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THE ROLE OF DIET IN BEHAVIOUR

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The behavioural disorders, frequently labelled hyperkinesis, hyperkinetic impulse disorder, hyperactivity, Minimal Brain Dysfunction (MBD) and Attentional Deficit Disorder (ADD), are among the most critical problems of our contemporary culture.

Truancy, vandalism, violence and assault among school children coupled with a persistent drop in scholastic achievement is a universal problem affecting the school population of every so-called developed country. Every procedure for the control of behavioural disorders has not been successful; every technique for the improvement of learning has not been productive, while every modality for the rehabilitation of delinquents has failed us. Since all these procedures have been structured upon psychosocial concepts, it becomes necessary to look elsewhere for the answers, which is to the biosciences, including genetics, molecular genetics, pharmacogenetics, behavioural toxicology, behavioural teratology, immunochemistry, immunology, allergy and endocrinology, with a focus upon nutrition, which encompasses all these disciplines.

The various behavioural disorders are not new. They are as old as the human race, having always been with us and perhaps always will be with us, since they appear to be an expression of the process of evolution—the process of selection and adaption as enunciated by Darwin and Wallace in the mid-nineteenth century.

In medical writings which appeared even before 400 B.C. there are descriptions of deficits identical with many that are currently associated with behavioural disorders.

This is not remarkable, with the recognition that at the time of conception and during gestation women have always been exposed to a variety of potential mutagens, e.g. cosmic rays, infection, jaundice, toxaemia, haemorrhage and trauma. These mutagenic agents operated very slowly over a period of centuries and, at times, millions of years to produce individuals with altered genotypic and phenotypic patterns—the biological profile. This is the process of selection and adaptation.

But in recent years the alterations in the biological profile have been accelerated by thousands of mutagenic agents provided by the increased concentration of pollutants in the atmosphere, water, soil and food. Many of the pollutants represent newly synthesized compounds unknown to man prior to the last two centuries.

In addition to the mutagenic agents which have always operated during gestation and conception we must now include the widespread use of medications, many of them capable of altering the genetic profile, as well as the use of alcohol—not merely alcoholism but even so-called social drinking. The widespread use of tobacco, particularly cigarette smoking, is also a matter of concern in altering the biological profile.

Contemporary obstetrical practices, which include anaesthetics, medications and the position of the mother during delivery, must be considered. Today the mother during labour is placed upon her back with thighs flexed at the abdomen and the legs flexed on the thighs—a position that predisposes to compression of the blood vessels, which may lead to transitory periods of anoxia and hypoxia in the newborn child. Primitive women squatted during labour and delivered their babies without the assistance of a midwife or accoucheur, or any of the contemporary implements.

Asphyxia of the infant at birth, induced by a variety of causes, e.g. infection, haemorrhage, jaundice and compression of the cord, may play a role in producing neurological insults of various degrees, while prematurity and immaturity may also receive consideration.

These agents, acting either in unison or in concert, may provide mutagens which alter the genetic profile, providing each individual with a unique biological pattern capable of interacting with a variety of environmental agents. This interaction between the

biological or genetic profile and agents in the environment influences a variety of physiological systems whose altered function may be expressed very early as subtle behavioural disorders. In behavioural toxicology these very subtle behavioural disturbances are considered as the earliest manifestations of toxicological insults.

The various potential mutagenic agents which have been mentioned are not infrequently labelled as primary aetiological factors for the various behavioural and learning disorders observed clinically. On the contrary, they are not primary causes but rather predisposing agents which prepare the biological pattern, permitting it to interact with one or more of the activating chemicals in the environment. No reaction occurs unless the genetic pattern encounters the appropriate environmental agent. Accordingly, even the activating environmental agent is not a primary cause, since both complementary situations are essential—the appropriate genetic profile and the chemical or agent that reacts specifically with the genetic pattern, i.e. the multifactorial expression of genetics.

The multifactorial effect in its simplest form is illustrated by Zurich haemoglobinopathy, a condition characterized by maethemoglobinemia.

The haemoglobin molecule is constituted by two alpha chains, with 141 amino acids each, and two beta chains, each with 146 amino acids. In Zurich haemoglobinopathy, at the 63-position of the beta chain the amino acid arginine is substituted for histidine, the usual constituent at this position. Under normal conditions these individuals show no unusual symptoms aside from a slightly shortened life span of the reticulocytes (red cells). When an individual with the arginine substitution encounters an oxidant agent like sulfanilamide, the haemoglobin denatures and precipitates to produce fulminant haemolytic anaemia.

The maternal blood supply feeds the foetal circulation with a variety of mutagens which occur in showers distributed at random throughout the developing foetus. This situation is comparable to the random dissemination of pollens in the atmosphere.

This random distribution of mutagens leads to the involvement of various classes of genes, e.g. structural, regulatory, architectural and temporal, giving each individual a unique biological profile, so that genotypically no two individuals are identical, not even identical twins. However, most of the genetic alterations are silent unless activated by an appropriate environmental agent. Following activation the clinical manifestations expressed vary over a broad spectrum from overt clinical patterns that are readily identified even by non-professionals to very subtle behavioural disorders which are difficult to identify and therefore frequently overlooked. Even in the absence of mutagenic alterations it is conceivable that the thousands of synthetic chemicals present in the environment may either activate a silent point mutation or through its pharmacological behaviour influence physiological functions which may be manifested as disturbed behaviour. The pharmacological behaviour of a compound in turn is also governed by the genetic profile of the individual, since pharmacological behaviour is determined by enzymes, which are proteins whose structures are controlled by the genetic profile of the individual.

