

# Relevant Research

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

★ = Double Blind Placebo Controlled Study

### 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."

## 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 thousands of additives eliminated by the Feingold Program are ignored.
- **The increasing use of synthetic colorings without adequate testing:** A recent study of children (Husain 2006) revealed that the children are eating **2 to 8 times more than the acceptable daily intake (ADI)** of food coloring. The authors are concerned about the possible health effects on the children. This study took place in Kuwait. How much food coloring are American children eating?
- Since companies refuse to reveal how much coloring is in their products, how do you know how much you are actually eating? To give you an idea, two of our students at a university in Atlanta measured the following:
  - 1 TB red frosting (or other opaque item) = 150 mg of Red #40 (3 TB to frost a cupcake = **450 mg**)
  - 1 TB green ketchup = 150 mg of Yellow #5 + Blue #1 mixed (2 TB for fries and a burger = **300 mg**)
  - Small cup (6 oz) red powder-mix drink (Red #40) = **18 to 20 mg**

**NO STUDY of 400-800 mg food dyes has ever been done on any children – normal or ADHD!**

## Medication for ADHD

*Stimulant medications work - often dramatically. Unfortunately, one of the worst side effects of these drugs – small vessel disease – can only be diagnosed 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 D, 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. 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. In all of them, chromosome abnormalities were tripled. The relationship between chromosome abnormalities and cancer is well-documented.

**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.

**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 were examined. Heart damage was found in all cases, even with the smallest doses 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*). Authors do not know what this chemical does or if it is toxic. **Note:** *Taking Ritalin to drink more alcohol without passing out is a new “party” activity.*

**Olfson** 2006 – In this case-matched study, antidepressant drug treatment in children under 19 was significantly associated with suicide attempts and deaths. Antidepressants are sometimes used with ADHD treatment.

**Public Citizen** 2007 – In their newsletter, *Best Pills Worst Pills*, they report that all stimulant medications for ADHD, and also Adderall, must carry “**Black Box warnings**” about the risk of these medications to cause increased blood pressure, stroke, heart attack, new or worse behavior, bipolar illness, increased aggression, psychotic or manic symptoms, as well as sudden death in patients with heart defects.

**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.

## ADHD and Autism Research

**Bateman** 2004 ★ In a large group of normal toddlers, a small (20 mg) amount of coloring with benzoate preservative caused adverse effects detectable by parents. Bateman suggests 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. After 2 years, 5 of them had never re-offended. The authors recommend this approach for criminal justice, education, and health agencies.

**Boris** 1994 ★ **73%** of 26 children with ADHD responded well to an elimination diet. 16 of them were given a double-blind test with 100 mg of color and suspected foods. **ALL** reacted to it. Boris concludes, “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 **not** 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.”

**Brenner** 1979 – Lab tests – 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 27.*)

**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.

### Risk-Benefit Analysis

*By Philip Handler, Pres.\**

*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 whatsoever.

*\*2 terms, 1969-81. He also received the National Medal of Science.*

**Carter** 1993 ★ **75.6%** of 78 children diagnosed with "hyperactive behavior" 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 a diet without synthetic additives. *Note: The study was done in Australia where much more calcium propionate is used in bread than in U.S. Products containing this preservative are marked in the Feingold Foodlist and Shopping Guides.*

Some researchers prefer the "oligoantigenic (*few foods*) diet" which eliminates all additives and allows only a very few foods. It is useful for a short trial for diagnosis, but not for satisfactory long-term use.

**Dumbrell** 1978 – This was a study on the nutritional quality of the Feingold Diet. Dumbrell concluded that the Feingold Diet was superior to the normal diet in nutritional quality.

**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. Other symptoms such as headaches, abdominal pain, and seizures also improved.

**Egger** 1989 ★ **80%** of 45 children with epilepsy, and also physical or behavioral problems, recovered or improved on a "few foods" diet. Headaches, abdominal pain, and hyperactive behavior stopped in all those whose seizures stopped, and in some of those whose seizures did not. In double-blind, placebo-controlled provocation studies, symptoms returned in **94%** of the children when 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 these children.

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. Goyette 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 one week on an additive-containing diet. Gross concluded that the Feingold Diet has no merit, although he conceded 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 [www.diet-studies.com/adhd.html#Gross](http://www.diet-studies.com/adhd.html#Gross).*

**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 food intake of 150 mg per day was not enough, but getting an extra 1.5 g 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. 1.5 g of DHA is 1500 mg – 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.”

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

**Harley 1978** ★ 36 school-age boys were tested with 2 diets after stopping their medication. Only 22 of them were neurologically normal and had normal EEGs, while 14 had various neurological problems besides ADHD. 13 children improved on the Feingold Diet. 12 of them (92%) were in the group having the control (additive-containing) diet first. This fits with Dr. Feingold's findings that it takes longer for a child who was on stimulants to respond to the diet. In this study, Harley trusted product ingredient lists, not Feingold materials, and did not eliminate preservatives.

The “control” diet also 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.

**Harper 1978** – Both during baseline and while following the “hyperkinesis diet,” nutrient intakes of 54 hyperactive children “compared favorably with the Recommended Dietary Allowances.”

**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 2-8. Authors call for studies into potential adverse health effects associated with the high intake of these artificial colors.

**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** ★ Mothers of some of these children reported more symptoms during the challenge period, but objective tests did not show a significant deterioration. Tests were given the DAY AFTER a challenge with cookies containing only ONE mg 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 in 24 hours.

**Lien 2006** – In a survey of 5,498 children in Norway, 4 or more glasses of sugar-containing soft drinks per day were associated with mental health problems and hyperactivity. *Note: Soda usually contains corn syrup (not ordinary sugar) as well as synthetic coloring, flavoring, and preservative chemicals. This study shows a correlation between soda and mental health, but does not prove causation.*

**Mattes 1981** ★ Calling it a “high dose,” Mattes gave cookies with only 13 mg of food dye to children well established on the Feingold Diet. He concluded there was “no evidence of a food coloring effect.” *Note: After being on the diet for some time, most children can handle an occasional exposure to food additives without obvious effects.*

**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 (CD) were assessed for ADHD symptoms before and 6 months after starting a gluten-free diet. Their Connors 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.*

**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 Connors Scale and the ADHD Rating Scale. “In young children with ADHD an elimination diet can lead to a statistically significant decrease in symptoms.”

**Pollock** 1990 ★ Artificial food colors had “an adverse effect” on the Connors 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, other 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. Reactions and length of time the children were affected depended on the dose. Investigators used their own more sensitive questionnaire, not the Connors questionnaire.

