

The Feingold Diet: An Assessment of the Reviews by Mattes, by Kavale and Forness and Others

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In 1973 at a meeting of the American Medical Association, the distinguished pediatrician and allergist Ben Feingold reported that food additives were responsible for the hyperactivity in 40 to 50% of the hyperactive children he had seen in his practice. Feingold had no idea that his report would raise the storm of controversy that ensued and did not imagine that the remaining nine years of his life would be spent in defending what his critics choose to refer to as "Feingold's theory" or "Feingold's hypothesis." He quickly found himself at odds with both the medical establishment and the packaged food industry.

A number of studies have been done in an attempt to verify (or vilify) Feingold's report. It is not surprising that the completion of these studies has failed to resolve the controversy, because Feingold's critics and supporters tend to view the evidence differently.

The preceding papers by Mattes and by Kavale and Forness have reviewed a substantial proportion of the evidence bearing on the value of the Feingold additive-free diet. The editor invited this comment. Although originally stimulated by the Mattes and the Kavale and Forness reviews, the comments also bear upon the other similar reviews which have appeared recently, such as those by The Nutrition Foundation (1980), the American Council on

Science and Health (Whelan, Stare & Sheridan, 1980) and the National Institutes of Health (Consensus Development Conference Statement 1982).

All of these reviews of research on the Feingold Diet come to essentially the same conclusion: The Feingold diet is of no value, or, at best, of marginal value for a few children, as a means of reducing hyperactivity. In my opinion, such a conclusion is certainly unwarranted, probably incorrect, and very likely damaging.

Why do I come to this unkind and critical assessment of the diligent effort of so many colleagues? The answer can be expressed in one simple four letter word: "gigo." Gigo, in computerese, stands for "garbage in, garbage out." That is, if the incoming data are of no value, no amount of massaging, analysis, or manipulation will increase its value.

How can I justify applying the term gigo to a body of data it has taken nearly a decade to collect? Let me count the ways:

1. Most of the studies are nearly irrelevant. Feingold called our attention to the fact that over 3,000 additives had been

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placed in our food supply with absolutely no testing for behavioral effects. Such substances include colors, flavors, preservatives, thickeners, moisteners, and about eight other categories of additives. He felt it was important, even after the fact, to do behavioral testing. But where does one begin? In a 1975 meeting in Glen Cove, New York, sponsored by the Nutrition Foundation, he recommended *as a starting point* that a few artificial colors be the focus of the first studies. A deluge of studies on food dyes and hyperactivity followed. In 1981, Feingold protested, "I recommended that, in view of the complexity of the problem and the many compounds involved, studies be designed focusing upon the limited list of colors, which lend themselves to better control. This statement is repeatedly distorted by the Nutrition Foundation to imply that I implicate colors as the most important factor. Neither I nor anyone else has the data today to support such a contention." How researchers can claim they have tested "the Feingold diet," which eliminates over 3,000 additives, by conducting experiments on fewer than 10 dyes, is beyond me.

2. **The dosage levels were ridiculously small.** Even if one were to accept the wholly unwarranted conclusion that seven to 10 food colorings were the overwhelming important factor in the Feingold diet, one would still have to reject the bulk of the studies, since the researchers used almost trivially small doses of colorings in trying to provoke hyperactivity in the children. By and large, the studies used doses of 1.6 to 26 milligrams of colorings per day, 26 mg/day being the Nutrition Foundation's estimate of the per capita daily consumption of these colorings. However, the FDA, making a more objective analysis of the data, found the daily consumption of colorings to be 59 mg/day for children ages one to five and 76 mg/day for children six to 12 years old. The 90th percentile consumption figures were 121 mg/day to age five and 146 mg/day for the six to 12 year olds. Maximum consumption was estimated at 312 mg/day. No wonder the studies "disproved" Feingold's hypothesis! When Swanson and Kinsbourne (1980) used colorings at about 90th percentile values, the effects were clearly supportive. Could you be convinced that handguns

were not lethal by studies using popguns to test the lethality hypothesis?

3. **Failure to recognize role of subject nutritional status.** Some investigators have used as subjects children who had been on the Feingold diet, with purportedly good effects, before they were "challenged" by the experimentally provided food colors. Other investigators have alternated Feingold diets with challenge diets containing additives. The researchers then conclude the challenge to have provoked no effects, or lesser effects, than Feingold had reported. But children who have been on the Feingold diet for a time tend to be healthier than run-of-the-mill hyperactive children who have not been on the Feingold diet and are thus more able to withstand the food additive challenge. The reason is that the Feingold diet tends to keep the child from consuming sugary, non-nutritious "junk food." The child consequently increases his intake of genuine food containing the vitamins, minerals, amino acids and other nutrients necessary for proper functioning of the brain.

The adverse effects of a junk food diet on hyperactivity and learning disabilities was dramatically illustrated in a study by Kershner and Hawke (1979). Hyperactive children placed on a no junk food diet for six months improved greatly on scales measuring hyperactivity, attention span, irritability, discipline, "getting into things," and peer-relations (all at the $p < .01$ level) and "talks too much" and speech problems (at the .02 and .05 levels, respectively). Regrettably, Kershner and Hawke failed to use a control group of hyperactive children who continued to eat junk food for the six months, so this finding might be questioned, but anyone familiar with this area of research can testify that such marked improvement is not typically found in untreated children over a six month period. If it were, the pharmacological industry would be much less profitable. (Kershner and Hawke had started out to evaluate megavitamin therapy, but the no junkfood diet brought about such great improvement in both groups that the children given extra vitamins had little hyperactivity left to correct.) Prinz, Roberts and Hantman (1980), like Kershner and Hawke, found sugar-loaded additive-containing foods to be associated with high levels of hyper-

activity in their double blind study of dietary correlates of hyperactivity. Lester, Thatcher and Monroe-Lord (1982) found significant negative relationships between four of their five cognitive measures and the proportion of refined carbohydrate foods in the diets of their sample of 184 school children.