The failure to appreciate that many subtle behavioural disorders may be present with learning disabilities may explain to some degree the ineffectiveness of modalities for correction of learning disabilities. Until behaviour is controlled most procedures designed to improve learning deficits will not be successful.

In some individuals the insult may be of sufficient severity to produce damage to either autosomal or sex chromosomes to account for anatomical malformations, which, when less severe, are expressed as epicanthus, anomalies of the ear, high arched palate, slight incurving of the fifth finger and syndactylism. All these anatomical defects are frequently observed in the hyperactive or so-called MBD child. More severe clinical disturbances appear as Down's syndrome, Turner's syndrome, Klinefelter's syndrome, and a variety of other disturbances usually associated with mental retardation.

PREVALENCE

Except for the general agreement that behavioural disorders occur more frequently among boys than girls (reports range from a low of four or five boys to one girl, to a high of nine boys to one girl) there is no consensus regarding prevalence.

Figures for prevalence range from a low of two hyperkinetic children in a total population study of 2199 children aged 10–11 in the Isle of Wight, U.K., to a high of over 22% in grades one to six by Wender in a study in Montgomery County, Virginia, U.S.A.

This wide variation in figures for prevalence can be attributed to several factors. First, and extremely important, is the practice of cataloging the various deficits into specific compartments to serve as precise, independent clinical entities. No consideration is given to the fact that all the deficits contributing to the various independent classifications are actually an expression of an identical basic disturbance—a multifactorial reaction between the genetic profile and an environmental activator. Accordingly, the

fragmentation of various deficits into a variety of entities other than hyperkinesis, MBD or ADD is not justified. Such compartmentalization detracts from the true estimates for prevalence. All the patterns should be counted in arriving at estimates for prevalence.

At times the emotional pattern may vary widely from individual to individual and seem to divert the observer from the recognition that an identical fundamental occurrence, the multifactorial effect, operates in every situation.

A second factor to consider in arriving at estimates for prevalence is the pharmacological behaviour of the various compounds involved. Since the response is pharmacological and not allergic, which involves the immune system, it is conceivable that every individual is a potential candidate to react, depending upon the dosage of the compounds. Most of the cases that are included in estimates for prevalence are those who present a very obvious behavioural or learning disorder. However, a large segment of the population may react very subtly and not be identified as part of the behavioural syndrome. In view of this consideration the estimates for prevalence would exceed considerably the higher figure of 22%.

A third consideration in arriving at figures for prevalence is the so-called crowding syndrome of experimental psychology. When a group of animals is confined to an enclosure, and particularly if one or two of the animals are slightly more active and aggressive, all the animals become hyperactive. Sections of the brains of these animals reveal an increase in the neurotransmitter serotonin, which in some way appears to be related to behaviour.

Extrapolating this situation to the classroom with varying numbers of children, it is conceivable that with the presence of one, or at the most two, very active, disturbing and disruptive children, all the members of the class become hyperactive. It is possible that studies based upon teachers' observations may be biased because of this phenomenon, leading to the incorrect diagnosis of hyperkinesis in many children who are perfectly normal. A number of these incorrectly diagnosed individuals are managed with behaviour-modifying medications.

It is interesting to note that in some situations the teacher, too, becomes hyperactive.

Recognizing this unfavourable response of normal children to the activities of disturbed children, it becomes apparent that the commonly applied practice of mainstreaming is inadvisable.

The great lability so frequently observed in the clinical pattern may also contribute to the wide variations for estimates of prevalence. At one moment the subject may be calm, placid, cooperative, with practically no manifestation of a disturbed behaviour. The same individual without premonition or provocation may suddenly display varying degrees of

Table 1. Symptomatic terminology

Aggressive behaviour disorder	Hyperkinetic impulse disorder
Aphasoid syndrome	Hyperkinetic syndrome
Attention disorder	Hypokinetic syndrome
Character impulse disorder	Interjacent child
Clumsy child syndrome	Learning disabilities
Conceptually handicapped	Perceptual cripple
Dyslexia	Perceptually handicapped
Educationally handicapped (California State legislature AB464)	Primary reading retardation
	Psychoneurological learning disorders
Hyperexcitability syndrome	Specific reading disability
Hyperkinetic behaviour syndrome	

Table 2. Organic terminology

Association deficit pathology	Minimal cerebral palsy
Cerebral dysfunction	Minimal chronic brain syndromes
Cerebral dys-synchronization syndrome	Minor brain damage
	Neurophrenia
Choreiform syndrome	Organic behaviour disorder
Diffuse brain damage	Organic brain damage
Minimal brain damage	Organic brain disease
Minimal brain injury	Organic brain dysfunction
Minimal cerebral damage	Organic drivenness
Minimal cerebral injury	

disturbed behaviour. Because of this great lability many estimates based upon a variety of psychological tests are not valid.

The great variability in the clinical pattern can in great measure explain the confusion that exists in the literature regarding terminology, aetiology and management of the disorder. Tables 1 and 2 list some of the labels encountered in the literature. Depending upon the orientation of the observer—educator, psychologist, psychiatrist, paediatrician—and depending upon the dominant symptoms observed at the time of examination or testing, the observer is influenced in great measure.

CLINICAL PATTERNS

Recognizing the unlimited variations in the genetic profiles of individuals and the thousands of potential activating agents in the environment, it becomes apparent that a large number of a great variety of target symptoms should be identified with behavioural disturbances and learning disabilities. The study, funded by the U.S. Food and Drug Administration, conducted jointly between the University of California at Berkeley and the Kaiser-Permanente Medical Care Program, and supervised by Dr. Hicks Williams and Dr. Bernard Weiss *et al.*, has reported over 360 target symptoms.*

The deficits occur in three categories (see Table 3 for list of the commonly encountered deficits): those related to behaviour, those related to muscle coordination and those related to the higher centres, cognition and perception.

