The Effects of Diet on Behavior: Implications for Criminology and Corrections

by Diana Fishbein, Ph.D., and Susan Pease, Ph.D.

with a review by Orville B. Pung

A series reporting the results of applied research in corrections for administrators and practitioners
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THE EFFECTS OF DIET ON BEHAVIOR: IMPLICATIONS FOR CRIMINOLOGY AND CORRECTIONS
Diana Fishbein and Susan Pease Page 1

APPLYING DIETARY THEORIES TO INMATE POPULATIONS
Orville B. Pung Page 45
The Robert J. Kutak Foundation takes pride in its association with the National Institute of Corrections to provide funding for the publication of this series of monographs designed to narrow the gap between researchers and corrections practitioners and policymakers.

The Foundation was created in 1983 to honor the memory of the late Robert J. Kutak, who was struck down at the age of 50 with a fatal heart attack in January of that year. The Foundation was established by his colleagues in the law firm of Kutak Rock & Campbell, along with other friends and associates, to carry on some of the work to which he devoted so much of his time, energy, and talent.

Prominent among those activities was his abiding interest in corrections. He helped draft the legislation creating the National Institute of Corrections and served as the first chairman of the Institute’s Advisory Board. He was a member of the President’s Task Force on Prisoner Rehabilitation and the American delegation to the Fourth and Fifth United Nations Congresses on the Prevention of Crime and Treatment of Offenders. He was a member of the National Advisory Commission on Criminal Justice Standards and Goals and vice-chairman of the American Bar Association Commission on Correctional Facilities and Services.

Bob Kutak had personally visited dozens of correctional facilities, ranging from the largest federal prisons to local and community jails. He had a keen interest in the improvement of corrections in America at all levels and held a strong belief that applied research has a critical role to play in attaining that goal.

The Board of Directors of the Kutak Foundation is convinced that none of its many activities would be more appreciated by Bob Kutak than the joining of the Institute and the Foundation in an effort that holds such great potential.

The Foundation is especially grateful to Dr. Steven W. Horn and former U.S. Senator Roman L. Hruska, both members of its Board of Directors, who have taken the lead in bringing this important project to fruition.

Harold L. Rock
President, Kutak Foundation
EDITOR'S NOTE

Does food directly affect people's behavior, particularly deviant or illegal behavior? The popular press has suggested that people act differently if they consume certain types of foods (e.g., sugar, food dyes, and other additives). But what do the facts suggest? And if there is a relationship between food and antisocial behavior, what are the implications for corrections administrators?

The topic of this monograph, the effects of diet on behavior, was a particularly difficult one, in that the authors had to summarize a body of research that is highly technical and requires considerable sophistication in the biological sciences. They have done an admirable job, and this review provides a wealth of information for corrections administrators and practitioners.

Dr. Diane Morris, an Assistant Professor at the University of Massachusetts Medical Center and a Research Associate at the Harvard School of Public Health, conducted a technical review of an earlier draft. Her comments significantly improved the final monograph.

The next issue of Research in Corrections will present a review of pretrial release decisionmaking written by Stevens H. Clarke, of The Institute of Government, University of North Carolina. In the final issue for 1988, Dr. Douglas McDonald, of Abt Associates, Inc., will discuss correctional costs.

Articles are now being commissioned for 1989; we would be pleased to hear from individuals who would like to contribute research papers or serve as practitioner respondents. All correspondence should be addressed to Joan Petersilia, The RAND Corporation, 1700 Main Street, P.O. Box 2138, Santa Monica, CA 90406-2138.

Joan Petersilia
The National Institute of Corrections is pleased to offer with the Robert J. Kutak Foundation the second monograph in the Research in Corrections series. The monograph series is designed to provide high-quality summaries of research for correctional practitioners. Each monograph seeks to convey the key research findings on a selected topic in a clear and policy-relevant fashion, along with the responses of one or more correctional practitioners to the operational issues that arise in applying those findings in real-life settings.

In this monograph, the authors, Drs. Diana Fishbein and Susan Pease, provide an overview of current knowledge and speculation concerning the relationship between diet and behavior, particularly as it relates to corrections practice. Orville Pung, Commissioner of the Minnesota Department of Corrections, discusses application of their findings to management of inmate populations.

This is a complex topic which draws on a fast-growing body of research in the biological and behavioral sciences. At a time when there are frequent articles in the media regarding nutrition and the impact of specific foods on the problem behaviors of juveniles and adults, it is important to gain a clear understanding of how dietary factors (e.g., refined carbohydrates, food additives, trace elements) influence behavior and to cautiously review those studies that explore the link between diet and the antisocial behavior of offenders.