**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.

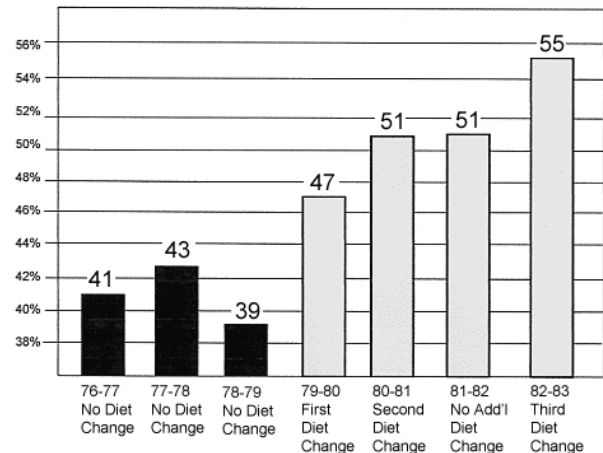
**Salzman** 1976 – **93%** of 15 children given the Australian version of the Feingold Diet improved in the areas of overactivity, distractibility, impulsiveness and excitability. Sleep and enuresis (*bed-wetting*) problems were resolved partially or completely.

**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. Each improvement came closer to the Feingold Diet, and was accompanied by an improvement in test score averages on national tests. 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 2 years below grade level before the change. Afterwards, only 4.9% of them were more than 2 years below grade level.

**Schoenthaler** 1991 – Improving the diet in 813 state facilities (*jails*) resulted in “significantly improved conduct, intelligence, and/or academic performance...”

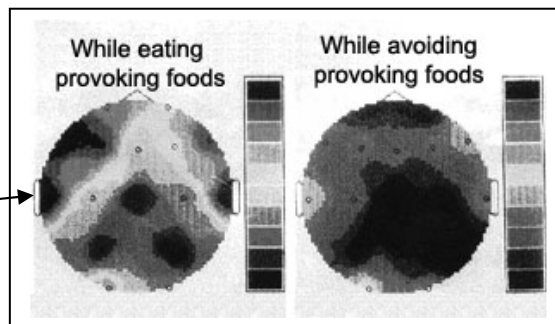


**Swanson** 1980 ★ After a few days on an additive-free diet, 40 children were given 100-150 mg of mixed food dyes, or placebo. Half the children were hyperactive and half were normal controls. A learning test showed that the learning ability of the hyperactive children (but NOT the controls) was worse 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 two or three pieces of colored candy, one cupcake with bright colored frosting, or a few cups of Kool-Aid. If 100 mg is to be considered “toxic,” then why is it so freely allowed in the supermarket, while its use by researchers is restricted?*

**Uhlig 1997** ★ This study is the first 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, hyperactive 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 cannot exist – after all, a headache, too, is subjective.*

*However, research has revealed there ARE biochemical differences in children with ADHD. We wonder why these differences continue to be ignored, and why followup studies have difficulty being funded (Ward and Swanson, personal communication). 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. *Note: High copper would indicate low zinc. See the Ward studies, page 28.*

**Oades 1998** ★ Over 2 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.”

**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 tested ... and fixed.*

**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 Foodlist and Shopping Guide.*

**Barnes** 1998 – Even high dose corticosteroids do not control 5% of patients with asthma. 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 anti-inflammatory drugs in some aspirin-sensitive people.

**Devereux** 2006 – Since 1960, the prevalence of asthma and allergic disease has increased sufficiently to become a major public-health concern. Concurrently, there have been marked changes in the Western diet, and it has been proposed that these changes have contributed to the increase in the prevalence of asthma and allergy, with the most recent evidence indicating that maternal diet during pregnancy might be particularly important in the development of childhood asthma.

**Devereux** 2006 – In a longitudinal study, 1,861 children were followed from conception to 5 years old. The mother’s intake of foods containing vitamin E and zinc during pregnancy is strongly associated with the child’s risk of experiencing wheeze and asthma at age 5.

**Egger** 1983 ★ 93% of 88 children with frequent migraine recovered on the “few foods” additive-free diet. Other symptoms which improved included abdominal pain, behavior disorder, seizure, asthma, and eczema.

**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 5 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 zinc deficiency is a problem in ADHD, Ward determined that exposure to synthetic colorings cause children with ADHD to lose zinc, and Arnold found that children given zinc respond better to stimulant medications, while fatty acid supplements seem to improve symptoms of ADHD in cases of borderline zinc deficiency. It is known that many children with ADHD also have asthma. Will artificial coloring-induced zinc loss worsen their asthma? Do children with asthma alone also lose zinc upon exposure to colorings? These studies have not been done.*

**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.

**Jimenez-Aranda** 1996 – Yellow #5 was the most reactive additive in a study of 40 patients with chronic urticaria.

**Juhlin** 1981 ★ In 330 patients with recurrent urticaria, 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 patients with chronic urticaria (*hives*), author suggests looking for adverse reactions to food additives.

**Kalinke** 1999 ★ A 58 year old patient had progressive pigmented purpura (*called PPP, it is brown pigmentation of the skin spreading from the legs upward*). Controlled oral provocation testing revealed that food containing Yellow #5 triggered flares of the PPP. This case was followed for over 20 years.

**Litonjua** 2006 – Dietary and supplement intakes of 1290 pregnant women were studied. Higher intake of vitamin E and zinc by the mother was related to less wheezing in the child at 2 years. Authors conclude that consuming more antioxidants during pregnancy may decrease the risk of wheezing for the baby.

**Lockey** 1977 – Lockey developed tests for diagnosis of sensitivity, and created a diet at the Mayo Clinic for urticaria and asthma patients. *Note: This diet later was used and further refined by Dr. Feingold, who called it 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 “oligoantigenic” diet, their eosinophil count went down, and improvement of symptoms followed.

**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 – “Pseudo-allergic” 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 (AIA) is important because: 1) It is caused by inhibition of an enzyme; 2) It affects 9.8% of asthmatic adults; 3) AIA patients also have chronic sinusitis, nasal polyps, and inability to smell; 4) Some medications make AIA worse; 5) Some patient are sensitive to Yellow #5, sodium benzoate, parabens etc; 6) It can be fatal; 7) AIA will be less severe if correctly diagnosed and given appropriate medical treatment. *Note: Some asthma drugs contain these additives.*

- Sloper** 1991 ★ **74%** of 66 patients with eczema improved on a diet eliminating colors, preservatives, milk, eggs, and tomatoes. The longer a food had been avoided, the less likely was the chance of a positive food reaction. 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, it was found that some foods, food additives, tyramine, and acetylsalicylic acid can cause skin, intestinal, and respiratory reactions.
- Veien** 1991 ★ A severe eruption of leukocytoclastic vasculitis (*blood vessel inflammation*) occurred 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 lower zinc levels, causing one or more of the following symptoms: Overactivity, aggression, violence, poor speech, poor coordination, asthma, eczema.
- Warrington** 1986 – In patients with chronic additive-induced urticaria or aspirin-sensitive asthma, Yellow #5 causes the release of a chemical from certain cells which suggests a potential diagnostic test.
- 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 ★ Adverse reaction to aspirin or food additives is called intolerance or pseudoallergy, and related to prostaglandin imbalance. 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. Wuthrich said that drugs must no longer be synthetically colored. *Note: Now, more than 25 years later, they still are.*
- 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.