Each individual has his own pattern constituted of a mosaic of deficits drawn from one, two, or all three of the categories. Occasionally, only a single deficit may be present, which is not necessarily hyperactivity, yet the child belongs to the same syndrome, since the underlying disturbance is identical in every case.

Although hyperactivity is the most commonly encountered deficit, it is not always present and when present it may not be the most prominent deficit. For this reason the term hyperactivity is an inappropriate label. The use of the term hyperactivity has contributed to much of the confusion encountered in the literature regarding classification, aetiology and management.

The earliest expression of hyperactivity occurs *in utero*, when women may complain of inordinate activity of the baby.

In infancy, up to 2 years of age, crying, sleeplessness and fretfulness are the commonest features. Crying may be constant and not relieved by cuddling or interrupted by sleep. When held, the infant may hyperextend the body rather than relaxing, which is

*A Dietary Challenge Study of Artificial Food Colors in Children 1–7 Years Old with Behavioral Disturbances. Williams, Hicks, Weiss, Bernard *et al.* FDA Contract 223-76-2040, 15 December, 1978.

Table 3. Clinical patterns of H-LD or MBD

Group I.	Behavioural deficits
	1. Marked hyperactivity and fidgetiness
	Constant motion
	Rocks and jiggles legs
	Dances, wriggles hands
	Runs, does not walk
	Walks on toes
	In infancy, crib rocking and head knocking
	2. Excitable—impulsive
	Behaviour is unpredictable
	Panics easily—tolerance for failure and frustration is low
	Demands must be met immediately
	Diminished ability to experience pleasure
	3. Poor sleep habits
	Difficult to get to bed
	Hard to fall asleep
	Easily awakened
	Sleep interrupted by nightmares and crying out—occasionally will fear sleep

Table 3. *(continued)*

4. Short attention span—unable to concentrate

   Flits from one object to another
   Unable to sit through a school period
   Unable to sit through a meal
   Unable to sit through a TV programme

5. Compulsive aggression

   Disruptive at home and at school
   Compulsively touches everything and everyone
   Disturbs other children
   Molests pets
   Destructive (toys, household articles, structural components)

6. Perseveration

   Cannot be diverted from an action, even when life-threatening
   Punishment of no avail but leads to frustration and tantrums
   Immediately following punishment may repeat identical act

7. Self-mutilation

   Nail biting
   Scratching
   Picking and tearing skin
   Life-threatening behaviour
   Suicidal tendencies

8. Anti-social traits

   Lying and stealing
   Abusive—foul language
   Fighting—conflict with peers
   Unprovoked assault
   Defiant—disobedient
   Setting fires
   Withdrawn
   Delinquent

The entire behavioural pattern is beyond the individual's control.

Group II. Muscle incoordination

1. Gross muscles
   Exceptionally clumsy—trips when walking, collides with objects
   Cannot function in sports
   Cannot bicycle or swim

2. Fine muscles

   Eyes and hands do not seem to operate together
   Difficulty with buttoning or tying, writing and drawing
   Speech—stuttering, enunciation difficulties
   Reading—dyslexia

Group III. Cognitive and perceptual disturbances

Auditory memory deficits
Visual memory deficits
Deficits in comprehension
Disturbance in spatial orientation (up–down, right–left)
Difficulties in reasoning, e.g. simple mathematical problem, meaning of words

the usual response. Sleeplessness is an impressive feature. An infant of several months of age may sleep only a few hours a night, even in the absence of a daytime nap. In addition, headrocking and headknocking are commonly observed. The entire pattern is frequently erroneously diagnosed as colic, leading to frequent changes of formula. When these characteristics occur, walking frequently has an early onset. Quite often the disturbed behaviour is first apparent as a toddler in nursery school when confronted with a more structured environment. The child is unable to follow instructions or interact with the other children. Not infrequently aggressiveness is manifested by bossiness, hitting and biting the other children. The child does not even play alone, but is in constant motion, flitting from one object or task to another. Constant motion is a characteristic pattern.

The pattern becomes even more aggravated in kindergarten and through the grades, when the cardinal features are excitability, impulsiveness, short attention span, compulsive aggression and perseveration. Excitability coupled with impulsivity lead to unpredictable behaviour, while unfulfilled demands precipitate tantrums.

The short attention span and the inability to concentrate interfere with learning so that in spite of a high IQ these children fail at school. This behavioural pattern is perhaps the most important factor contributing to scholastic failure and in great measure is the cause of the persistent drop in scholastic performance reported over the past decade and more.

As has been indicated, the behavioural disturbances may be very subtle and not readily identified in the learning disabled child. Very commonly this leads to failure to respond to procedures employed for remediation of learning. These children are unable to learn until behaviour is controlled. This failure to identify a behavioural component occurs more frequently in the child who manifests only a single deficit, with no other overt target symptoms to alert the observer.

Compulsive aggression and perseveration typify the nature of the inherent disturbance underlying all the deficits of the syndrome, whose dominant feature is spontaneity, in the complete absence of premeditation, deliberation or provocation. Each act is beyond the child's control, as though some innate mechanism that governs all impulses is disturbed, resulting in the release of involuntary impulses which control all aspects of behaviour and at times even cognition and perception.

