrich B, 1999. Purpura Pigmentosa Progressiva in Type III Cryoglobulinemia and Tartrazine Intolerance. A Follow-up Over 20 years. Der Hautarzt. 1999 Jan;50(1):47-51.
- 119. Kamel MM, El-Lethey HS. 2011. The Potential Health Hazard of Tartrazine and Levels of Hyperactivity, Anxiety-Like Symptoms, Depression and Anti-Social Behaviour in Rats. J of American Science, 2011;7(6)
- 120. **Kaplan** B et al., 1989. Overall Nutrient Intake of Preschool Hyperactive and Normal Boys. *Journal of Abnormal Child Psychology*, April 1989, Vol. 17(2), pp.127-32.
- 121. Kaplan B et al., 1989. Dietary Replacement in Preschool-Aged Hyperactive Boys. Pediatrics, 1989, Vol. 83, pp. 7-17.
- 122. Kavale KA, Forness SR, 1983, Hyperactivity and Diet Treatment: A Meta-Analysis of the Feingold Hypothesis. *Journal of Learning Disabilities*, 1983 Jun-Jul;16(6):324-30.
- 123. Kellogg Report: 1989. The Impact of Nutrition, Environment & Lifestyle on the Health of Americans, by JD Beasley & J Swift, Institute of Health Policy & Practice, Bard College Center, 1989, Annandale-On-Hudson, NY 12504
- 124. Kelly KL, Rapport MD, DuPaul GJ, 1988. Attention Deficit Disorder and Methylphenidate: A Multi-Step Analysis of Dose-Response Effects on Children's Cardiovascular Functioning. *International Clinical Psychopharmacology*. 1988 Apr;3(2):167-81.
- 125. Khan 2011. Manganese Exposure from Drinking Water and Children's Classroom Behavior in Bangladesh. Environmental Health Perspectives, 2011 Oct;119(10):1501-6.
- 126. Kidd PM, 2000. Attention Deficit/Hyperactivity Disorder (ADHD) in Children: Rationale for its Integrative Management. Alternative Medicine Review 2000 Oct; 5 (5): 402-28.
- 127. **Kiddie** JY, et al. 2010. Nutritional Status of Children with Attention Deficit Hyperactivity Disorder: A Pilot Study. *International Journal of Pediatrics*, 2010:767318
- 128. Kim JY, et al. 2011. Aspartame-Fed Zebrafish Exhibit Acute Deaths with Swimming Defects and Saccharin-Fed Zebrafish Have Elevation of Cholesteryl Ester Transfer Protein Activity in Hypercholesterolemia. *Food & Chem. Toxicology*. 2011. Nov;49(11):2899-905.
- 129. Kleinman RE, et al. 2011. A Research Model for Investigating the Effects of Artificial Food Colorings on Children With ADHD. *Pediatrics* 2011; 127;e1575
- 130. Koutsogeorgopoulou L et al. 1998. Immunological Aspects of the Common Food Colorants, Amaranth and Tartrazine. *Veterinary and Human Toxicology*, 1998 Feb; 40(1); pp.1-4,
- 131. **Kroes** R, et al. 2000. Threshold of Toxicological Concern for Chemical Substances Present in the Diet: A Practical Tool for Assessing the Need for Toxicity Testing. *Food and Chemical Toxicology*. 2000 Feb-Mar;38(2-3):255-312.
- 132. Kroes R, Kozianowski G, 2002. Threshold of Toxicological Concern (TTC) in Food Safety Assessment. *Toxicology Letters* 2002 Feb 28;127(1-3):43-6.
- 133. Kroes R, et al. 2005 The Threshold of Toxicological Concern Concept in Risk Assessment. Toxicological Sciences. 2005 86(2):226-230; doi:10.1093/toxsci/kfi169
- 134. Lafferman JA, Silbergeld EK 1979. Erythrosin B Inhibits Dopamine Transport in Rat Caudate Synaptosomes. *Science* 1979. Jul 27; 205(4404): 410-2
- 135. Lancaster FE, Lawrence JF, 1999. Determination of Benzidine in the Food Colours Tartrazine (Yellow 5) and Sunset Yellow FCF (Yellow 6). Food Additives and Contaminants, 1999 Sep;16(9):381-90.
- 136. Lau K, et al. 2006. Synergistic Interactions Between Commonly Used Food Additives in a Developmental Neurotoxicity Test. *Toxicological Sciences*. 2006 Mar;90(1):178-87, 2005 Dec 13.
