Hyperkinesis and Learning Disabilities Linked to the Ingestion of Artificial Food Colors and Flavors

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The historical background of hyperkinesis and learning disabilities (H-LD) is reviewed briefly and followed by a discussion of food additives. Focus on artificial food colors and flavors as important etiologic agents is explained and supported by the favorable response of 30 to 50% of various samples of H-LD children managed with the Kaiser-Permanente (K-P) Diet.

The hyperkinetic syndrome, commonly labeled "hyperactivity," has captured public attention in practically every developed country. Whether this public interest represents a greater awareness of the problem or reflects an actual increase in incidence is an often debated question. However, among those who deal with children, particularly educators and teachers, as well as thousands of beleaguered parents, the consensus favors not only a real increase in the incidence of hyperkinesis but a prevalence of epidemic proportions affecting approximately 5,000,000 children in this country.

The increased frequency with which hyperkinesis is encountered has led to a common impression that the condition is a new syndrome, although it is perhaps as old as man. A search of the literature discloses clinical patterns described as early as 400 BC (Benton 1964-65) that were quite similar to those currently labeled as "hyperkinesis" or "minimal brain dysfunction" (MBD) in which hyperactivity is absent or not prominent. During the 18th, 19th, and early 20th centuries terms such as *tanzruh* (dance mania), fidgety Philip (Hoffman 1864), St. Vitus' dance (Abt 1929), and chorea minor (Gibson 1937) were often applied to clinical descriptions closely similar to the hyperkinetic syndrome or MBD.

The epidemic of encephalitis that occurred during World War I left in its wake a number of cases with true brain damage characterized by signs and symptoms that are now identified with hyperkinesis. Following the collation of the clinical experience of this period by Cohen and Kahn in 1934, it became common practice to label all patients presenting a similar clinical pattern as having brain damage (BD). Abetted by the reports of Strauss and his collaborators (Strauss & Lehtinen 1948, Money 1962, Strauss & Kephart 1955), the practice of labeling individuals as organically brain damaged without adequate substantiating evidence continued into the 1950s.

In the early 1960s, to mitigate the stigma associated with the diagnosis of brain damage, the word "minimal" was introduced. Later the diagnostic term was further tempered by substituting "dysfunction" for "damage"; this led to the commonly encountered term, "minimal brain dysfunction." In 1957 Laufer and Denhoff suggested the term "hyperkinetic

TABLE I. Etiologies in neurologic damage.

During Pregnancy
Toxemia of mother (eclampsia)
Hemorrhage
Infection
Drugs
Environmental toxicants (air, food, water)
During Delivery
Anesthesia
Trauma
Post Partum
Respiratory distress
Jaundice
Infection
Environmental toxicants

impulse disorder" which, through usage, has been abbreviated to "hyperkinesis" and frequently to "hyperactivity."

During the last 50 years, a considerable literature has accumulated which reports attempts to categorize the kaleidoscope of signs and symptoms representing this condition into specific clinical entities (Bax & MacKeith 1963). Depending upon the orientation of the observer and the dominant characteristic presented when the patient was examined, numerous labels were invented for variations of the identical problem (Clements 1966). Many times the descriptive classification carried with it a proposal for treatment and management, as well as hypotheses suggesting the etiology or underlying biological disorder.

A number of etiologies have been proposed for hyperkinesis (see Table I). Causal factors include toxemia, hemorrhage, and drugs during pregnancy; anesthesia causing asphyxia neonatorum and trauma during delivery; psychological and emotional factors; environmental pollutants of the air, water, and food supply.

One of the most widespread and critically important, yet not fully recognized, group of pollutants in the environment is food additives. Food additives may be classified as intentional or nonintentional. Nonintentional food additives are the chemicals and substances that accidently gain entrance into the food supply – e.g., insect parts; animal hairs and feces; soil, water, and air pollutants; packaging materials, etc. Intentional additives represent the chemicals that are deliberately introduced into the food supply for specific functions or purposes.

The classification of intentional food additives in Table II lists 13 categories consisting of 2,764 compounds compiled from data gathered by the National Science Foundation in 1965. This is not a complete list. The precise number of intentional additives may approach 3,800 or even 4,000. The exact number is not known.