It becomes understandable why a child may flit from object to object or task to task and is unable to sit through a session at school or even a TV programme. The child's behaviour alienates his peers and at times even the teacher, which leads to social rejection that complicates the clinical pattern with emotional disturbances.

Repeated lying, stealing and arson are common occurrences among these children, which at times prompts severe punishment which is of no avail. Punishment and even promise of rewards seem to aggravate the situation, since these children fail to understand why they are punished. They do not understand why they misbehave. Ask such a child, "Why did you do that?". The reaction is one of consternation and frustration followed by the response, "I do not know", which is correct.

Self-mutilation, e.g. nail biting, scratching, picking and tearing of the skin, and pulling out the hair, are commonly observed in the disturbed behavioural pattern. They respond to no procedure unless behaviour is controlled.

Suicidal tendencies are not rare among these children. The impulse can only be corrected through control of behaviour.

Most important is the recognition that all the expressions of a disturbed behaviour are beyond the child's control. Most of the children do not want to be bad. They do not want to be on drugs. They do not want to be in learning disabilities classes. They are not subintelligent.

Parents are frequently blamed and made to feel a sense of guilt for the child's disturbed behaviour. Teachers are also incriminated for the child's failure to learn. However, it must be recognized that neither the parents nor the teachers are at fault. These children are not educable unless disturbed behaviour is corrected.

MUSCLE INCOORDINATION

Muscle incoordination like all the other deficits may manifest great variations, both in the type and degree of involvement. In most cases the behavioural component dominates the clinical pattern, but occasionally an individual is encountered with pronounced muscular involvement and very slight and almost imperceptible behavioural deficits.

In most cases muscular involvement concerns only one group—either the gross muscles or the fine muscles. In some individuals both muscle groups may be involved, but this is not the common observation.

Gross muscle involvement leads to awkwardness. The child is gauche, trips easily and collides with objects. These children cannot participate in sports which involve throwing or catching a ball. They are unable to ride a bicycle or swim. These deficiencies lead to social ostracism which causes emotional disturbances.

With involvement of the fine muscles, eye-hand coordination is impaired, leading to difficulty in

writing, tying the shoes or buttoning the clothes. Involvement of the fine muscles of the hands also leads to writing difficulties, while involvement of the speech muscles interferes with clarity in speech, enunciation and, in some children, stuttering.

COGNITIVE AND PERCEPTUAL DISTURBANCES

Involvement of the higher centres, as listed in Table 3, is only rarely observed in the pre-school child; they appear to have their onset in grade one but more frequently at second or third grade levels or above.

Any one or any combination of deficits involving the higher centres may be observed as an isolated disorder, but more frequently, as with all deficits, they overlap with behavioural disorders and often also muscle incoordination. Here again, the behavioural disorder may be so subtle that it is not recognized as being present with the perceptual and cognitive deficits; however, in most children the behavioural component dominates the clinical pattern to a degree that the cognitive and perceptual deficits are masked and not recognised. In some children learning disability may be attributed to cognitive and perceptual deficits, when actually the learning disability is due to disturbed behaviour. With correction of behaviour in such individuals there are no cognitive and perceptual residuals.

On the other hand, there are children with disturbed behaviour and true involvement of perception and cognition. For such children a precise evaluation of the higher centres' involvement cannot be made until the behavioural component is controlled.

With auditory perceptual deficits the child can hear but is unable to remember what is heard. For such individuals instruction by the phonetics system is not successful.

Occasionally, an auditory deficit may be observed as the only symptom. For example an 11 year old girl was failing at school. Her only deficit was auditory perception. Following dietary management the auditory deficit cleared, and the child proved to be one of the leaders in her class.

With deficits in visual perception the individual sees but does not remember what is seen. For such children instruction by the flash system is not productive.

Disturbances in orientation, e.g. up–down, right–left, are not difficult to detect.

Disturbances of the higher centres interfere with understanding the meaning of abstract words or the reasoning of simple mathematical problems, e.g. addition, division or multiplication.

THE FEINGOLD DIET

Since fleas are indigenous in the San Francisco Bay Area, our laboratories became involved in studying the allergic reaction to the flea bite. Very early it was demonstrated that the flea bite reaction is induced by a low molecular weight chemical present in flea saliva. In immunology such low molecular weight compounds are called haptens. Following this our laboratories studied the haptenic mechanism in the immune response.

On the clinical side of our programme we undertook the study of reactions to low molecular weight compounds. Since any compound in existence, natural or synthetic, can induce an adverse reaction in any individual with the appropriate biological or genetic profile, nothing is exempt. Accordingly, it became necessary, on the basis of benefit compared with risk, to evaluate each compound or class of compounds. Applying this measure, it was learned that medications and food additives, because of their widespread distribution and usage, are the commonest causes of adverse reactions, affecting practically every system of the body. We focused upon these two categories of compounds in our clinical observations.

Since aspirin is the most widely used medication, we concentrated upon the clinical reactions attributed to this medication. Very early in our clinical observations it was noted that not infrequently individuals who react clinically to aspirin may fail to improve following the elimination of all aspirin and aspirin-containing compounds.

We learned from the German literature that a number of foods contain a natural salicylate radical—compounds which are not identical to aspirin but are structurally related. On the basis of this information a diet was structured, eliminating all foods purported to have a salicylate radical, as well as a few flavours with a salicylate radical. The favourable responses to dietary management increased in known aspirin-sensitive individuals, but some individuals still failed to respond.

The next important report in the literature concerned the observation that clinically individuals who are aspirin-sensitive will not infrequently react to food colours, particularly tartrazine (FD&C yellow No. 5), although the two classes of compounds are not structurally related.