- 137. LeClercq C, et al. 2000. Estimates of the Theoretical Maximum Daily Intake of Erythorbic Acid, Gallates, Butylated Hydroxyanisole (BHA) and Butylated Hydroxytoluene (BHT) in Italy: A Stepwise Approach. *Food and Chem Toxicology*. 38 (2000) 1075-1084.
- 138. Levy F, et al. 1978. Hyperkinesis and Diet: A Double-Blind Crossover Trial with a Tartrazine Challenge. *Medical Journal of Australia* 1978 Jan 28;1(2):61-4.
- 139. Lien L, et al. 2006. Consumption of Soft Drinks and Hyperactivity, Mental Distress, and Conduct Problems Among Adolescents in Oslo, Norway. American Journal of Public Health. 2006 Oct;96(10):1815-20.
- 140. Litonjua AA, et al., 2006. Maternal Antioxidant Intake in Pregnancy and Wheezing Illnesses in Children at 2 y of Age, *The American Journal of Clinical Nutrition*, 2006 Oct;84(4):903-11.
- 141. Liu J; Wuerker A, 2005. Biosocial Bases of Aggressive and Violent Behavior Implications for Nursing Studies. International Journal of Nursing Studies, 2005 Feb;42(2):229-41
- 142. Lockey SD, 1977. Hypersensitivity to Tartrazine (Yellow No. 5) and Other Dyes and Additives Present in Foods and Pharmaceutical Products. *Annals of Allergy*, 1977 Mar; 38(3); pp.206-10.
- 143. Longo G, et al. 1987. Food Allergy in Asthma. Diagnostic Significance of Peripheral Eosinophils. *Pedoatroa Medica e Chirurgica*, 1987 Nov-Dec;9(6):663-8.
- 144. Lowen T. 2011. Do Artificial Sweeteners Contribute to Rather than Combat Obesity? Minnesota Med., 2011. Aug; 94(8):20-3.
- 145. Lu C, et al. 2006. Organic Diets Significantly Lower Children's Dietary Exposure to Organophosphorus Pesticides. *Environmental Health Perspectives* 2006 Feb;114(2):260-3.
- 146. Machinski JM, Estimates of Maximum Limits of Food Colours Use in Brazil through the Danish Budget Method and the Bär and Würtzenmodified method. Food Addit Contam. 1998 May-Jun;15(4):481-6.
- 147. Magerl M, et al. 2010. Effects of a Pseudoallergen-free Diet on Chronic Spontaneous Urticaria: A Prospective Trial. Allergy. 2010 Jan;65(1):78-83
- 148. **Maher** TJ, Wurtman RJ, 1987. Review. Possible Neurologic Effects of Aspartame, a Widely Used Food Additive. *Environmental Health Perspective* 1987 Nov;75:53-7.
- 149. **Markowitz** JS, et al. 1999. Detection of the Novel Metabolite Ethylphenidate After Methylphenidate Overdose with Alcohol Coingestion. *Journal of Clinical Psychopharmacology*, 1999 Aug;19(4):362-6.

- 150. Mattes JA, Gittelman R, 1981. Effects of Artificial Food Colorings in Children with Hyperactive Symptoms: A Critical Review and Results of a Controlled Study. *Archives of General Psychiatry*. 1981 Jun;38(6):714-8.
- 151. McCann D, et al. 2007. Food Additives and Hyperactive Behaviour in 3-Year-Old and 8/9-Year-Old Children in the Community: A Randomised, Double-Blinded, Placebo-Controlled Trial. *The Lancet*, 2007, Nov 3; 370(9598):1560-7.
- McFadden SA, 1996. Phenotypic Variation in Xenobiotic Metabolism and Adverse Environmental Response: Focus on Sulfur-Dependent Detoxification Pathways. *Toxicology*, July 1996, Vol. 111(1-3), pp. 43-65.
- 153. **McFarlane** M, et al. 1997. Hepatic and Associated Response of Rats to Pregnancy, Lactation and Simultaneous Treatment with Butylated Hydroxytoluene. *Food and Chemical Toxicology*. 1997 Aug;35(8):753-67.
- 154. Mehedi N, et al. 2009. Reproductive Toxicology of Tartrazine (FD&C Yellow No. 5) in Swiss Albino Mice. American Journal of Pharmacology and Toxicology 4 (4): 128-133, 2009
- 155. **Meyer** AM, et al. 2006. Attenuation of the Pulmonary Inflammatory Response Following Butylated Hydroxytoluene Treatment of Cytosolic Phospholipase A2 Null Mice. *Am. J. Physiol. Lung Cell Mol. Physiol.* 2006. Jun; 290(6):L1260-6.