	<u>Vaccinated Children</u>	<u>Unvaccinated Children</u>
Asthma	25.6%	2.3%
Atopic Dermatitis	18.0%	2.3%
Asthma, Rhinitis, or Dermatitis	56.4%	9.3%

*Note: Since children who become more prone to allergic, autoimmune, or behavioral disorders after vaccination often 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, G.I. PAIN, SEIZURES: 93%** of 88 children with frequent migraine recovered on the “few foods” diet without additives. Other symptoms which improved included abdominal pain, behavior disorder, fits, asthma, and eczema.

- Egger 1985** – HEADACHE, G.I. PAIN, SEIZURES: **81.6%** of 76 overactive children improved on a few-foods diet without additives. Other symptoms such as headaches, abdominal pain, and seizures also improved.
- Egger 1989** ★ HEADACHE, G.I. PAIN, SEIZURES etc: **80%** of 45 children with epilepsy plus headaches, abdominal symptoms, or hyperactivity improved on an elimination diet. Symptoms improved in all those whose seizures stopped, and in some whose seizures did not stop. In double-blind challenge, symptoms returned in 15 of 16 children (**94%**)”
- Egger 1992** ★ MIGRAINE, ENURESIS: On a diet without additives, **76%** of 21 children whose migraine or hyperactive behavior had improved also stopped bed wetting.
- Faulkner-Hogg 1999** – CELIAC DISEASE: 39 adults who continued to have symptoms on a gluten-free diet were studied. Of 22 patients who switched from an almost-gluten-free to a no-detectable-gluten diet, 5 became well and 10 improved. Diarrhea, headache, nausea, and flatulence were provoked by amine, salicylate and soy, as well as gluten. *Note: Salicylate is addressed by the Feingold Diet, while both salicylate and amines are addressed by the Failsafe Diet. See [www.fedupwithfoodadditives.info](http://www.fedupwithfoodadditives.info)*
- Feingold 1979** – EYE PROBLEMS: Eye muscle disorders may respond well to the Feingold Diet. In addition, benzoates – both naturally occurring and those used as preservatives – should be eliminated. Dr. Feingold proposed that 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.” He said nystagmus and strabismus should not be ignored. Unfortunately, no further research on this has been found.
- 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 showed a reversible decrease in activity within 24 hours. Murphy says, “The Ketogenic Diet may be of use in the treatment of ADHD.” *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 would be interesting. See Lau 2006, page 33.*
- Neuman 1978** ★ ALLERGY: This was a randomized, controlled, clinical trial. 122 patients with allergies ate 50 mg Yellow #5 or placebo. They suffered the following reactions from the coloring: 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. *Note: 50 mg is not a high dose of Yellow #5.*
- 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 ...”
- Petitpierre 1985** ★ IBS: 14 patients with irritable bowel syndrome got better on an elimination diet. Then they were challenged “blindly” (they did not know when they received challenge and when it was a placebo). The challenges with foods or additives caused the typical symptoms of Irritable Bowel Syndrome. 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.
- Salzman 1976** – SLEEP & ENURESIS: **93%** of 15 children given the Feingold Diet improved in overactivity, distractibility, impulsiveness and excitability. Sleep and enuresis (*bed-wetting*) problems were resolved partially or completely

## Colorings and Flavorings

**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
- ❖ The balance between types of white blood cells was abnormal

### 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
- ❖ Damage your nerves

**Allen** 1984 – For most people with food intolerance, symptoms are 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.

**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.

**D'Souza** 1987 – Aspirin, Indomethacin and Yellow #5 (0.1-2.0 mg/kg) induced dose-dependent increases in carotid-sinus nerve (CSN) 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 (U.S.)** – “The color certification program is self-supporting because the law requires manufacturers to pay FDA a user fee for each pound of color the agency certifies.” **Note:** *They get paid per pound PASSED, not per pound EXAMINED - certainly a conflict of interest. In 2006 FDA certified almost 19 million pounds of color additives.*

**Food & Drug Administration (U.S.)** – The colorings are not certified to be *safe*. FD&C colors are certified to have no more than the following amounts of contaminants such as the following:

- ❖ Benzidine, not more than 1 part per billion. (*See Lancaster study, page 33.*)
- ❖ Lead, not more than 10 parts per million.
- ❖ Arsenic, not more than 3 parts per million.
- ❖ Mercury, not more than 1 part per million.

- Food & Drug Administration Public Health Advisory** 2003 – Letter warning physicians of blue discoloration of the skin, urine, and feces, of metabolic acidosis and death when Blue #1 is used to color enteral (*tube feeding*) solutions. FDA says that since Blue #1 is a mitochondrial toxin, it is “plausible but not proven” as the cause. FDA says Blue #1 has been used for tube feeding for 30 years but never evaluated for safety.
- Groten** 2000 – Combining unrelated additives is not a health concern because of the low doses involved. Authors claim no actual research is necessary. *Note: This report was copyrighted by the International Life Sciences Institute, formerly known as the Nutrition Foundation, and composed of the major food, chemical, and pharmaceutical companies, listed in part on page 20.*
- Hedman** 1981 – Tiny amounts of Yellow #5 cause contractions in the trachea smooth muscle tissue of guinea pigs.
- Koutsogeorgopoulou** 1998 – Results showed clear immuno-suppressive effects of Red #2 and Yellow #5.
- Kroes** 2000 – Describes the Threshold of Toxicological Concern and **de minimis** concepts (“*a little bit can’t hurt*”) used to evaluate additives. Thus, the cost and time needed to actually test additives for safety can be avoided.
- Kroes** 2002 – Using the Threshold of Toxicological Concern, a de minimis value can be set for chemicals of unknown toxicity. This method is now used by the US FDA and the WHO for evaluations of flavoring substances. ILSI (*International Life Sciences Institute, composed of companies that make food additives, pesticides, snack foods, etc.*) is heavily promoting this new method of “safety evaluation.”
- Lancaster** 1999 – FDA allows only 1 part per billion (1 ng/g = ONE 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 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 Blue #1 + MSG, and Yellow #10 + Aspartame. The combinations were synergistic, far more toxic than expected by adding up the effect of each one tested alone. Blue #1 + MSG was *4 times as toxic*, and Yellow #10 + Aspartame was *7 times as toxic*. *Note: Although Yellow #10 is not used in snacks in the U.S., it is commonly used in medications, cosmetics, etc.*
- National Academy of Sciences** 1979 – From 2 weeks of data on 12,000 people, the NAS determined that 99% of people eat up to an average of **327 mg** food dye per person per day. For reasons unknown, they divided this number by 5, to set the average daily intake of food dyes at 65 mg per day.
- Reyes** 1996 – All food dyes tested inhibit mitochondrial respiration. The 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 of the food dyes caused DNA damage in the mouse stomach, colon and bladder.
- 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 ★ 2 generations of mice were fed low doses of Red #2. The pups weighed more, had trouble turning over and finding a source of smell. Movement was affected, and more pups died.
- Tanaka** 1996 ★ Yellow #6 was fed to 2 generations of mice. There were some (unspecified) adverse effects on litter size, weight, and sex ratio. The pups had trouble with surface righting (*turning over*), negative geotaxis (*crawling upwards*), swimming direction, and swimming head angle. Their difficulty was dose-related.
- Tanaka** 2001 ★ Red #3 was fed to 2 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 conclusion of safety is based on nothing but conjecture.*
- Tsuda** 2001 ★ Very low doses of 3 azo food dyes caused DNA damage in the colon, lung, bladder, etc., when fed to mice. Damage was observable as early as three hours after they ate it. Tsuda says, “more extensive assessment of azo additives is warranted.”
- Vorhees** 1983 ★ When rats ate Red #40, it reduced reproductive success, parent and pup weight, brain weight, survival, and female vaginal development. Running wheel activity decreased, and open-field rearing activity increased. Red #40 produced physical and behavioral toxicity in pups at high doses (10%).
- Ward** 1990 ★ Yellow #5 reduced zinc in blood and saliva, and increased urinary zinc 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 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.