An additional observation out of Vane's laboratory in London reported that both indomethacin and the salicylates, including aspirin, although structurally unrelated inhibit the synthesis of prostaglandins (PGE_2).

This led us to the inclusion of food additives and, more specifically, the synthetic colours and flavours in the elimination diet.

Food additives are constituted of 13 categories of chemicals (Table 4), but of these we consider the most important to be the synthetic food colours and the synthetic flavours, which together with medications are the commonest causes of adverse

Table 4. Classification of intentional additives

1.	Preservatives	33
2.	Antioxidants	28
3.	Sequestrants	45
4.	Surface active agents	111
5.	Stabilizers, thickeners	39
6.	Bleaching and maturing agents	24
7.	Buffers, acids, alkalies	60
8.	Food colours	34
9.	Non-nutritive and special dietary sweeteners	4
10.	Nutritive supplements	117
11.	Flavourings—synthetic	1610
12.	Flavourings—natural	502
13.	Miscellaneous: yeast foods, texturizers, firming agents, binders, anticaking agents, enzymes	157
	Total number of additives	2764

reactions affecting practically every system of the body. The colours and flavours are the most pervasive of all the food additives, involving about 80% of our food supply. In addition, the wide distribution of these chemicals predisposes to the ingestion of reasonably high dosages of the chemicals.

In view of these observations on the behaviour of structurally unrelated classes of compounds and the clinical experiences, we hypothesized that other synthetic food colours and synthetic food flavours, although structurally unrelated, may have the potential for inducing adverse reactions in genetically predisposed individuals. In the absense of structural similarity and also in the absence of reliable tests for identification, we proceeded empirically to develop the Feingold Diet.

Part I of the diet eliminates all foods with synthetic colours, synthetic flavours and the antioxidant preservatives butylated hydroxytoluene (BHT) and butylated hydroxyanisole (BHA). The addition of the antioxidant preservatives was prompted by the clinical experience demonstrating that this class of compounds may induce behavioural disorders in some children. Part II includes the list of foods originally reported to have a natural salicylate radical (Table 5).

Table 5. Feingold Diet

Eliminate:

Part I. Artifical colours and flavours contained in food
Butylated hydroxytoluene (BHT)
Butylated hydroxyanisole (BHA)

Part II. Selected foods with natural salicylates

Almonds	Peaches
Apples (cider and cider vinegar)	Plums or prunes
Apricots	Tangerines
All berries	Cucumbers and pickles
Cherries	Green peppers
Currants	Tomatoes
Grapes or raisins (wine and wine vinegar)	Cloves
Nectarines	Coffee
Oranges	All teas
	Oil of wintergreen

The information regarding salicylates in foods is very limited. We are lacking data for the precise distribution in various foods. What other foods than those listed in the diet contain salicylates? We are also lacking information regarding the dosage of salicylates and their characterization in different foods. Are there variations in the salicylate content of different species of the same fruit or vegetable? Does the salicylate content vary with products grown in different areas? All this information has importance in the management of salicylate-sensitive individuals.

From clinical experience we have learned that many salicylate-sensitive individuals exhibit an accumulative effect for these compounds. In other words, some individuals may not experience an immediate reaction following ingestion, but after the consumption of the chemical over a period of time, varying from days in some individuals to even a month or more in others, a reaction will occur. Dosage apparently is also a determining factor. On the other hand, many individuals manifest an immediate reaction following the ingestion of salicylates even after abstinence for many months.

Since the diet has been publicized, salicylates have been reported as present in white potatoes, bananas and coffee. Except in individuals with exquisite salicylate sensitivity it is not necessary to eliminate these foods routinely.

Our initial observation of a behavioural disorder attributed to food additives was in a woman in her early forties who reported to the Allergy Department because of intense itching and swelling involving her face. The diagnosis was angioneurotic edema, commonly referred to as giant hives. After

tudying the case the reaction was attributed to the ngestion of food factors, most likely food additives. Since the adverse reaction to food additives is not an llergic response but rather a pharmacological nechanism, there are neither clinical nor laboratory ests for identification of the offending factors. Diagnosis must be made by trial and error.

Since food additives were suspected, the patient was managed with the diet eliminating all synthetic food colours, synthetic flavours, and the list of foods purported to have a salicylate radical. Her response was complete and immediate. About 10 days following her favourable response, a phone call was received from the Chief of Psychiatry of the medical centre, inquiring about our treatment. The woman had been under psychotherapy for 2 years because of a behavioural disorder—conflict with her husband and her peers, her inability to adapt to her environment. Under psychotherapy no response in the behavioural pattern was observed, but almost immediately following dietary therapy not only the angioedema improved but also her behaviour, and further, it was observed that any infraction of the diet induced an early return of both her angioedema as well as the behavioural disorder.

The staff was alerted, and we found other patients, not only adults but also children whose behaviour improved following dietary management for the somatic complaint.

Our initial interest was focused upon the children whose parents would report that not only the somatic disturbance which brought them to the medical centre was improved following dietary intervention, but also the child's behaviour. Children who were uncontrollable, destructive, abusive, in conflict with their siblings and their peers at school coupled with scholastic failure showed a rapid and often dramatic improvement in behaviour while on dietary management followed very frequently by scholastic improvement.