- 156. Meyer O, Hansen E, 1980. Behavioural and Developmental Effects of Butylated Hydroxytoluene Dosed to Rats in Utero and in the Lactation Period. *Toxicology* 1980;16(3):247-58.
- 157. Millichap JG & Yee MM. 2012. The Diet Factor in ADHD. Pediatrics. Feb 2012. 129(2):330-337.
- 158. **Mizutan**i T, 2009. Toxicity of Xanthene Food Dyes by Inhibition of Human Drug-Metabolizing Enzymes in a Noncompetitive Manner. J Environ Public Health. 2009;2009:953952
- 159. Moutinho ILD et al. 2007. Prolonged Use of the Food Dye Tartrazine (FD&C Yellow No.5) and its Effects on the Gastric Mucosa of Wistar Rats. *Brazilian J. of Biology*. 67(1): 141-145, 2007
- 160. **Mpountoukas** P et al. 2010. Cytogenetic Evaluation and DNA Interaction Studies of the Food Colorants Amaranth [Red 3], Erythrosine [Red 3] and Tartrazine [Yellow 5].
- 161. MTA 1999. A 14-Month Randomized Clinical Trial of Treatment Strategies for Attention-Deficit/Hyperactivity Disorder, Arch.Gen. Psychiatry, Dec. 1999, Vol.56, pp. 1073-1086.
- 162. MTA 2009. The MTA at 8 Years: Prospective Follow-Up of Children Treated for Combined-Type ADHD in a Multisite Study. J Am. Acad. Child Adolesc. Psychiatry, 2009 May; 48(5): 484-500.
- 163. Murphy P, Burnham WM, 2006. The Ketogenic Diet Causes a Reversible Decrease in Activity Level in Long-Evans Rats. *Experimental Neurology*. 2006 Sep;201(1):84-9
- 164. Nakao H, et al. 2003. Formaldehyde-Induced Shrinkage of Rat Thymocytes. Journal of Pharmacological Sciences, 2003 Jan;91(1):83-6.
- 165. National Academy of Sciences, 1977 Survey of Industry on the Use of Food Additives, published 1979.
- 166. Naveena BM, et al. 2008. Comparative Efficacy of Pomegranate Juice, Pomegranate Ring Powder Extract and BHT as Antioxidants in Cooked Chicken Patties. *Meat Science*. 2008 Dec; 80(4): 1304-8.
- 167. Negrao, BL, Crafford D, Viljoen M, 2009. The Effect of Sympathomimetic Medication on Cardiovascular Functioning of Children with Attention-Deficit/Hyperactivity Disorder. *Cardiovascular J of Africa*, Vol. 20, No. 5, September/October 2009.
- 168. Neuman I, et al. 1978. The Danger of "Yellow Dyes" (Tartrazine) to Allergic Subjects. Clinical Allergy. 1978 Jan;8(1):65-8.
- Newman LC, Lipton RB 2001. Migraine MLT-down: An Unusual Presentation of Migraine in Patients with Aspartame-triggered Headaches. *Headache*. 2001 Oct;41(9):899-901.
- 170. Niederhofer H, Pittschieler K, 2006. A Preliminary Investigation of ADHD Symptoms in Persons with Celiac Disease. J of Attention Disorders. 2006 Nov;10(2):200-4.
- 171. Niederhofer H. 2011. Association of ADHD and Celiac Disease. Primary Care Companion to CNS Disorders. 2011. 13(3)pii.
- 172. Nigg JT, et al. 2012. Meta-Analysis of ADHD or ADHD Symptoms, Restriction Diet, and Synthetic Food Color Additives. J Am Acad Child Adolesc Psychiatry. 2012. Jan; 51(1):86-97.e8.
- 173. NIH Twelfth Report on Carcinogens 2011, http://ntp.niehs.nih.gov/ntp/roc/twelfth/roc12.pdf
- 174. Nsouli TM, et al. 1994. Role of Food Allergy in Serous Otitis Media. Annals of Allergy 1994 Sep;73(3):215-9.
- 175. **Oades** RD, Daniels R, Rascher W. 1998. Plasma Neuropeptide-Y Levels, Monoamine Metabolism, Electrolyte Excretion and Drinking Behavior in Children with Attention-Deficit Hyperactivity Disorder. *Psychiatry Research* 1998 Aug 17;80(2):177-86.