## The Three Preservatives

- 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 who had responded well to a diet removing synthetic additives and tyramine. *Note: This study was done in Australia, where much more calcium propionate is used in bread than in the U.S. While not eliminated by the Feingold Program, products containing this preservative are marked with a (CP) symbol in the Foodlist books.*
- Fisherman** 1973 ★ 250 mg BHT in food caused an asthma attack within 75 minutes in some asthmatic patients.
- Kahl** 1983 – Feeding rats BHT increases some chemicals but decreases others in hepatic (*liver*) microsomes.

- Kahl** 1984 – Studying the action of BHA and BHT on cells and organs, Kahl was hoping they may protect against cancer. Although they do protect against radiation and have anti-tumor actions, their use in the prevention of human cancer was judged “unlikely” in light of their ability to promote tumors themselves.
- Kahl** 1993 – The toxicology of BHA, BHT, and vitamin E (*alpha-tocopherol*) is described. At high doses all antagonize vitamin K and interfere with blood clotting. BHT is toxic to the lungs and causes liver tumors. BHA causes tumors of the forestomach. Kahl says all published findings agree that BHA and BHT are tumor promoters, but vitamin E is not carcinogenic and is safe to use in higher doses.
- 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, there was a significant negative effect on body weight in both generations, and developmental problems in the pups, starting during the lactation period.
- NIH Eleventh Report on Carcinogens** 2005 – BHA is “reasonably anticipated to be a human carcinogen based on sufficient evidence of carcinogenicity in experimental animals ... No data were available to evaluate the carcinogenicity of BHA in humans.” *Note: They have been saying this every year since 1986.*
- 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*). These findings were described in clinical and gruesome detail.
- Sarafian** 2002 – Exposure to marijuana smoke and BHA is 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 – BHT has adverse effects on the liver of rats. Like tobacco and many drugs, it is metabolized by *cytochrome P450* in the liver, where it becomes a harmful pro-oxidative compound instead of the antioxidant it is supposed to be.
- Stokes** 1974 ★ 0.5% BHA or BHT was fed to pregnant mice and their offspring. Compared to controls, BHA-treated pups explored more, and slept, groomed, and learned less. BHT-treated pups slept less, learned less, and were more aggressive.
- Stolze** 1999 – The combination of BHA and TBHQ was shown to cause harmful effects on 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 3 generations. At the lowest (0.015%) level, body weight of the pups was increased at birth and during lactation for each generation. A few neurobehavioral parameters (e.g., turning over and crawling uphill) were affected at all levels.
- Thompson** 1988 – BHA interacts with BHT in the lungs of mice by stimulating formation of hydrogen peroxide which increases the ability of BHT to bind to protein. Both of these things directly injure 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 up to a dose of 500 mg/kg. However, when added to small amounts of BHT, the BHA significantly increased the lung weight in a dose-dependent manner.

**Thompson** 1988 – In rat liver mitochondria, BHA and BHT inhibited respiratory control of cells by stimulating state 4 respiration. They also affected the mitochondrial membrane, causing calcium release and mitochondrial swelling. There was a rapid decrease in ATP (*energy source*) levels and then cell death.

**Thompson** 1989 – Like BHA, 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 – 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 (NK) cell activity of splenocytes (*cells in spleen that kill invaders*). **Note:** *This means BHT affects the immune system.*

**Yu** 2000 – The proposed use of BHA as a 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, it was seen that BHA and BHT strongly inhibit certain processes important to calcium ion depolarization of GABA and glutamate neuron transmission.

## 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 and Shopping Guide 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 cane sugar with no problem.*

**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. In some patients, it causes chronic diarrhea or other bowel problems. 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.

Children who eat a lot of “sugar” are probably eating a lot of corn syrup. 20% of Feingold members report a sensitivity to corn syrup.

Some other names for corn syrup are: Dextrose, Glucose, Corn Sweetener, High Fructose Corn Syrup, Maltodextrin, and Corn Syrup Solids.

**Hallfrisch** 1990 – When HFCS was introduced in 1967, it was recommended as a replacement for “regular” sugar for diabetic and obese people. Although HFCS causes a smaller increase in blood glucose and insulin than sugar does, there are a number of undesirable changes that don’t show up immediately. It is absorbed from the small intestine and metabolized in the liver. When eating more fructose than glucose, it may be malabsorbed. It 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**

*Sugar is allowed on the Feingold Program. Sugar not labeled “cane sugar” is usually made from beets. Some people who appear to be sugar-intolerant may be reacting to chemicals used in the manufacture of beet sugar. They may do better with cane sugar. Honey can sometimes be a salicylate, depending on the type of blossom the bees visited, but is usually well tolerated.*

**Inam 2006** ★ Serotonin is a neurotransmitter important in mood, stress, and attention. One group of rats was fed a standard rat diet, while another group was fed a standard diet with 25% table sugar for 5 weeks. Both groups were then tested with a medication that would indicate serotonin response. The study showed that sugar induced a change in the serotonin receptor’s ability to receive messages both before and after the synapse (*space between neurons*).