In order to confirm these observations we chose to gather a sample of children whose primary complaint was a behavioural disorder. Since children with such primary complaints do not report to an allergy department, it became necessary to solicit such individuals from the professionals in the community. We succeeded in gathering a sample of 25 children who had been under the care of the local university medical centre as well as competent professionals in the community. Most of these subjects for periods varying from several months to several years had been subjected to conventional modalities for therapy, e.g. behaviour-modifying medication, psychotherapy, psychological management and family counselling, etc., with no success. Following a few weeks of dietary management the disturbed behaviour of the children was controlled, followed in most cases by a rapid improvement in scholastic performance. This group of 25 children was reported initially at the Annual Meeting of the American Medical Association in New York in June 1973. Following this presentation the observations were reported in both the lay and professional press throughout the world.

APPLICATION OF THE DIET

Since there are no tests, either clinical or laboratory, to identify the offending agents, strict compliance with the diet is absolutely essential for a successful response. Implementation of the diet is not difficult, nor is it more costly than the routine fare.

Initially, in view of the lack of full disclosure, some difficulty may be encountered in identifying permissible foods. But once the items are located, the problem is simple and becomes routine.

Full disclosure of ingredients in food is deficient in practically every country, which makes necessary the research of the local food supply. Research should be conducted locally, since there are variations from one locale to another, even for the same foods, and for products of the same manufacturer.

To meet this problem, in many countries parent associations constituted of families with a successful experience with dietary management have been organized to investigate the local food supplies and compile lists of permissible items. With the availability of this necessary information, shopping is expedited, and implementation of the diet is facilitated.

Success with the diet makes it imperative that all family members comply. One cannot expect a child to adhere strictly to a programme with the availability of prohibited items in the home. Requiring the compliance of all family members is not an unreasonable request, since the chemicals eliminated are harmful to all individuals, producing disturbances affecting various systems of the body. In addition, many of these compounds are suspected to be carcinogenic. Why must one wait 10–20 years to make this determination? It must be appreciated that the safety of these compounds has never been proved.

THE RESPONSE TO DIETARY MANAGEMENT

Strict application of the diet will be followed by a favourable response in the behavioural pattern in 40–50% of the children. In the younger age group, from infancy through 4 or 5 years of age, the observed

improvement in behaviour may occur in 60% of subjects. In the school-aged child, control of behaviour leads to improvement in scholastic performance. At any age the response is impaired in the presence of true neurological damage; however, even in the presence of true brain insults a considerable improvement in the behavioural pattern may be observed with the persistence of deficits involving the muscles and higher centres.

Failure to respond to strict implementation of the diet is an indication to look beyond the diet. Depending upon the biological profile, anything in existence, either natural or synthetic, may be a provoking agent. Unfortunately, this may present a formidable problem, since there are no reliable clinical or laboratory tests for identification of the offending agent. There are reports in the literature advocating a variety of test procedures, e.g. the sublingual test, skin tests with varying materials, and laboratory testing, including radioimmunoassay and cytotoxic blood tests, but upon closer scrutiny it will be learned that none is reliable. In the absence of specific reliable techniques to fill the void in identification, many frustrated individuals become ready prey for exploitation.

The only available procedure for individuals who fail to respond to the prescribed diet is a carefully documented medical history, which may serve as a guide for the recognition of a provocative agent, which, when suspected, can be identified only by trial and error.

Toxicants contaminating the atmosphere, water and soil may be activating agents, but fortunately their concentrations are usually of such low order that they are not the common causes of behavioural disorders. Gross contamination of the environment by chemicals is generally identified because of the high incidence of involvement in a community, characterized by overt disturbances which are not limited to behaviour. Cancer is a common sequel.

From clinical observations with dietary management several generalizations have been formulated:

(1) The younger the child the more rapid the response.
(2) The younger the child the more complete the response.
(3) If behaviour improves, the prognosis for all deficits clearing is excellent.

Intrauterine hyperactivity can in most cases be attributed to offending agents ingested by the mother and transmitted to the foetus. Control of the mother's diet is usually followed by a rapid cessation of hyperactivity.

The dominant characteristics of infant responses from birth to 24 months has been described.

It has been observed that most of these troubled infants are receiving paediatric vitamin drops, which contain synthetic vitamins as well as synthetic colours and flavours. When the vitamin drops are discontinued all the symptoms clear within 24–36 hr, while restoration of the vitamins is followed with a recurrence of symptoms within 24–36 hr.

Children from 2 to 5 years of age show a favourable response to the diet within 3–5 days, with a complete recurrence of the pattern within 24–36 hr following a challenge. The recurrence of symptoms may persist for several hours or in some children, for several days. This pattern will vary with the degree of sensitivity of the child to the offending agents and to the dosage of the chemicals ingested.

The school-aged child, i.e. from kindergarten through the grades or about 5 years of age through 13 years, presents several patterns of response.

The first pattern includes those children who, following dietary intervention, experience an improved behavioural pattern both at home and at school, followed very quickly with improved scholastic performance. There are no residual deficits. The child is happy, calm, obedient, cooperates, with no conflicts with either siblings or peers. The home environment which had been disruptive and in constant turmoil is now serene and happy.

The second pattern includes the children whose behaviour at home has improved but is still not controlled at school. Upon studying the situation, it is learned that the child has established a reputation among his peers and with the teacher for being a bully—annoying, disruptive, destructive, unpleasant and failing to comply with routine regulations. As a result, the child is isolated from the group and enjoys no social acceptance.

The rapid transformation in the behavioural pattern, within a matter of weeks, and at times even within days, is not recognized or appreciated by either the teacher or the peers, which leads to persistent social rejection and at times even unjustified disciplinary action. This leads to frustration and rebellion which is expressed in a continued disturbed behavioural pattern and failure to learn. For such children a change in classroom or even better a complete change in schools will provide a new set of peers, a new teacher and the opportunity to socialize without the prejudice of his previous reputation. Following such an adjustment, improved behaviour at school will soon be followed by improved scholastic performance.