- 176. Olfson M, Marcus SC, Shaffer D, 2006. Antidepressant Drug Therapy and Suicide in Severely Depressed Children and Adults: A Case-Control Study. Archives of General Psychiatry. 2006 Aug;63(8):865-72.
- 177. Pachor ML, et al. 1989. Is the Melkersson-Rosenthal Syndrome Related to the Exposure to Food Additives? A Case Report. Oral Surgery, Oral Medicine, and Oral Pathology 1989 Apr;67(4):393-5.
- 178. **Park** EH, et al. 1990. Induction of Hepatic Tumors With Butylated Hydroxyanisole in the Self-Fertilizing Hermaphroditic Fish Rivulus Ocellatus Marmoratus. *Jpn J Cancer Res.* 1990 Aug;81(8):738-41.
- 179. Peiperl MD, Prival MJ, Bell SJ, 1995. Determination of Combined Benzidine in FD&C Yellow No.6 (Sunset Yellow FCF). Food Chem. Toxicol. 1995 Oct.33(10): 829-39.
- 180. Pellow J, et al. 2011. Complementary and Alternative Medical Therapies for Children with ADHD. Altern Med Rev 2011. 16(4):323-37.
- 181. Pelsser LM, Buitelaar JK, 2002. Favourable Effect of a Standard Elimination Diet on the Behavior of Young Children with Attention Deficit Hyperactivity Disorder (ADHD): A Pilot Study. Ned Tijdschr Geneeskd 2002 Dec 28;146(52):2543-7.
- 182. **Pelsser** LM, et al. 2009. A Randomised Controlled Trial into the Effects of Food on ADHD. *European Child & Adolescent Psychiatry*. 2009 Jan;18(1):12-9.
- 183. Pelsser LM, et al. 2010. Effects of Food on Physical and Sleep Complaints in Children with ADHD: A Randomised Controlled Pilot Study.
- 184. Pelsser LM, et al. 2011. Effects of a Restricted Elimination Diet on the Behaviour of Children with Attention-Deficit Hyperactivity Disorder (INCA study): A Randomised Controlled Trial. *Lancet*. 2011. Feb 5; 377(9764):494-503.
- 185. Peng W et al. 2009. Systemic Administration of an Antagonist of the ATP-Sensitive Receptor P2X7 Improves Recovery After Spinal Cord Injury. Proc Natl Acad Sci U S A. 2009 Jul 28;106(30):12489-93.
- 186. Pestana S, Moreira J, Olej B, 2010. Safety of Ingestion of Yellow Tartrazine by Double-Blind Placebo Controlled Challenge in 26 Atopic Adults. Allergol Immunopathol (Madr), 2010. 38(3): 142-146.
- 187. Petitpierre M, Gumowski P, Girard JP, 1985. Irritable Bowel Syndrome and Hypersensitivity to Food. Annals of Allergy 1985 Jun; 54(6):538-40.
- 188. Pollock I, Warner JO, 1990. Effect of Artificial Food Colours on Childhood Behaviour. Arch Dis Child 1990 Jan;65(1):74-7.
- 189. Prival MJ, Peiperl MD, 1993. Determination of Combined Benzidine in FD&C Yellow No. 5 (Tartrazine), Using a Highly Sensitive Analytical Method. *Food Chem. Toxicol* 1993 Oct. 31(10): 751-8.
- 190. **Raby** SE, 1995. The Examination of the Link Between Pesticides in Food and Learning Disorders in Children, Dominical College Masters Theses, Faculty of the Dominical College Dept.of Ed.
- 191. Reyes FG, Valim MF, Vercesi AE. 1996. Effect of Organic Synthetic Food Colours on Mitochondrial Respiration. *Food Additives and Contaminants*. 1996 Jan;13(1):5-11.

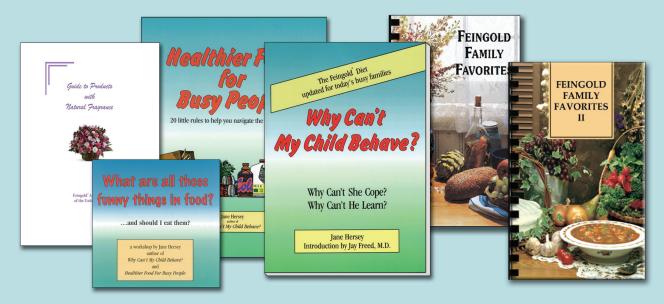
- 192. Rimland B, 1983. The Feingold Diet: An Assessment of the Reviews by Mattes, by Kavale and Forness and Others. Journal of Learning Disabilities, 1983 Jun-Jul;16(6):331-3.