Of all additives introduced into foods, synthetic colors and flavors are perhaps the most common. By virtue of this, synthetic

*TABLE II. Classification of intentional additives.**

Preservatives	33
Antioxidants	28
Sequestrants	45
Surface active agents	111
Stabilizers, thickeners	39
Bleaching and maturing agents	24
Buffers, acids, alkalies	60
Food colors	34
Nonnutritive and special dietary sweeteners	4
Nutritive supplement	117
Flavorings – synthetic	1610
Flavorings – natural	502
Miscellaneous: yeast foods, texturizers, firming agents, binders, anticaking agents, enzymes	157
Total number of additives	2764

**Compiled from data gathered by the National Science Foundation in 1965*

TABLE III. Adverse reactions induced by flavors and colors.

1. Respiratory
   - Rhinitis
   - Nasal polyps
   - Cough
   - Laryngeal edema
   - Hoarseness (laryngeal nodes)
   - Asthma
2. Skin
   - Pruritus
   - Dermatographia
   - Localized skin lesions
   - Urticaria
   - Angioedema
3. Gastrointestinal
   - Macroglossia
   - Flatulence and pyrosis
   - Constipation
   - Buccal chancres
4. Neurological Symptoms
   - Headaches
   - Behavioral disturbances
5. Skeletal System
   - Arthralgia with edema

colors and flavors are the most common cause of adverse reactions, affecting practically every system of the body (Table III). It is because of this comparatively high frequency of reactions attributed to the added colors and flavors that we have focused our attention upon these two classes of additives. This does not imply that the remaining categories do not cause adverse reactions. No chemical is exempt, since any compound in existence, whether natural or synthetic, may induce an adverse reaction if its consumer has the appropriate genetic profile, i.e., predisposition. This being true, it becomes essential to evaluate each compound or class of compounds for its benefit compared with the risk associated with its use.

In addition to being the most common cause of adverse reactions, the synthetic colors and flavors have no nutritional value. Their function is purely cosmetic, so that deleting them from the food supply would cause no significant loss. Accordingly, on balance, the risk associated with synthetic colors and flavors outweighs their benefits.

Of all observed reactions to such compounds, perhaps the most dramatic and most critical are the behavioral disturbances. Initially, it may be surprising that food additives can cause behavioral disturbances. Closer analysis allays surprise. Except for terminology, there is no difference between certain compounds when they are used as medicines or when they are introduced into foods as additives. Both are low molecular weight compounds. The availability of behavior-modifying drugs is common knowledge. There are drugs that stimulate, drugs that depress, and others that modify the subject's mood. It is not remarkable that among the thousands of additives in the food supply there may be compounds with similar effects upon behavioral and emotional patterns.

It is surprising indeed to recognize that none of the thousands of chemicals introduced into food as additives has ever been subjected to pharmacological studies such as those that are required for a compound before it can be licensed for use as a drug (Schmidt 1975). Certainly, there is little knowledge of the behavioral toxicology of these additives.

The patient who first attracted my attention to the possibility of a link between behavioral disturbances and the ingestion of artificial food colors and flavors was a 40-year-old woman who reported to the Allergy Department because of angioedema of the face and periorbital region (Feingold 1973). Her food intake was restricted according to the K-P Diet (Table IV) and her angioedema cleared. During the initial interview, the patient had failed to report that she had been in psychotherapy for two years because of a behavioral disturbance characterized by hostility toward her husband, inability to socialize with her peers, and conflict with her coworkers. While she adhered to the K-P Diet, her behavior improved. She also noted that any infraction of the diet induced an immediate recurrence of both the angioedema and the disturbed behavioral pattern.

Having been alerted to a possible link between food additives and behavior, we observed other adults with a similar association, and also children with the apparent same relationship. Since the children were reporting to the Allergy Department, their primary complaints were somatic -- e.g., pruritus, urticaria, angioedema, localized skin lesions, nasal

TABLE IV. The Kaiser-Permanente (K-P) Diet.