In the third pattern of response the child's behaviour improves at home but is not accompanied by improved behaviour at school. Following the controlled behaviour at home, an evaluation of the pattern at school may reveal the persistence of

disturbances in cognition, perception and the higher centres. When behaviour is controlled, identification can be made of the specific deficits; however, until behaviour is controlled the various tests for evaluation of the deficits are not valid.

Following identification of the specifics of higher centre involvements, the management should provide for tutoring on a one-to-one basis, with a focus upon the deficits. These children are very susceptible to the slightest stress factor, which may be the presence of one other individual while being instructed. A sudden change in light or sound intensity can be a distracting factor, preventing the child from concentrating upon the learning problem. In every instance the focus of teaching must be upon the deficits identified.

Since the response to such management may be very slow, varying from a few months to a year or more, success is contingent upon extreme patience coupled with moral support and encouragement of the child.

The absence of persistent neurological deficits in the younger child, following control of behaviour, and the greater incidence of persistent neurological deficits as the child grows older suggests that the offending chemicals operate slowly over a period of years to induce neurological damage. The degree of damage will vary from child to child, depending upon the genetic profile of the individual, the inherent toxicity of the compound, and the dosage.

Levitan demonstrated that following persistent perfusion of the nerve cell by micropipetting techniques, irreversible changes in the nerve may be induced. In this respect the slowly developing irreversible nerve alterations associated with food activities differ from the more rapidly developing brain damage expressed as retardation in phenylketonuria (PKU). However, it appears that in some few individuals food additives or their metabolites may induce rapidly developing brain damage expressed as retardation similar to that occurring in PKU.

The importance of emphasis upon the deficits is supported by studies indicating that stimulation of an injured nerve structure may lead to regeneration of dentrites. However, in the presence of incomplete damage to a cell or group of nerve cells the participation of collateral nerve structures may be encouraged, leading to a restoration of function. In any event, if behaviour is controlled, the prognosis for ultimate complete response is very encouraging.

As the child passes through puberty there may be a spontaneous alteration in the behavioural pattern, with lessening of hyperactivity and aggressiveness as well as many of the outward signs of a disturbed behaviour. This alteration in the behavioural pattern is frequently misinterpreted as 'growing out' of the condition—an attitude not infrequently expressed by both parents and some professionals. However, these children do not 'grow out' of their disturbed patterns, since the basic predisposing condition—the biological profile—persists throughout the lifespan of the individual.

The persistence of deficits interferes with the individual's ability to cope with the environment and compete with peers. In many adolescents learning disabilities persist but not in all cases. For some adolescents the ability to learn presents no problem; however, the ability to adjust and adapt to the environment is the chief disturbance. As a result, these individuals become frustrated. They develop a low self-esteem, become withdrawn and anti-social, with a tendency toward lying, stealing and various unlawful acts. These individuals are ready prey for exploitation by various youth and adult criminal gangs.

The initial presentation of the concept linking diet to behaviour at the Annual Meeting of the American Medical Association (AMA) in June 1973 was preceded by a press conference scheduled by the AMA. The press conference immediately generated international interest, which was soon followed by literally thousands of reports of favourable responses to the Feingold Diet for the management of behavioural disorders and learning disabilities.

Along with the reported successes great scepticism and concern regarding the validity of the concept were expressed by many members of the professional community as well as the food industry, represented by the Nutrition Foundation of New York.

The critics contended that controls were lacking in the initial study to rule out non-specific factors, e.g. placebo and Hawthorne effects as primary influences leading to desired and anticipated responses in the treated subjects.

No consideration was given to the structure of the initial sample reported in June 1973. Each of the 25 children in the original sample was diagnosed as hyperactive by leading professionals in the community. Each of the children for periods varying from 1 to 3 years had been subjected to every known therapeutic modality, including behaviour-modifying medications, psychotherapy, psychological management, family counselling and behaviour modification, but to no avail. Following a few weeks of management with the Feingold Diet, a rapid and often dramatic reversal of the behavioural disorder was observed, which in most cases was followed by improved scholastic performance. The question was never considered: "Why should a few weeks of dietary management have a greater non-specific influence than years of involvement and concern on the part of parents and professionals?"

It has already been indicated that very young infants develop disturbed behaviour from paediatric vitamin drops which contain synthetic food colours and flavours, while elimination of the vitamin drops is followed by control of the disturbed behaviour. It is not likely that this clinical observation in infants can be attributed to non-specific or placebo effects.

A critical review of each clinical study reported to date supports the contention that food additives do cause hyperactivity. Without exception each investigator reports that a small subclass of children, particularly at the preschool level, do react to food additives, while management with the Feingold Diet controls the behavioural disorder. The observation of disturbed behaviour in the very young, as has been previously indicated, is consistent with the basic tenet in behavioural toxicology that the younger the individual the more susceptible they are to toxic insults. This response is attributed to the inability of the immature individual to metabolize the toxic agents as efficiently as older individuals.

The current controversy revolves around numbers. The critics of the hypothesis contend that only a small number, perhaps 5–10% of children, react adversely to food additives and salicylates rather than the 50% favourable responses reported by me.

In view of the thousands of compounds involved and in view of the complexity of most compounds, particularly the flavours, studies were designed focusing upon the food dyes, and particularly erythrosin, FD&C Red No. 3. Without exception the studies with dyes confirm that these chemicals do disrupt the nervous system.