- 193. Roberts HJ, 2001. Book: Aspartame Disease: An Ignored Epidemic, West Palm Beach: Sunshine Sentinel Press. 1018 p.
- 194. Robson WL, et al. 1997. Enuresis in Children with Attention-Deficit Hyperactivity Disorder. Southern Med Journal 1997 May;90(5):503-5.
- 195. Rose TL 1978. The Functional Relationship Between Artificial Food Colors and Hyperactivity. J of Applied Behavior Analysis 1978 Winter;11(4):439-46
- 196. Rosenkranz HS, Klopman G, 1990. Structural Basis of the Mutagenicity of 1-amino-2-naphthol-based Azo Dyes. *Mutagenesis* 1990 Mar;5(2):137-46.
- 197. Rowe KS, 1988. Synthetic Food Colourings and "Hyperactivity": a Double-Blind Crossover Study. Australia Paediatric Journal, April 1988, Vol. 24 (2), pp. 143-7.
- 198. **Rowe** KS, Rowe KJ, 1994. Synthetic Food Coloring and Behavior: A Dose Response Effect in a Double-Blind, Placebo-Controlled, Repeated-Measures Study. *Journal of Pediatrics*, November 1994, Vol. 135, pp.691-8.
- 199. **Ruppert** PH, Dean KF, Reiter LW, 1985. Development of Locomotor Activity of Rat Pups Exposed to Heavy Metals. *Toxicology and* Applied Pharmacology 1985 Mar 30;78(1):69-77.
- 200. Safer AM, al-Nughamish AJ, 1999. Hepatotoxicity Induced by the Anti-Oxidant Food Additive, Butylated Hydroxytoluene (BHT), in Rats: An Electron Microscopical Study. *Histology and Histopathology* 1999 Apr;14(2):391-406.
- 201. Sagiv SK, et al. 2010. Prenatal Organochlorine Exposure and Behaviors Associated with Attention-Deficit Hyperactivity Disorder in School-Aged Children. *American Journal of Epidemiology*, 2010, Mar 1; 171(5):593-601.
- 202. Sakakibara H, Suetsugu S, 1995. Aspirin-Induced Asthma as an Important Type of Bronchial Asthma. Nihon Kyōbu Shikkan Gakkai zasshi, 1995 Dec;33 Suppl:106-15.
- 203. Salamy J, et al. 1982. Physiological Changes in Hyperactive Children Following the Ingestion of Food Additives. International Journal of Neuroscience 1982 May;16(3-4):241-246.
- 204. Salzman LK, 1976. Allergy Testing, Psychological Assessment and Dietary Treatment of the Hyperactive Child Syndrome. *Medical Journal of Australia* 1976 Aug 14;2(7):248-51.
- 205. Sarafian TA, et al. 2002. Synergistic Cytotoxicity of Delta(9)-tetrahydrocannabinol and Butylated Hydroxyanisole. *Toxicology Letters* 2002 Jul 21;133(2-3):171-9.
- 206. Sasaki YF, et al. 2002. The Comet Assay with 8 Mouse Organs: Results with 39 Currently Used Food Additives. *Mutation Research* 2002 Aug 26;519(1-2):103-19.
- 207. Scadding GK et al. 1988. Poor Sulphoxidation Ability in Patients with Food Sensitivity. *British Medical Journal*, 1988 Jul 9; 297 (6641): 105-7.
- 208. Schab DW, Trinh NH, 2004. Do Artificial Food Colors Promote Hyperactivity in Children with Hyperactive Syndromes? A Meta-Analysis of Double-Blind Placebo-Controlled Trials. *Journal of Developmental and Behavioral Pediatrics*. 2004 Dec;25(6):423-34.
- 209. Schmidt MH et al. 1997. Does Oligoantigenic Diet Influence Hyperactive/Conduct-Disordered Children -- A Controlled Trial. *European Child & Adolescent Psychiatry*, 1997 Jun;6(2):88-95.
- 210. Schnoll R, Burshteyn D, Cea-Aravena J, 2003. Nutrition in the Treatment of Attention-Deficit Hyperactivity Disorder: A Neglected but Important Aspect. Applied Psychophysiology and Biofeedback. 2003 Mar;28(1):63-75.
- 211. Schnyder B, et al. 1999. Food Intolerance and Food Allergy. Schweiz Med Wochenschr, 1999 Jun 19; 129(24): 928-33.