Omit the following, as indicated:

I. Foods containing natural salicylates

Almonds
Apples (cider & cider vinegars)
Apricots
Blackberries
Cherries
Cloves
Cucumbers and pickles
Currants
Gooseberries
Grapes or raisins (wine & wine vinegars)
Mint flavors
Nectarines
Oranges
Peaches
Plums or prunes
Raspberries
Strawberries
All tea
Tomatoes
Oil of wintergreen

The salicylate-containing foods may be restored following 4 to 6 weeks of favorable response provided no history of aspirin sensitivity exists in the family.

II. All foods that contain artificial colors and flavors

III. Miscellaneous items

All aspirin-containing compounds
All medications with artificial colors and flavors
Toothpaste and toothpowder (substitute salt and soda or unscented Neutrogena® soap)
All perfumes

Note: Check all labels of food items and drugs for artificial coloring and flavoring. Since permissible foods without artificial colors and flavors vary from region to region, it is not practical to compile a list of permissible foods. Each individual must learn to read the ingredients on the label. When added colors and flavors are specified, the item is prohibited. If in doubt, the food should not be used. Instead, it is advisable to prepare the substitute at home from scratch.

symptoms, and at times gastrointestinal complaints. Early in the course of these observations, none of the parents volunteered information that a child was experiencing behavioral disturbances, often associated with problems at school. After the K-P Diet was ordered for treatment of the physical complaint, the parents would report not only control of the physical problem, but also a marked change in the child's behavioral pattern (Feingold 1975).

To test whether the observations of the parents would be confirmed, we arranged for management by the K-P Diet of children whose primary complaint was a behavioral disturbance, usually labeled MBD or hyperkinesis. Using the Conners Rating Scale (Conners 1969), ratings were made prior to the initial visit and periodically following dietary management, initially at 2-week and then at 4-week intervals. We were soon able to confirm the parents' earlier reports. Children with a history of signs and symptoms* usually leading to a diagnosis of MBD or hyperkinesis, when managed with the K-P Diet, experienced a marked change in behavioral pattern within 3 to 21 days, depending upon the age of the child. Children who had been receiving various behavior-modifying drugs could discontinue these agents, while the behavioral pattern continued to improve. When rated by teachers on a quarterly or semester basis, children who had had difficulty at school showed a marked adjustment to the classroom environment and

**The history developed at the initial visit covered all developmental periods – prenatal, perinatal, infancy, nursery school, kindergarten, elementary and secondary school. For older patients, performance before and after puberty was stressed.*

rapid improvement in scholastic achievement. Any dietary challenge, inadvertent or deliberate, induced a recurrence of the behavioral disturbance which persisted for 24 hours to four days, so that a child experiencing an infraction only twice a week could have a persistence of the clinical pattern.

A double-blind crossover study funded by the National Institute of Education and directed by Dr. C. Keith Conners of the University of Pittsburgh (Conners, Goyette, Southwick, Lees, & Andrulonis 1976) has confirmed that dietary management favorably influences hyperactivity at the .005 level of significance on a teacher-rating scale and at .05 on a parent-rating scale. The subjects of this study initially comprised 57 children who were reviewed in advance of the investigation. Through attrition, chiefly failure to comply with the structure of the study, the group was finally reduced to 15 children who fulfilled all the requirements of the protocol. Five of the 15 children demonstrated unequivocally that dietary management influenced hyperactivity as long as there was full compliance with the diet. Any infraction or challenge was followed within hours by a recurrence of the behavioral pattern.

The Food Research Institute (1976) of the University of Wisconsin conducted a double-blind crossover study on 36 boys of school age (6 to 12 years) and 10 children who were three to five years of age. In the school age group, four children in the sample showed significant improvement as rated by both parents and teachers and/or on several of the objective measures employed. The younger children (age 3 to 5) showed a greater positive response to the experimental diet as indicated by parent rating. All ten mothers in this group rated their child's behavior as improved as did four of the seven fathers in this sample.

The numerous variables of the hyperkinetic syndrome coupled with the many environmental variables do not permit valid statistical conclusions on the basis of the short-term, segmental observations employed in both the Conners and the Wisconsin study. These studies merely confirmed that the K-P Diet influences behavior.

All our clinical observations have been replicated in a pilot clinical study (Cook & Woodhill 1976) in Australia, directed by a psychiatrist with the Sydney Ministry of Health, and the chairperson of Prince Henry Hospital's Department of Nutrition at Little Bay, New South Wales.