Levitan and Augustine employed erythrosin, FD&C Red No. 3, in a micropipetting technique with the buccal ganglion cell of the mollusc *Navanax inermis*. These investigators observed an altered conductivity of the cell membrane with a disturbance of electrolyte balance. These investigators also studied the effect of the dye on the neuromuscular junction of the frog's leg. A disruption of function was again observed.

Silbergeld reported on *in vitro* studies with homogenates of brain and demonstrated an interference by the dyes with dopamine uptake.

Swanson and Kinsbourne, recognizing the pharmacological behaviour of the dyes, which indicates that dosage is an important factor, conducted a clinical study on hyperactive children who responded to the Feingold Diet, challenging them with 150 mg of dye, which contrasts strikingly with the various studies employing the Nutrition Foundation cookies, which contain only 13 mg of a mixture of dyes. Without exception the children in Swanson and Kinsbourne's study showed a disturbance in learning ability.

An increasing number of studies are being reported using dyes at the cellular level. In each case a disturbance of the nervous system was observed.

In animal studies with the dyes, conducted by Shaywitz *et al.*, a disturbed behaviour was observed following administration of the dyes.

Assurance of a favourable response to dietary management entails more than distributing a list of chemicals to be avoided. Identification of permissible foods in the local food supply is greatly hampered by inadequate disclosure of food chemicals in practically all Western countries. In the U.S.A. the disclosure of food additives is not required for dairy products or for alcoholic beverages. Not infrequently, a product may be labeled 'No Preservatives Added'; however, such a statement is no assurance that additives are not present. For example: A food processor may incorporate a food additive, e.g. BHT, into a ingredient used in food production, but this may not be disclosed, since it was not introduced by the manufacturer of the finished product.

To cope with this situation a number of parent associations interested in dietary management have sprung up in the U.S.A., Canada, Australia, New Zealand, and to a lesser degree in Britain, whose objective is to research the local food supply for the presence or absence of forbidden chemicals, which may vary from region to region. The compilation of lists of permissible foods expedites shopping and also assures success with the diet.

For dietary management to be successful, consideration must also be given to socio-economic factors. Perhaps the most important socio-economic condition is the marked increase in the number of women, and particularly the number of mothers, who are economically active. In the U.S.A. it is estimated that over 53% of all school children are from homes with both parents working, while more than one out of three school children are from single parent homes. In the parochial school system of Chicago, Illinois, constituted of over 400 schools, a survey revealed that 40% of the children took lunch at school but had breakfast and dinner at home alone, with no supervision. It can be readily appreciated that a void exists in our social structure, which involves a large segment of the school population, who are leading a solitary existence with practically no supervision over their nutritional and health requirements.

For any programme of dietary management to succeed in the treatment of behavioural disorders, learning disabilities and delinquency, it is essential that provisions and procedures be developed to fill the

deficiency in our social structure. It is not likely that the socio-economic factors contributing to the situation will be ameliorated within the foreseeable future. Therefore, it is imperative that programmes be developed to compensate for the existing deficiency. The most logical and reasonable answer would be for the schools to develop programmes to meet the needs of the community. The schools should recognize the void that exists in society and extend their interests and responsibilities of the school beyond the school grounds, which has been traditionally the policy of most institutions of learning. The environment has changed and new situations have developed, making it imperative for the schools to participate in programmes that will avert and ultimately resolve the critical deficiences in our educational system. The primary responsibility of the school is to educate, which cannot be achieved unless the disturbances of the children are corrected, permitting them to be educated.

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. The prospect for controlling and eliminating the major contaminants of the environment is not too promising for the immediate future; however, an informed public, which should lead to greater commitment and involvement, would be followed by the containment and then reversal and resolution of this critical and important present-day situation involving the health and behaviour of both our contemporary population and also future generations. Public recognition and participation in the problem are mandatory to correct the insidious downgrading of the human race, which is already evident.

NOTE ADDED IN PROOF

Dr. Ben F. Feingold finalised this paper in the few days before his death (see Obituary, p. 199). Although the text was complete, Dr. Feingold had not had time to list his references, and as it was not a task that could easily be taken over by another person, we decided to go ahead and publish this, his last paper, without the references.

The Editors

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OBITUARY

BEN F. FEINGOLD MD

Ben Feingold true to his nature spent his last energies in passing on his knowledge and ideas to others. We are both honoured and humbled that he considered our new journal worthy of his last efforts, for not only did he prepare this work for publication in *Ecology of Disease*, he did it knowing it was to be his last. He had previously joined our Editorial Board and gave freely, as he always did, of his time and experience.

Ben was a good physician, a fine allergist, and perhaps above all, a pioneer of great courage. In 1973 he told a startled audience at the American Medical Association about his theory that hyperactivity in children may be linked to certain colour additives in food and drink—many were the sceptics and the food and drug industry rounded on him sharply. Not deterred, he continued with his studies and showed both by research and remedial treatment that his theory warranted serious thought if not outright acceptance. The results of his treatment were too stunning to be ignored for long—hitherto hyperactive children became calm and attentive when put on the Feingold Diet. Ben was not able to win over all the sceptics but parents thought better and formed the *Feingold Association of the United States* and recommended the diet throughout the country at meetings of affiliated groups.

In the same year that he delivered his paper to the American audience he spoke of his ideas in London at the *Man/Food Equation Symposium*. It was at that meeting that I first met Ben and his wife Helene. From the start of our friendship he showed his generosity by his gentle but firm encouragement to me during the period when this journal was just a germ of idea. Although not well, Ben was always willing to help out. He will be missed, but there are a lot of 'Feingold' parents and their children who will remember him with us.

ARTHUR BOURNE