- 212. Schoenthaler S, 1983. Diet and Crime: An Empirical Examination of the Value of Nutrition in the Control and Treatment of Incarcerated Juvenile Offenders. *International Journal of Biosocial Research*, 1983: 4(1); 25-39
- 213. Schoenthaler S, Doraz W. 1983a. Types of Offenses Which Can be Reduced in an Institutional Setting Using Nutritional Intervention: A Preliminary Empirical Evaluation. *International Journal of Biosocial Research*, 1983: 4(2); 74-84.
- 214. Schoenthaler S. 1983b. The Northern California Diet-Behavior Program: An Empirical Examination of 3,000 Incarcerated Juveniles in Stanislaus County Juvenile Hall. *International Journal of Biosocial Research*, 1983: 5(2); 99-106.
- 215. Schoenthaler SJ, 1985. Institutional Nutritional Policies and Criminal Behavior, Nutrition Today, 1985: 20(3); 16.
- 216. Schoenthaler SJ, Doraz WE, Wakefield JA. 1986. The Impact of a Low Food Additive and Sucrose Diet on Academic Performance in 803 New York City Public Schools. *International Journal of Biosocial Research*, 1986, 8(2); 185-195.
- 217. Schoenthaler, SJ, Doraz WE, Wakefield JA. 1986a The Testing of Various Hypotheses as Explanations for the Gains in National Standardized Academic Test Scores in the 1978-1983 New York City Nutrition Policy Modification Project, International Journal of Biosocial Research, 1986, 8(2): 196-203.
- 218. Schoenthaler S, Moody J, Pankow L, 1991. Applied Nutrition and Behavior. Journal of Applied Nutrition, November 1, 1991, Vol. 43.
- 219. Shaywitz BA, Goldenring JR, Wool, RS. 1979. Effects of Chronic Administration of Food Colorings on Activity Levels and Cognitive Performance in Developing Rat Pups Treated with 6-Hydroxydopamine. *Neurobehavioral Toxicology* 1979 Spring;1(1):41-7.
- 220. Shimada C, et al. 2010. Differential Colon DNA Damage Induced by Azo Food Additives Between Rats and Mice. *Journal of Toxicological Sciences* 2010;35(4):547-54.
- 221. Siman CM, Eriksson UJ, 1996. Effect of Butylated Hydroxytoluene on Alpha-Tocopherol Content in Liver and Adipose Tissue of Rats. *Toxicology Letters* 1996 Oct;87(2-3):103-8.
- 222. Sinaiko RJ 1996. The Biochemistry of Attentional/Behavioral Problems. www.feingold.org/Research/sinaiko.html
- 223. Sinn N 2008. Nutritional and Dietary Influences on Attention Deficit Hyperactivity Disorder. Nutrition Reviews. 2008 Oct;66(10):558-6.
- 224. **Sloper** KS, Wadsworth J, Brostoff J, 1991. Children with Atopic Eczema: Clinical Response to Food Elimination and Subsequent Double-Blind Food Challenge. *Quarterly J of Medicine*, 1991 Aug; 80(292):677-93.
- 225. Soubra L, et al. 2007. Dietary Exposure of Children and Teenagers to Benzoates, Sulphites, Butylhydroxyanisol (BHA) and Butylhydroxylouen (BHT) in Beirut (Lebanon). *Regulatory Toxicology & Pharmacology*. 2007 Feb;47(1):68-77.
- 226. Spencer PS, Bischoff JC, 1984. Skin as a Route of Entry for Neurotoxic Substances. Dermatoxicology (1984) 3rd Ed. p.629-630 Wash. DC.
- 227. Stevens LJ et al. 2011. Dietary Sensitivities and ADHD Symptoms: 35 Years of Research. Clin Pediatr (Phila). 2011 Apr;50(4):279-93.
- 228. **Stokes** JD, Scudder CL, 1974. The Effect of Butylated Hydroxyanisole and Butylated Hydroxytoluene on Behavioral Development of Mice. *Developmental Psychobiology* 1974 Jul;7(4):343-50.
- 229. **Stolze** K, Nohl H, 1999. Free Radical Formation and Erythrocyte Membrane Alterations During MetHb Formation Induced by the BHA Metabolite, Tert-Butylhydroquinone. *Free Radical Research*. 1999 Apr;30(4):295-303.
- 230. Swain AR, Dutton SP, Truswell AS, 1985. Salicylates in Foods. Journal of the American Dietetic Association 1985 Aug;85(8):950-60.