Both the Department of Health, Education, and Welfare in this country and the Medical Research Council of Australia are funding further studies of the problem.

RESPONSES TO MANAGEMENT WITH THE K-P DIET

Five separate programs, representing a total of 360 children managed with the K-P Diet, showed favorable responses ranging from 30 to 50% of the sample, depending upon the mean age of the children and the presence or absence of a history suggestive of neurologic damage. Precise determination for the percentage of responders to dietary management will require large samples, perhaps 1,000 subjects or more, studied longitudinally over a period of several years. At this level, it is important to recognize that dietary intervention does influence the behavioral deficits of the hyperkinetic syndrome and, particularly, hyperactivity.

All of the deficits associated with the hyperkinetic or MBD syndrome listed in Table V are not observed in every child. Not only does each child have his own mosaic of deficits, but for any given child, the pattern may vary from day to day, and at times even from hour to hour. Hyperactivity is usually the dominant feature of the pattern, but it is not always present. One child may exhibit features of only a single group listed in Table V; in other children, various combinations of deficits drawn from one or more of the three groups may characterize the behavioral pattern. At times only a single deficit may be observed. However, if this deficit is a critical one -- e.g., an auditory perceptual or a visual perceptual disturbance -- severe learning disabilities may result.

Although we have observed the response to dietary management for five years, we are still unable to predict from history, physical examination, and neurologic and psychometric tests the ultimate response of the individual. Similarly, assumptions regarding the speed and

TABLE V. Descriptive characteristics of clinical pattern of H-I.D.

GROUP I

Marked Hyperactivity and Fidgetiness
- Constant motion
  - Rocks and jiggles legs
  - Dances, wiggles hands
  - Runs, does not walk

In infancy, crib rocking, head knocking, fretfulness

Compulsive Aggression
- Disruptive at home and at school
- Compulsively touches everything and everyone
- Disturbs other children
- Perseverates – Cannot be diverted from an action even when life threatening

Excitable – Impulsive
- Behavior is unpredictable
- Panics easily
- Frustration leading to temper tantrums

No Patience
- Low tolerance for failure and frustration
- Demands must be met immediately

Short Attention Span
- Unable to concentrate

Poor Sleep Habits
- Difficult to get to bed
- Hard to fall asleep
- Easily awakened

GROUP II

Gross Muscle Incoordination
- Exceptionally clumsy
  - Trips when walking
  - Collides with objects
- Cannot function in sports
- Cannot bicycle or swim

Fine Muscle Incoordination
- Eyes and hands do not seem to operate together
- Difficulty with: buttoning and tying
  writing and drawing
  speech - stuttering
  reading - dyslexia

GROUP III

Cognitive and Perceptive Disturbances
- Auditory memory deficits
- Visual memory deficits
- Deficits in understanding
- Difficulty in reasoning, e.g., a math problem

Normal or high IQ but fails at school

Boys involved 7:1

Rarely more than one child in a family affected

degree of response to dietary management cannot be made on the basis of estimates of neurologic damage, previous use of medicines, or age of the child. Children with a history suggesting a possible cause for neurologic damage may experience a complete recovery on the diet, while others with a completely negative history may fail to respond, or may show a partial response, such as improved behavior with deficits in coordination, or cognition, or perception. The history cannot always be precise in disclosing neurotoxic factors. Not infrequently the mother, relying upon memory, cannot accurately reconstruct the events before or during pregnancy. In addition, consideration must be given to less overt factors, such as environmental pollutants of air, soil, and water, which serve as neurobehavioral toxicants during gestation or early childhood.

A determination can be made only through strict application of the diet.

When a favorable response follows dietary management, the initial improvement is in the behavioral pattern (Table V, Group I). Control of aggression, impulsiveness, and the tendency to perseverate results in a calmer child; the improvement in mood enables the child to concentrate, which leads to improved attention. If there are no cognitive or perceptual deficits, there is rapid improvement in learning. The child who was abusive, disobedient, incorrigible, and disdainful of attention moves toward becoming affectionate, lovable, and responsive to guidance.