*Note: Keep this study in mind for the child with continuing behavior problems even on the Feingold Program. Too many children do get over 25% of their calories from sugar or – even worse – from corn syrup.*

**Wolraich 1994** ★ Children whose parents said they were “sugar-sensitive” were tested with a series of 3 diets: One with sugar, one with aspartame, and one with saccharin. None of these sweeteners had an adverse effect on the children’s behavior. 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 3 diets were without artificial food colorings, flavorings, and preservatives, this improvement is not surprising.*

*Note: Most candy and soda contains CORN SYRUP – not table sugar – but this study did not test corn syrup.*

This study is often quoted to “prove” that parents are poor judges, and that sugar is not harmful to behavior.

However, this 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.

**ASPARTAME**

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

**Butchko 2002** – “It is clear that aspartame is safe, and there are no unresolved questions regarding its safety under conditions of intended use.” *Note: This study was done by the NutraSweet Company.*

**Lau 2006** – Food coloring + aspartame was found to be synergistic, i.e., far more toxic to developing neurons than expected by just adding up the effect of each additive given alone.

**Maher 1987** – Some people suffer neurological or behavioral reactions to aspartame. If mice are given enough aspartame to elevate plasma phenylalanine levels more than tyrosine levels (which happens in humans), seizures are more easily induced.

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 it was known that aspartame can trigger brain tumors. He said, **“If the FDA violates its own laws, who is left to protect the public?”**

**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. More than 6 times that concentration has been measured in humans given large doses of aspartame.

**Roberts 2001**– Aspartame-induced disorders in children include headache, confusion, convulsions, irritability, depression, intellectual deterioration, antisocial behavior, rashes, asthma and unstable diabetes, as well as actual addiction to aspartame-containing products.

## OTHER ARTIFICIAL SWEETENERS

Coming under the heading of “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 and Shopping Guide 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. In order to be measured by the spoonful like sugar, Splenda is bulked up with corn syrup solids, and contains *almost* 5 calories per teaspoon (legally but not truthfully called “zero”).

Feingold members who had used Splenda were asked for input, and reported that Splenda had caused the following symptoms: Racing heart, stomach ache, head banging and crying (in an autistic child), asthma attack, depression, increased yeast infection problems, and memory loss (in an adult). It is not known whether these effects were caused by the sucralose or the corn syrup component.

Other sweeteners include Tagatose (from milk), Trehalose (from starch), Acesulfame potassium, Neohesperidine DC (from oranges). “Natural” artificial sweeteners from plant proteins are being developed. So far, The Feingold Association has no opinion on whether any of these sweeteners are safe to use for people on the Feingold Program. At this time, no products containing them are listed in the Foodlist and Shopping Guides

## AGAVE

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

## STEVIA

Stevia is allowed on the Feingold Program.

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)

## ALCOHOL SUGARS

Alcohol sugars are allowed on the Feingold Program. Care should be taken not to overdo them, since too much has a laxative effect.

When a sugar name ends in “ol” that means it is an alcohol sugar. Commonly used alcohol sugars: Sorbitol, Mannitol, Xylitol, Polyols (from hydrogenated starch hydrolysates)

## PST / Sulfation Pathways

**Alberti** 1999 ★ Autistic children showed a low level of sulfation, indicating difficulty in detoxifying or metabolizing certain compounds.

**Bamforth** 1993 ★ Yellow #5 and artificial *vanillin* flavoring inhibit the enzyme dopamine sulfotransferase. Vanillin also inhibits by 50% 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.

**Harris** 1998 – Low doses of salicylic acid (*aspirin*) consistently and selectively inhibited the P form of the enzyme phenol sulfotransferase (PST) by **50%**. *Note: Thus, if a child is already low in this enzyme, salicylate would make it worse.*

**McFadden** 1996 – People with environmental intolerance or chronic disease may have impaired sulfation, related to intolerance of phenol and tyramine containing foods. “It may be a factor in the success of the Feingold Diet.” *Note: The Feingold Diet does not eliminate tyramine, but the Australian Failsafe Diet does.*

**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.

## Animal Research – Additives, Behavior and Neurology

**Carrie** 2002 ★ An omega-3 supplement improved learning ability and vision in old mice whether fed a balanced diet or an omega-3 deficient diet. *Note: This is not part of the basic Feingold Program, but research has shown this essential fatty acid to be a helpful addition to everyone’s diet.*

**Meyer** 1980 ★ When BHT was given to pregnant rats, it had a negative effect on body weight in both generations, and pups had developmental problems.

**Ruppert** 1985 ★ A single exposure to the metals cadmium or tin produced hyperactivity in the rat pups. Authors conclude these metals are neurotoxic to the developing nervous system.

**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 slept less, were more aggressive, with learning difficulties.

**Tanaka** 1993 ★ Red #2 was fed to mice. The pups weighed more, had trouble turning over, finding a source of smell, and more died. Movement activity of male pups was affected.

**Tanaka** 1993 ★ BHT was fed to mice for 3 generations. In the group that got the least (0.015%), body weight of the pups increased. Turning over and crawling uphill were abnormal in all the treatment groups, but Tanaka nevertheless concluded that these doses had “little effect” on the mice.

**Tanaka** 1996 ★ When 2 generations of mice ate Yellow #6, the pups weighed more and had dose-related difficulty with surface righting (*turning over*), negative geotaxis (*crawling upwards*), and swimming.

**Tanaka** 2001 ★ 2 generations of mice ate Red #3. Movement and other changes were dose-related.

**Tanaka** 2005 ★ 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 (*Yellow #5*) is presumed to be much lower. It would therefore appear that the level of 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 ★ Red #40 was fed to rats for 2 generations. It reduced reproductive success, brain weight, survival, and female vaginal development. Running wheel activity decreased, and rearing activity (*standing up*) increased. Authors say that “Red #40 produced physical and behavioral toxicity in developing rats.”

Exposing mice and rats to some toxins damages their GABA neurons (the “brakes” of the nervous system), resulting in HYPERACTIVITY.

Can some food additives also damage GABA neurons? Shall we rev up these neurons with stimulant medication, or try to stop the damage by avoiding the food additives?

**NOTE:** Ruling out toxic metal exposure should be part of the diagnostic workup for children with behavior problems. See [hripte.org](http://hripte.org) for more information.

## Reviews of Research

**Anthony** 1999 – An elimination diet is effective in most cases, and 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.

**Baumgaertel** 1999 – Scientific evidence suggests that individualized dietary management and trace element supplementation is effective in some children.