Obviously, research on the Feingold diet which fails to take into account the differences between Feingold diet-fed children and normally malnourished hyperactive children is likely to produce only confusing, confounded results.

4. Failure to recognize and control relevant variables. As is evident from the foregoing point, the world is a complicated place, and a lack of appreciation for the many factors involved may lead researchers to an invalid conclusion. Brenner (1979), for example, has reported that artificial colorings and flavorings cause hyperactivity only in children with high blood copper levels. On comparing 20 nine year-olds who benefited from the Feingold diet (parent reports) with 14 nine year-olds who did not benefit, despite close adherence to the diet, Brenner found the difference in their copper levels, 142.8 vs 114.8 mg/dl, to be highly significant ($p < .025$). There are many possible explanations for this finding (e.g., the copper may form a neurotoxic compound with an additive; it may deplete a protective substance; it may increase the intestinal absorption of the additives, etc.), but the actual mechanism is unknown. As a result of acid rain and other factors, different geographic areas differ greatly in the copper content of food and water supplies, and a Feingold diet study carried out in a low copper area may thus lead to conclusions not generalizable to other areas. Even within the same geographic area, some homes use copper water pipes while others do not, so different degrees of responses to the additives are to be expected.

Copper ingestion is just one of a host of known and unknown complicating factors. Fluorescent lighting in the schoolroom may be another confounding factor. As Mayron, Ott, Nations and Mayron (1974), among others, have shown, fluorescent lighting can increase the hyperactivity of school children. Feingold (1975) cites a study conducted at the University of West Virginia in which fruit flies exposed to

fluorescent light after being fed yellow food dye died of hyperactivity-caused exhaustion.

Copper ingestion and polluted light are merely examples. In view of our appalling ignorance of such matters, it behooves us to be cautious indeed in proclaiming a study to have proven Feingold wrong because the "Feingold effect" was reportedly not observed in a given setting.

5. Arbitrary negative conclusions. Despite the pronouncements of negative outcomes by the reviewers, the data themselves are by no means so unfavorable to Feingold. In discussing the Harley study — the very large and costly program sponsored by the makers of Coca Cola, Fruit Loops, C & H Sugar, etc., through their Nutrition Foundation — an editorial in the September 22, 1979, issue of prestigious *Lancet* reported, "In the preschool group, however, all 10 mothers and seven of the fathers rated their children as improved on the additive-free diet. Harley and his colleagues admit to unease about their discrepant findings, but, although they offer several reasons for preferring the negative conclusions of the school-age group, their data remain strongly in favor of some dietary effect on the behavior of certain preschool children."

I might add, in further reference to the Harley study, that I have in my possession a letter sent by Dr. Harley to a Feingold Society mother (January 24, 1977) in which Harley proudly explains that "the children made approximately one to two dietary infractions a week during our study." Since it takes up to four days for the effects of such an infraction to wear off, Dr. Feingold, who sent me a copy of the Harley letter, was understandably chagrined. And, of course, not all infractions are dietary. None of the researchers inform us as to whether the children had taken cough syrups, other medications, or children's vitamins, many of which contain Feingold-prohibited additives.

Despite the anti-Feingold bias so evident from the above *Lancet* quote and in many of the studies and reviews, all studies, without exception, do concede that some children do respond to the diet. In view of the weaknesses in the design and conduct of the studies, and the insensitive and subjective

behavioral measures typically employed, these findings speak strongly for the robustness of the Feingold effect.

6. Inadequate attention to animal and in vitro studies. Unlike school children, laboratory rats cannot trade a tuna sandwich for a Twinkie, or drink an illicit Kool-Aid on the way home from school. Thus animal studies can be quite revealing. Nevertheless, the reviews pay scant, if any, attention to animal studies, and to in vitro studies, of additives. The study by Goldenring, Wool, Shaywitz, Batter, Cohen, Young, and Teicher (1980) reported 163% more activity and 128% greater failures in avoidance learning in rat pups given small amounts of food dyes, as compared to controls. The dosage level was equated to the average US per capita intake for humans. Similarly, the many test-tube studies showing food colorings to damage nerve tissue, such as those by Augustine and Levitan (1980) and Lafferman and Silbergelb (1980), are all but ignored by the reviewers.

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

Who needs artificially colored and flavored food anyway? For millennia the human body — and mind — has evolved and thrived on real food. It is prudent to feed our children, and ourselves, real food, not the denatured, "refined," additive-laden artificial foods that emanate from factories. What is the cost to us, to our country, and to our civilization of allowing ourselves to be seduced into consuming the gaudy colors and deceptive flavors that are used to make non-nutritious food appear desirable?

Weiss (1982) cites David P. Rall as posing an intriguing question: Suppose that thalidomide, rather than inducing structural deformities, had instead depressed IQ scores by 10%; would we ever have suspected it of adverse

effects? The answer is all too obvious. We are all very much aware of a sharp decline in academic ability in our youngsters, including the 17 year drop in SAT scores. We are also aware of an enormous upsurge in youth crime during these decades. While there are a multitude of proposed alternative causes for these continuing disasters (e.g., Rimland and Larson, 1981), let us heed the insights and warnings of the prophetic Ben Feingold and remove the unnecessary pollutants from our food supply. Prudence demands no less.

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