Correction of the behavioral pattern may be followed rapidly by improved muscular coordination (Table V, Group II). Improvement of the gross muscle involvement corrects awkwardness in gait and permits participation in sports, such as swimming, ball games, and bicycle riding. Improvement of the fine muscle coordination leads to improved writing and drawing skills and, in some children, improved speech.

Cognition and perception (Table V, Group III) are next to respond; improvement in these permits increased scholastic achievement. Learning ability may show a slow improvement over months or even years. An improved behavioral pattern always precedes the correction

in coordination, cognition, and perception. The latter deficits do not improve unless behavior responds to the diet. Cognitive and perceptual deficits are those that persist most commonly, causing learning disabilities even after a marked improvement in behavior and muscle coordination.

Age influences the speed and degree of response to dietary management. Usually, the younger the child, the more rapid and more complete the response. In early infancy improvement or reversal of all signs and symptoms may occur within 24 to 48 hours after elimination of pediatric vitamin drops, a rich source of synthetic colors and flavors. The two- to five-year-old child may improve after five days, while the five- to 12-year-old child may respond in 10 to 14 days. In some children, particularly those who have received long-acting drugs, e.g., dextroamphetamine Spansules® or large doses of behavior-modifying drugs [amphetamine, methylphenidate (Ritalin®), Stelazine®, Mellaril®, Tofranil®, Elavil®, Vistaril®, Cylert®], the response may be delayed for 3 to 5 weeks. Very often, following such delays the improvement may occur abruptly rather than develop gradually.

The older child, postpubescent or adolescent, usually requires a longer period, frequently several months, before improvement is noted; even then, the response is not always complete. The highest incidence of failure to respond to dietary management is observed among patients treated during adolescence.

As the child passes through puberty, a spontaneous improvement in the behavioral pattern may be observed. If the deficits persist, the adolescent cannot perform to his full potential. This makes it difficult for him to cope with his environment, leading to frustration resulting in withdrawal, antisocial behavior, lying, stealing, and ultimately, in many cases, juvenile delinquency.

BEHAVIORAL TOXICOLOGY

The behavioral disturbances with learning disabilities attributed to the ingestion of artificial food colors and flavors represent a small band in the broad spectrum of the newly emerging discipline of behavioral toxicology. Accordingly, the variety of clinical patterns observed can best be interpreted relative to some considerations that are basic to this discipline.

Since molecules of almost any substance can cross the placental barrier, it must be recognized that any environmental compound, whether ingested, inhaled, or injected, has the potential of being toxic to the fetus. The developing organism does not have the same capacity as a fully developed person to metabolize and detoxify potentially toxic substances. Accordingly, the fetus, particularly during the stage of organ differentiation, is highly susceptible to the insults of any substance crossing the placental barrier.

The teratogenic damage which may be manifest as either overt or covert alterations in the organism is governed by the genetic profile of the individual, the nature and doses of the offending compound, and the stage of organ development at the time of the insult. It is conceivable that substances of low toxicity or in small doses can induce covert alterations in the organism which can later manifest as behavioral disturbances without gross functional impairment or structural birth defects. The overt damage to organs and to the nervous system is usually obvious, and its patterns are well known.

It is not generally recognized that the disturbances caused by such alterations are not necessarily obvious at birth, but emerge as the child grows older. Nor is it commonly appreciated that teratogens which have an affinity for developing brain centers may induce subtle alterations which may manifest in later life as behavioral disturbances and learning disabilities. In 1968 Nair and Dubois reported that a morphological or biochemical lesion may remain dormant and not be manifest as a behavioral disorder or functional impairment until later life. This may explain the delayed onset of the behavioral disturbance and learning disabilities so frequently noted when the history suggests possible intrauterine damage.

Relative to the mode of action of artificial food colors and flavors, there are two possibilities to consider. The food additives may play a primary role as the sole etiologic agent. Or, they may serve as irritants superimposed

upon a substratum created by any one of the commonly cited causes of neurologic damage (Table I). In either situation, the toxicants may be ingested by the mother during pregnancy or encountered by the patient during extrauterine life.

Although it is possible that the artificial colors and flavors ingested by the mother may play a primary role in the induction of teratogenic alterations, there is as yet no supporting evidence for this concept. The primary extrauterine role is suggested by the complete reversal of all deficits, both behavioral and learning, following the elimination of the colors and flavors from the diet. The completeness of the favorable response and the ability to induce a rapid recurrence within hours indicate that this is a functional disturbance.