**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.”

**Jacobson** 1999 – After reviewing 25 years of research, the *Center for Science in the Public Interest* (CSPI) recommends NIH-sponsored research on additives, FDA testing of additives for behavioral effects, and an FDA ban on use of synthetic dye in products for children. Also, CSPI says the FDA must stop denying that food additives contribute to ADHD, and advise the public that methylphenidate (Ritalin etc) causes cancer and is a poor first choice for treating ADHD. The CSPI also says that fast food chains, hospitals, summer camps and schools should make their meals without food dyes.

**Kavale** 1983 – In a meta analysis of early studies in the 1970’s on artificial colorings only, they concluded that the diet has no significant benefit. Unfortunately, this old analysis continues to be quoted by ADHD experts.

**Kellogg Report** 1989 – This 735 page report funded by the Kellogg and Ford foundations looks at health in the United States. 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.” They conclude that “what Americans need most of all is an instinctive preference for whole foods and a healthy sense of suspicion about processed foods.”

**Kidd** 2000 – Major contributors to ADHD include adverse responses to food additives and foods, sensitivity to 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.

**Rimland** 1983 – Invited by publishers to comment on the Mattes, Kavale & Forness reviews of the early studies, Rimland concludes “GIGO = garbage in / garbage out.” He makes the following points:

- 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 of these compounds are in use 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 – The increase in ADHD raises the possibility of a widespread risk factor. In a new meta-analysis of all appropriate double-blind placebo-controlled trials evaluating the effects of AFCs (*artificial food colors*), Schab found they were consistent with accumulating evidence that “neurobehavioral toxicity may characterize a variety of widely distributed chemicals.” **Note:** *AFCs are only part of what is eliminated by the Feingold Program.*

**Schnoll** 2003 – Schnoll concluded that food additives, refined sugars, food sensitivities/allergies, and fatty acid deficiencies are all linked to ADHD, and that diet modification should be part of the treatment protocol.

**Schnyder** 1999 – Adverse reactions to foods may be caused by toxic, enzymatic, pharmacological, “pseudo-allergic” or allergic mechanisms. Diagnosis can usually be based on the history and results of a diet.

**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.*

## LIST OF CITATIONS

1. **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.
2. **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.
3. **Alberti** A, et al. 1999. Sulphation Deficit in "Low-Functioning" Autistic Children: A Pilot Study. *Biological Psychiatry* 1999 Aug 1;46(3):420-4.
4. **Allen** DH, et al. 1984. Adverse Reactions to Foods. *Medical Journal of Australia* 1984, Sep 1; 141 (5 Suppl): S37-42.
5. **Anthony** HM, Maberly DJ, Birtwistle S. 1999. Attention Deficit Hyperactivity Disorder, *Archives of Disease in Childhood* 1999;81:189 (August).
6. **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.
7. **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.
8. **Arai** Y, et al. 1998. Food and Food Additives Hypersensitivity in Adult Asthmatics. III. Adverse Reaction to Sulfites in Adult Asthmatics, *Aerugi* 1998 Nov; 47 (11); pp.1163-7.
9. **Arnold** LE, 1999. Treatment Alternatives for Attention-Deficit/Hyperactivity Disorder (ADHD), *Journal of Attention Disorders*, Vol. 3 No. 1 (April 1999), 30-48.
10. **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.
11. **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.
12. **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.
13. **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.
14. **Barnes** PJ & Woolcock AJ, 1998, Difficult Asthma, *European Respiratory Journal.* 1998 Nov; 12(5); pp.1209-18.
15. **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.
16. **Baumgaertel** A, 1999. Alternative and Controversial Treatments for Attention-Deficit/Hyperactivity Disorder, *Pediatric Clinics of North America.* 1999 Oct;46(5):977-92.
17. **Bauer** AK, et al, 2001. Butylated Hydroxytoluene (BHT) Induction of Pulmonary Inflammation: A Role in Tumor Pro-motion, *Experimental Lung Research.* 2001 Apr-May;27(3):197-216.
18. **Bauer** AK, et al. 2005. Toll-like Receptor 4 in Butylated Hydroxytoluene-Induced Mouse Pulmonary Inflammation and Tumorigenesis, *Journal of the National Cancer Institute.* 2005 Dec 7;97(23):1778-81.
19. **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.
20. **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.
21. **Berdonces** JL, 2001. Attention Deficit and Infantile Hyperactivity, *Revista de Enfermeria* 2001 Jan; 24 (1): 11-4.
22. **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.
23. **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.
24. **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.
25. **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.
26. **Brenner** A, 1979. Trace Mineral Levels in Hyperactive Children Responding to the Feingold Diet, *Journal of Pediatrics* 1979 Jun;94(6):944-5.