Raymond C. Brown
Director, National Institute of Corrections
THE EFFECTS OF DIET ON BEHAVIOR: IMPLICATIONS FOR CRIMINOLOGY AND CORRECTIONS
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PREFACE

As criminal justice moves into the twenty-first century, the biological sciences will play an increasingly important role in the study of the causes and control of criminal behavior. Criminal justice theorists and practitioners must begin to include the biological perspective in the study of human behavior, as evidence is mounting that many behavioral disorders, such as learning disabilities, antisocial personality, hyperactivity, and many forms of mental illness, have a biological basis.

The explosive growth of information in the biological sciences has resulted in tremendously detailed and complex constructs, and it is difficult to communicate these highly technical ideas to the people who might benefit most from them, those who deal with criminal offenders directly. This monograph presents a general overview of current knowledge and speculation concerning the relationship between diet and behavior, particularly as it relates to criminal justice theory and practice.

It is important that criminal justice agencies keep abreast of scientific findings in the biological sciences that may help explain some aspects of antisocial behavior. It is hoped that this review will help to enhance the preparedness of the criminal justice system in dealing more effectively with criminal offenders.

INTRODUCTION

The possible relationship between diet and criminal or delinquent behavior has generated considerable interest among criminologists, nutritionists, criminal justice administrators, and juvenile justice administrators. Some criminologists and practitioners are embracing what appears to be a rather simple solution to the complex problem of managing criminal and juvenile offenders in institutional settings. Several researchers (e.g., D’Asaro et al., 1975; Schauss, 1980; Schoenthaler, 1985) have suggested that a change in diet will significantly reduce behavior problems among these offenders. Others (Gray, 1986; Gray and Gray, 1983; Love and Pease, 1987; Pease and Love, 1986) have criticized
that research on methodological grounds and have argued that current information about the relationship between diet and behavior indicates that a simple change in diet would not result in drastic behavioral changes for the majority of people.

In the midst of this controversy (see letters to the editor, Nutrition Today, 1985), we take the position that the relationship has been neither empirically supported nor refuted. The controversy can be resolved only when clear empirical evidence is developed on the effects of dietary changes within a reasonable theoretical framework that is supported by scientists from all relevant disciplines.

With the exception of some early attempts to identify an association between gross anatomical features and crime (Ferri, 1929; Garofalo, 1914; Lombroso, 1918), the study of biological factors by criminologists is relatively recent. This neglect of biological contributions to criminal behavior may be partly a result of the fact that the American legal system is based on the concept of “free will” and individual responsibility for one’s behavior. The idea that behavior may be the product of chemical reactions in the body seems to contradict the very basis on which our legal system rests.

It has also been difficult to incorporate findings in the biological sciences into criminal justice practices because of the speed with which highly complex scientific advances are being made. Man’s knowledge of his physical self is growing so rapidly that medical journals are out of date within a year’s time. This growing body of knowledge on the relationships between biological factors and human behavior has drawn the attention of some criminologists, who have formed the new subdiscipline of “biocriminology.”

This paper examines the relationship between diet and behavior in terms of both methodological and theoretical issues. First, we present an introduction to the field of biocriminology, i.e., the study of socio-environmental and biogenetic contributions to human behavior. Then several parameters of the relationship between diet and behavior are examined. This discussion considers neurotransmitter imbalances, food sensitivities and allergies, hypoglycemia and refined carbohydrates, reactions to food dyes and other food additives, and dietary trace elements. We next review recent studies of the relationship between diet and antisocial behavior in juvenile and adult offender populations and propose some criteria for research methods. Finally, recommendations are made for criminal justice practitioners and administrators who wish to develop policies that are responsive to what is known about diet and behavior.

**WHAT IS BIOCRIMINOLOGY?**

Modern criminology has been dominated by the academic disciplines of sociology and psychology. Criminal behavior has been explained in terms of such sociological variables as socioeconomic status, group cohesion, and unemployment, and such psychological variables as intrapsychic conflict, neurosis, psychosis, and inadequate parenting. The study of biological variables in
modem criminology was introduced by C. Ray Jeffery in his 1977 book, Crime Prevention Through Environmental Design. Dr. Jeffery argues that all behavior is learned and that learning is a biological as well as a social process.