- 231. Swain A, Soutter V, Loblay R, Truswell AS. 1985. Salicylates, Oligoantigenic Diets, and Behaviour. The Lancet, 1985 Jul 6;2(8445):41-2.
- 232. Swanson J, Kinsbourne M, 1980. Food Dyes Impair Performance of Hyperactive Children on a Laboratory Learning Test. Science Magazine, March 28, 1980, Vol. 207. pp.1485-7.
- 233. Sweeney EA, Chipman JK, Forsythe SJ, 1994. Evidence for Direct-Acting Oxidative Genotoxicity by Reduction Products of Azo Dyes. Environmental Health Perspectives 1994 Oct;102 Suppl 6:119-22.

- 234. **Takami** M, et al. 1999. Antioxidants Reversibly Inhibit the Spontaneous Resumption of Meiosis. *American Journal of Physiology*. 1999 Apr;276(4 Pt 1):E684-8.
- 235. Tanaka T, Oishi S, Takahashi O, 1993. Three Generation Toxicity Study of Butylated Hydroxytoluene Administered to Mice. *Toxicology Letters* 1993 Mar;66(3):295-304.
- 236. Tanaka T, 1993. Reproductive and Neurobehavioral Effects of Amaranth Administered to Mice in Drinking Water. *Toxicology and Industrial Health.* 1993 Nov-Dec;9(6):1027-35.
- 237. Tanaka T, 1996. Reproductive and Neurobehavioral Effects of Sunset Yellow FCF Administered to Mice in the Diet. *Toxicology and Industrial Health.* 1996 Jan-Feb;12(1):69-79.
- 238. **Tanaka** T, 2001. Reproductive and Neurobehavioural Toxicity Study of Erythrosine Administered to Mice in the Diet. *Food and Chemical Toxicology*. 2001 May;39(5):447-54.
- 239. **Tanaka** T, 2006. Reproductive and Neurobehavioural Toxicity Study of Tartrazine Administered to Mice in the Diet. *Food and Chemical Toxicology*. 2006 Feb; 44(2): 179-87.
- 240. Thompson DC, Trush MA, 1988. Studies on the Mechanism of Enhancement of Butylated Hydroxytoluene-Induced Mouse Lung Toxicity by Butylated Hydroxyanisole. *Toxicology & Applied Pharmacology* 1988 Oct;96(1):122-31.
- 241. **Thompson** DC, Trush MA, 1988. Enhancement of Butylated Hydroxytoluene-Induced Mouse Lung Damage by Butylated Hydroxyanisole. *Toxicology & Applied Pharmacology* 1988 Oct;96(1):115-21.
- 242. Thompson D, Moldeus P, 1988. Cytotoxicity of Butylated Hydroxyanisole and Butylated Hydroxytoluene in Isolated Rat Hepatocytes. Biochemical Pharmacology. 1988 Jun 1;37(11):2201-7.
- 243. Thompson DC, Trush MA, 1989. Enhancement of the Peroxidase-Mediated Oxidation of Butylated Hydroxytoluene to a Quinone Methide by Phenolic and Amine Compounds. *Chemico-Biological Interactions*. 1989;72(1-2):157-73.
- 244. Thompson DC, Cha YN, Trush MA, 1989. The Peroxidase-Dependent Activation of Butylated Hydroxyanisole (BHA) and Butylated Hydroxytoluene (BHT) to Reactive Intermediates. Formation of BHT-quinone methide Via a Chemical-Chemical Interaction. Journal of Biological Chemistry. 1989 Mar 5;264(7):3957-65.
- 245. Tripathi M, et al. 2010. Intake Pattern of Synthetic Colours by Different Age and Socio-Economic Consumer Groups of Lucknow, India. International Journal of Food, Nutrition and Public Health, 2010. 3(1):19-31
- 246. **Tryphonas** H, et al. 1999. The Effect of Butylated Hydroxytoluene on Selected Immune Surveillance Parameters in Rats Bearing Enzyme-Altered Hepatic Preneoplastic Lesions. *Food and Chemical Toxicology*. 1999 Jul;37(7):671-81.
- 247. **Tsuda** S, et al. 2001. DNA Damage Induced by Red Food Dyes Orally Administered to Pregnant and Male Mice. *Toxicological Sciences* 2001 May;61(1):92-9.
- 248. Uhlig T, et al. 1997. Topographic Mapping of Brain Electrical Activity in Children with Food-induced Attention Deficit Hyperkinetic Disorder. *European Journal of Pediatrics.* 1997; 156; 557-561.