27. **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.
28. **Butchko** et al, 2002. Aspartame: Review of Safety, NutraSweet Company, *Regulatory Toxicology and Pharmacology* 2002 Apr;35(2 Pt 2):S1-93.
29. **Cade** R et al. 2000. Autism and Schizophrenia: Intestinal Disorders. *Nutritional Neuroscience*, March 2000.
30. **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.
31. **Carrie** I, et al. 2002. Docosahexaenoic Acid-Rich Phospholipid Supplementation: Effect on Behavior, Learning Ability, and Retinal Function in Control and n-3 Polyunsaturated Fatty Acid Deficient Old Mice, *Nutritional Neuroscience*. 2002 Feb;5(1):43-52.
32. **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.
33. **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.
34. **Ceserani** R, et al. 1978. Tartrazine and Prostaglandin-System, *Prostaglandins and Medicine*. 1978 Dec;1(6):499-505..
35. **Chang** JC, 2005. Increase of Insulin Sensitivity by Stevioside in Fructose-Rich Chow-Fed Rats, *Hormone & Metabolic Research*, 2005 Oct;37(10):610-6.
36. **Cockell** KA; Bonacci G; Belonje B. 2004. Manganese Content of Soy or Rice Beverages is High in Comparison to Infant Formulas. *Journal of the American College of Nutrition*, 2004 Apr;23(2):124-30.
37. **Connors** CK, et al. 1976. Food Additives and Hyperkinesis: A Controlled Double-Blind Experiment. *Pediatrics* 1976 Aug;58(2):154-66.
38. **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.
39. **Devereux** G, 2006a. The Increase in the Prevalence of Asthma and Allergy: Food for Thought. *Nature Reviews, Immunology*, 2006 Nov;6(11):869-74.
40. **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. Epub 2006 Jun 8.
41. **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.
42. **Dumbrell** S, 1978. Is the Australian Version of the Feingold Diet Safe? *Medical Journal of Australia*. 1978 Dec 2;2(12):548, 569-70.
43. **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.
44. **Egger** J, et al. 1985. Controlled Trial of Oligoantigenic Treatment in the Hyperkinetic Syndrome. *The Lancet*, March 9, 1985.
45. **Egger** J, et al. 1989. Oligoantigenic Diet Treatment of Children with Epilepsy and Migraine. *Journal of Pediatrics* 1989 Jan; 114(1): 51-8.
46. **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.
47. **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.
48. **El-Zein** RA, et al, 2005. Cytogenic Effects in Children Treated with Methylphenidate. *Cancer Letters*, 2005 Dec 18; 230(2):284-91.
49. **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.
50. **Feingold** BF, 1979. Dietary Management of Nystagmus. *Journal of Neural Transmission*, 1979, Vol. 45 (2), pp. 107-115.
51. **Feingold** BF, 1982. The Role of Diet in Behaviour. *Ecology of Disease*. 1982. 1(2-3) pp.153-65.
52. **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.
53. **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.
54. **Food & Drug Administration (U.S.)** – Color Additives Fact Sheet. [www.cfsan.fda.gov/~dms/cos-221.html](http://www.cfsan.fda.gov/~dms/cos-221.html)
55. **Food & Drug Administration (U.S.)** – Report on the Certification of Color Additives. [www.cfsan.fda.gov/~dms/col-06-4.html](http://www.cfsan.fda.gov/~dms/col-06-4.html)
56. **Food & Drug Administration** - Public Health Advisory 2003, [www.cfsan.fda.gov/~dms/col-ltr2.html](http://www.cfsan.fda.gov/~dms/col-ltr2.html) Reports of Blue Discoloration and Death in Patients Receiving Enteral Feedings Tinted with the Dye, FD&C Blue No. 1.
57. **Food & Drug Administration** October 2004 Press Release re new Black Box Warning mandated for antidepressants. [www.fda.gov/bbs/topics/news/2004/NEW01124.html](http://www.fda.gov/bbs/topics/news/2004/NEW01124.html)
58. **Food & Drugs**, Title 21 – Part 74 – Food & Drug Administration Listing of Color Additives Subject to Certification. [www.access.gpo.gov/nara/cfr/waisidx\\_99/21cfr74\\_99.html](http://www.access.gpo.gov/nara/cfr/waisidx_99/21cfr74_99.html)
59. **Gaby** AR, 2005. Adverse Effects of Dietary Fructose. *Alternative Medicine Review*, 2005 Dec;10(4):294-306.
60. **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.
61. **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
62. **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.
63. **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.
64. **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. *Journal of the American Academy of Child and Adolescent Psychiatry*. 1987 Jan;26(1):53-5.
65. **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.
66. **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.
67. **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.
68. **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.
69. **Harley**, JP et al. 1978. Hyperkinesis and Food Additives: Testing the Feingold Hypothesis. *Pediatrics*, 1978. June Vol 61 (6) p. 818-827.
70. **Harper** PH, Goyette CH, Connors CK, 1978. Nutrient Intakes of Children on the Hyperkinesis Diet. *Journal of the American Dietetic Association*. 1978 Nov;73(5):515-9.

71. **Harris RM**, Waring RH, 1996. Dietary Modulation of Human Platelet Phenol-sulphotransferase Activity. *Xenobiotica*. 1996, Dec; 26 (12): 1241-7.
72. **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.
73. **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.
74. **Henderson TA**, Fischer VW, 1995. Effects of Methylphenidate (Ritalin) on Mammalian Myocardial Ultrastructure. *American Journal of Cardiovascular Pathology*. 1995;5(1):68-78.
75. **Hong SP** et al. 1989. Oral Provocation Tests with Aspirin and Food Additives in Asthmatic Patients. *Yonsei Medical Journal*, 1989. Dec.30(4); pp.339-45.
76. **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.
77. **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.
78. **Inam QU**, Haleem MA, Haleem DJ, 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.
79. **Jacobson MF**, Schardt D, 1999. *Diet, ADHD & Behavior: A Quarter-Century Review*, publ.1999 by Center for Science in the Public Interest, Washington, DC.
80. **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.
81. **Juhlin L**, 1981. Recurrent Urticaria: Clinical Investigation of 330 Patients. *British Journal of Dermatology*, 1981 Apr;104(4):369-81.
82. **Juhlin L**, 1987. Additives and Chronic Urticaria, *Annals of Allergy* 1987 Nov;59(5 Pt 2):119-23.
83. **Kahl R**, Kahl GF, 1983. Effect of Dietary Antioxidants on Benzo[a]pyrene Metabolism in Rat Liver Microsomes. *Toxicology* 1983;28(3):229-33.
84. **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.
85. **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.
86. **Kalinke DU**, Wuthrich B, 1999. Purpura Pigmentosa Progressiva in Type III Cryoglobulinemia and Tartrazine Intolerance. A Follow-up Over 20 years. *Hautarzt* 1999 Jan;50(1):47-51.
87. **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.
88. **Kaplan B** et al. 1989. Dietary Replacement in Preschool-Aged Hyper-active Boys. *Pediatrics*, 1989, Vol. 83, pp. 7-17.
89. **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.
90. **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
91. **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.
92. **Kidd PM**, 2000. Attention Deficit/Hyperactivity Disorder (ADHD) in Children: Rationale for its Integrative Management. *Alternative Medicine Review* 2000 Oct; 5 (5): 402-28.
93. **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.
94. **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.
95. **Kroes R**, Kozianowski G, 2002. Threshold of Toxicological Concern (TTC) in Food Safety Assessment. *Toxicology Letters* 2002 Feb 28;127(1-3):43-6.
96. **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
97. **Lancaster FE**, Lawrence JF, 1999. Determination of Benzidine in the Food Colours Tartrazine (FD&C Yellow #5) and Sunset Yellow FCF (FD&C Yellow #6). *Food Additives and Contaminants*, 1999 Sep;16(9):381-90.
98. **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; [Epub ahead of print].
99. **Levy F**, et al. 1978. Hyperkinesia and Diet: A Double-Blind Crossover Trial with a Tartrazine Challenge. *Medical Journal of Australia* 1978 Jan 28;1(2):61-4.
100. **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.
101. **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.
102. **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
103. **Lockey SD**, 1977. Hypersensitivity to Tartrazine (FD&C Yellow No. 5) and Other Dyes and Additives Present in Foods and Pharmaceutical Products. *Annals of Allergy*, 1977 Mar; 38(3); pp.206-10.
104. **Longo G**, et al. 1987. Food Allergy in Asthma. Diagnostic Significance of Peripheral Eosinophils. *Pediatrics Medica e Chirurgica*, 1987 Nov-Dec;9(6):663-8.
105. **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.
106. **Maher TJ**, Wurtman RJ, 1987. Review. Possible Neurologic Effects of Aspartame, a Widely Used Food Additive. *Environmental Health Perspective* 1987 Nov;75:53-7.
107. **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.
108. **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.