In addition to the functional disturbances induced by the artificial colors and flavors, it is conceivable that irreversible neurologic damage may result, particularly from continued exposure to the chemical over many years. Such damage could explain the persistence of various degrees of muscle incoordination and learning disabilities when a dramatic improvement in the behavioral pattern follows dietary management. Since the higher association centers are the last to differentiate, they are the most susceptible to neurotoxicants. In the human, these centers are not fully developed at birth and are ready targets for damage which can be manifest as the hyperkinetic syndrome. It is conceivable that the high incidence of failure to respond among adolescents may be attributed to irreversible neurologic damage.

As secondary agents acting upon pre-existing neuropathology, the synthetic colors and flavors can produce a variety of clinical patterns – e.g., the hyperkinetic syndrome, seizures labeled as petit mal, mental retardation, and learning disabilities. The secondary role is suggested by a history positive for neurologic damage attributed to other causes, and improvement of various degrees in response to dietary management.

For example, the elimination of colors and flavors may be followed by improved behavior but persistence of muscle incoordination with perceptual and cognitive deficits. Seizures may be controlled without the use of drugs; but muscle incoordination and learning ability improve only partially or fail completely to respond. In retardation the clinical response may be dramatic, as evidenced by improved behavior, better coordination of both fine and gross muscles, and improved learning ability. All of these gains induce a marked transformation in the patient, whose expression becomes more alert and bright, his social adjustment improves, permitting him to function as a self-sufficient person who does not require one-to-one attention or instruction. In most patients labeled as "retarded," however, the level of learning ability usually remains below the normal estimated for age.

At times, residuals may persist with nothing in the history to suggest a cause. Behavioral toxicology is not yet sufficiently developed to provide guidelines for correlation of behavioral patterns and learning disabilities with all of the potential neurotoxicants in the ecosystem.

CONCLUSION

Artificial food colors and flavors have the capacity to induce adverse reactions affecting every system of the body. Of all these adverse reactions, the nervous system involvement, as evidenced by behavioral disturbances and learning disabilities, is the most frequently encountered and most critical, affecting millions of individuals in this country alone.

The K-P Diet, which eliminates all artificial food colors and flavors as well as foods with a natural salicylate radical, will control the behavioral disturbance in 30 to 50% (depending upon the sample) of both normal and neurologically damaged children. – *Allergy Department, Kaiser-Permanente Medical Center, 2200 O'Farrell Street, San Francisco, Calif. 94115.*

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news

THE VALIDITY AND reproducibility of the subcutaneous provocative food test were not confirmed by a double-blind study, according to a report presented to the American Academy of Allergy. Results of the study, conducted by Dr. L.V. Crawford, chairman of the Academy's Food Committee, revealed that fewer than half of the patients who reacted positively to an initial subcutaneous injection of possibly allergenic food extract yielded positive results when retested. Of the 61 patients participating in the study, more than half reacted positively to saline placebos, and 12 patients reacted positively to both placebo injections. Results also revealed that of 21 patients with allergic rhinitis and asthma, but with no history of food sensitivity, oral challenge tests were negative, although 14 of these patients had at least one positive subcutaneous food test. Of 22 patients who had clinical histories of food sensitivity and positive oral challenge tests, 20 had one or more positive subcutaneous food tests, but 16 of these patients also reacted to at least one placebo. Of nine patients with allergic rhinitis, asthma, and with histories of food allergies, eight reacted positively to subcutaneous food tests but had negative oral challenge tests, and six reacted positively to placebos.

THE "FEINGOLD FAMILIES" are newly active. Parents who have seen their hyperactive children improve or return entirely to normal on the additive-free Feingold diet have organized a national association. One of the purposes of the group is to persuade food processors to provide more complete and detailed labeling on their products. Another purpose is to encourage the national use of a logo or design which would identify foods that are free of artificial flavors and colors and make shopping easier. The Feingold group will answer inquiries and send essential information to any interested party. Contact the Feingold Association of the United States, 759 National Press Building, Washington, D.C. 20045.