- 249. Umemura T, et al. 2006. Nine-Week Detection of Six Genotoxic Lung Carcinogens Using the RasH2/BHT Mouse Model. *Cancer Letters*, 231 (2006) pp. 314-318.
- 250. Van Bever HP, Docx M, Stevens WJ, 1989. Food and Food Additives in Severe Atopic Dermatitis. Allergy 1989 Nov;44(8):588-94.
- 251. Veien NK, Krogdahl A, 1991. Cutaneous Vasculitis Induced by Food Additives. Acta Dermato-Venereologica 1991;71(1):73-4.
- 252. Verhagen H, et al. 1990. Estimate of the Maximal Daily Dietary Intake of BHA and BHT in the Netherlands. *Food & Chemical Toxicology* 1990 Apr;28(4):215-20.
- 253. Vorhees CV, et al. 1983. Developmental Toxicity and Psychotoxicity of FD and C Red Dye No. 40 (Allura Red AC) in Rats. *Toxicology* 1983;28(3):207-17.
- 254. Walsh WJ, et al. 1997. Elevated Blood Copper/Zinc Ratios in Assaultive Young Males. Physiology & Behavior, 1997 Aug;62(2):327-9.
- 255. Wang GJ, et al. 1994. Methylphenidate Decreases Regional Cerebral Blood Flow in Normal Human Subjects. Life Sci. 1994;54(9):143-6.
- 256. Ward NI, et al. 1990. The Influence of the Chemical Additive Tartrazine on the Zinc Status of Hyperactive Children: A Double-Blind Placebo-Controlled Study. *J Nutr Med*; 1 (1). 1990. 51-58.
- 257. Ward NI, 1997. Assessment of Chemical Factors in Relation to Child Hyperactivity. Journal of Nutritional & Environmental Medicine (Abingdon); 7 (4). 1997. 333-342.
- 258. Waring RH, et al. 2008. Phytoestrogens and Xenoestrogens: The Contribution of Diet and Environment to Endocrine Disruption. J Steroid Biochem Mol Biol. 2008. Feb; 108(3-5):213-20.
- 259. Warrington RJ, Sauder PJ, McPhillips S, 1986. Cell-Mediated Immune Responses to Artificial Food Additives in Chronic Urticaria. *Clinical Allergy* 1986 Nov;16(6):527-33.
- 260. Weiss B, et al. 1980. Behavioral Responses to Artificial Food Colors. Science, 1980, Vol. 207, 1487-1489.
- 261. Weiss B, 1982. Food Additives and Environmental Chemicals as Sources of Childhood Behavior Disorders. *Journal of the American Academy of Child Psychiatry* 21,2:144-52, 1982.
- 262. Weiss B, 2012. Synthetic Food Colors and Neurobehavioral Hazards: The View from Environmental Health Research. *Environmental Health Perspectives*. 2012. Jan. 120(1):1-5.
- 263. Williams JI, et al. 1978. Relative Effects of Drugs and Diet on Hyperactive Behaviors: An Experimental Study. *Pediatrics*. 1978 Jun;61(6):811-7.
- 264. Wolraich ML, et al. 1994. Effects of Diets High in Sucrose or Aspartame on the Behavior and Cognitive Performance of Children. *New England Journal of Medicine*. 1994 Feb 3;330(5):301-7.
- 265. Worm M, et al. 2001. Increased Leukotriene Production by Food Additives in Patients with Atopic Dermatitis and Proven Food Intolerance. *Clinical and Experimental Allergy*. 2001 Feb;31(2):265-73.
- 266. Wuthrich B, Fabro L, 1981. Acetylsalicylic Acid and Food Additive Intolerance in Urticaria, Bronchial Asthma and Rhinopathy. Schweiz Med Wochenschr 1981 Sep 26;111(39):1445-50.
- 267. Yoneyama H, et al. 2000. The Effect of DPT and BCG Vaccinations on Atopic Disorders. Arerugi 2000 Jul;49(7):585-92.
- 268. Yu R, Mandlekar S, Kong AT, 2000. Molecular Mechanisms of Butylated Hydroxylanisole-Induced Toxicity: Induction of Apoptosis Through Direct Release of Cytochrome C. *Molecular Pharmacology*. 2000 Aug;58(2):431-7.
- 269. Zoccarato F, et al. 1987. Inhibition by Some Phenolic Antioxidants of Ca2+ Uptake and Neurotransmitter Release from Brain Synaptosomes. *Biochemical & Biophysical Research Communications*. 1987 Jul 31;146(2):603-10.

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