109. **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, Sep 7 published online.
110. **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.
111. **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.
112. **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.
113. **Nakao H**, et al. 2003. Formaldehyde-Induced Shrinkage of Rat Thymocytes. *Journal of Pharmacological Sciences*, 2003 Jan;91(1):83-6.
114. **National Academy of Sciences**, 1977 Survey of Industry on the Use of Food Additives, published 1979.
115. **Neuman I**, et al. 1978. The Danger of "Yellow Dyes" (Tartrazine) to Allergic Subjects. *Clinical Allergy*. 1978 Jan;8(1):65-8.
116. **Niederhofer H**, Pittschieler K, 2006. A Preliminary Investigation of ADHD Symptoms in Persons with Celiac Disease. *Journal of Attention Disorders*. 2006 Nov;10(2):200-4.
117. **NIH Eleventh Report on Carcinogens 2005**, [ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s027bha.pdf](http://ntp.niehs.nih.gov/ntp/roc/eleventh/profiles/s027bha.pdf)
118. **Nsouli TM**, et al. 1994. Role of Food Allergy in Serous Otitis Media. *Annals of Allergy* 1994 Sep;73(3):215-9.
119. **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.
120. **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.
121. **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.
122. **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 Geneesk* 2002 Dec 28;146(52):2543-7.
123. **Petitpierre M**, Gumowski P, Girard JP, 1985. Irritable Bowel Syndrome and Hypersensitivity to Food. *Annals of Allergy* 1985 Jun; 54(6):538-40.
124. **Pollock I**, Warner JO, 1990. Effect of Artificial Food Colours on Childhood Behavior. *Arch Dis Child* 1990 Jan;65(1):74-7.
125. **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.
126. **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.
127. **Roberts HJ**, 2001. *Aspartame Disease: An Ignored Epidemic*, West Palm Beach: Sunshine Sentinel Press. 1018 p.
128. **Robson WL**, et al. 1997. Enuresis in Children with Attention-Deficit Hyperactivity Disorder. *Southern Med Journal* 1997 May;90(5):503-5.
129. **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.
130. **Rowe KS**, 1988. Synthetic Food Colourings and "Hyperactivity": a Double-Blind Crossover Study. *Australia Paediatric Journal*, April 1988, Vol. 24 (2), pp. 143-7.
131. **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.
132. **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.
133. **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.
134. **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.
135. **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.
136. **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.
137. **Sarafian TA**, et al. 2002. Synergistic Cytotoxicity of Delta(9)-tetrahydrocannabinol and Butylated Hydroxyanisole. *Toxicology Letters* 2002 Jul 21;133(2-3):171-9.
138. **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.
139. **Scadding GK** et al. 1988. Poor Sulphoxidation Ability in Patients with Food Sensitivity. *British Medical Journal*, 1988 Jul 9; 297 (6641): 105-7.
140. **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.
141. **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.
142. **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.
143. **Schnyder B**, et al. 1999. Food Intolerance and Food Allergy. *Schweiz Med Wochenschr*, 1999 Jun 19; 129(24): 928-33.
144. **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
145. **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.
146. **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.
147. **Schoenthaler SJ**, 1985. Institutional Nutritional Policies and Criminal Behavior, *Nutrition Today*, 1985; 20(3); 16.
148. **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.
149. **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.
150. **Schoenthaler S**, Moody J, Pankow L, 1991. Applied Nutrition and Behavior. *Journal of Applied Nutrition*, November 1, 1991, Vol. 43.

151. **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.
152. **Sinaiko** RJ 1996. The Biochemistry of Attentional/Behavioral Problems. [www.diet-studies.com/sinaiko.html](http://www.diet-studies.com/sinaiko.html)
153. **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.
154. **Spencer** PS, Bischoff JC, 1984. Skin as a Route of Entry for Neurotoxic Substances. *Dermatotoxicology* (1984) 3<sup>rd</sup> Ed.p.629-630 Wash. DC
155. **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.
156. **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.
157. **Swain** AR, Dutton SP, Truswell AS, 1985. Salicylates in Foods. *Journal of the American Dietetic Association* 1985 Aug;85(8):950-60.
158. **Swain** A, Soutter V, Loblay R, Truswell AS. 1985. Salicylates, Oligoantigenic Diets, and Behaviour. *The Lancet*, 1985 Jul 6;2(8445):41-2.
159. **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.
160. **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.
161. **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.
162. **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.
163. **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.
164. **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.
165. **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.
166. **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.
167. **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.
168. **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.
169. **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.
170. **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.
171. **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.
172. **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.
173. **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.
174. **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.
175. **Van Bever** HP, Docx M, Stevens WJ, 1989. Food and Food Additives in Severe Atopic Dermatitis. *Allergy* 1989 Nov;44(8):588-94.
176. **Veien** NK, Krogdahl A, 1991. Cutaneous Vasculitis Induced by Food Additives. *Acta Dermato-Venereologica* 1991;71(1):73-4.
177. **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.
178. **Walsh** WJ, et al. 1997. Elevated Blood Copper/Zinc Ratios in Assaultive Young Males. *Physiology & Behavior*, 1997 Aug;62(2):327-9
179. **Wang** GJ, et al. 1994. Methylphenidate Decreases Regional Cerebral Blood Flow in Normal Human Subjects. *Life Sci*. 1994;54(9):143-6.
180. **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.
181. **Ward** NI, 1997. Assessment of Chemical Factors in Relation to Child Hyperactivity. *Journal of Nutritional & Environmental Medicine (Abingdon)*; 7 (4). 1997. 333-342.
182. **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.
183. **Weiss** B, et al. 1980. Behavioral Responses to Artificial Food Colors. *Science*, 1980, Vol. 207, 1487-1489.
184. **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.
185. **Williams** JI, et al. 1978. Relative Effects of Drugs and Diet on Hyperactive Behaviors: An Experimental Study. *Pediatrics*. 1978 Jun;61(6):811-7.
186. **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.
187. **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.
188. **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.
189. **Yoneyama** H, et al. 2000. The Effect of DPT and BCG Vaccinations on Atopic Disorders. *Alerugi* 2000 Jul;49(7):585-92.
190. **Yu** R, Mandlekar S, Kong AT, 2000. Molecular Mechanisms of Butylated Hydroxyanisole-Induced Toxicity: Induction of Apoptosis Through Direct Release of Cytochrome C. *Molecular Pharmacology*. 2000 Aug;58(2):431-7.
191. **Zoccarato** F, et al. 1987. Inhibition by Some Phenolic Antioxidants of Ca<sup>2+</sup> Uptake and Neurotransmitter Release from Brain Synaptosomes. *Biochemical & Biophysical Research Communications*. 1987 Jul 31;146(2):603-10.