From this early work, biocriminology has evolved into an interdisciplinary behavioral science incorporating such diverse areas as sociology, psychology, economics, anthropology, environmental design, and, most important, behavioral biology, which emphasizes the study of the brain and behavior. As shown in Figure 1, previously separate disciplines within the broad spectrum of behavioral biology and the brain sciences are merging to form new behavioral disciplines, such as psychopharmacology (the study of the effects of drugs on the mind and behavior), psychoneuroendocrinology (the study of the effects of hormones on mood and behavior), and behavioral nutrition (the study of the effects of food on mood and behavior). The study of the brain and its workings may lead to new, potentially effective strategies for criminal justice practices and techniques (Fishbein and Thatcher, 1986; Hippchen, 1978; Jeffery, 1979; Lewis, 1981).

It is important to emphasize that we are not attempting to explain behavior in terms of neurons and chemicals. We begin, rather, by drawing a rough analogy

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Fig. 1-The cumulative process model of science illustrates the interrelationships among scientific disciplines. The physical sciences deal with minute details of the behavior of energy and matter. As we approach the study of animal/human behavior, disciplines incorporate these physical principles to explain behavior as it arises from both physical and sociocultural properties. Thus the study of human behavior necessarily adopts a multidisciplinary perspective.
between the brain-behavior relationship and the output of a computer. The brain and the computer both use internal programs to process stimuli from their environment. A computer that is operationally intact can produce scrambled output when it receives faulty instruction. It can also produce scrambled output if the internal wiring is faulty, even though the instructions are accurate. The operation of the brain is far more complex than a computer, of course. A criminogenic environment may produce a criminal even though the brain is operating at optimal capacity. Conversely, some biological malfunction in the brain may result in behavior that is culturally identified as antisocial, even when the individual is in an environment that is noncriminogenic. Most antisocial behavior is a result of an interaction between deleterious conditions of both the environment and the brain. As illustrated in Figure 2, the more of these deleterious conditions the individual is exposed to, the greater the likelihood of criminality. This interaction is clearly demonstrated in the link between learning disabilities and delinquency.

Learning disabilities and intellectual deficits have been consistently associated with delinquency, criminality, and the inability to make optimal behavioral decisions. A review of the literature strongly suggests that learning disabilities, as reflected in intelligence and school performance measures, are correlated with and may predispose individuals to behavioral disruptions and delinquency (Critchley, 1968; Hippchen, 1978; Hirschi and Hindelang, 1977; Holzman, 1979;...

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**THE DEVELOPMENTAL STEPS OF MALADAPTIVE BEHAVIOR**

- **Insult or Trauma**
  - Nutrition
  - Toxins
  - Head Trauma
  - Inborn Error of Metabolism
  - Physical Abuse
  - Stimulus Depreciation
  - Substance Abuse
  - Physical Sensitivities
  - Psychological Trauma

- **Behavioral Responses**
  - Learning Disability
  - Hyperactivity
  - Impulsivity
  - Psychopathy
  - Schizophrenia
  - Aggression
  - Depression
  - Mania
  - Mental Burnout
  - Reduced Potential

- **School Problems**
  - Rejection
  - School Peers
  - Family
  - Isolation
  - Underachievement
  - Poor Self Concept
  - Frustration
  - Guilt
  - Association w/ like peers

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Fig. 2-Certain suboptimal conditions of our environment and our biological state contribute to the development of psychological or behavioral problems. Exposure to such deleterious factors places an individual at risk for delinquency or criminality by increasing the likelihood of maladaptive or antisocial behavior.
Margolin et al., 1955; Poremba, 1975; Robins, 1966; Rutter et al., 1970; Tama-
pol, 1970; Wolfgang et al., 1972; Yeudall, 1977). Children suffering from a
learning disability may experience a variety of behavioral difficulties whose out-
come depends upon both the source of the learning disability and the child’s
social environment. One suggested sequence of events is as follows: The learn-
ing disability leads to failure in school, which leads to a dropping out of school,
which precipitates involvement in delinquent behavior, possibly resulting in
involvement with the juvenile justice system. The disability may compromise
the child’s decisionmaking ability and further increase his or her risk of anti-
social behavior.

Learning disabilities have frequently been traced to prevailing brain abnormali-
ties (Holzman, 1979; Tamapol, 1970; Thatcher and Lester, 1985; Yeudall et al.,
1985). Deleterious influences or risk factors impair or modify brain functioning,
which may lead to or exacerbate a learning disability. The disability, in turn,
may affect the brain’s ability to regulate behavioral responses and may place the
individual at risk for delinquency and criminality. Support for such a view is
provided by studies (cited above) that demonstrate relationships between learn-
ing disabilities and antisocial behavior. Consequently, we would expect to find
physiological differences between antisocial individuals and “normal” individu-
als. The literature provides evidence that more abnormalities in brain function
do indeed exist among delinquent and antisocial individuals (Fishbein et al.,
1988a; Hare, 1970; Mark and Ervin, 1970; Monroe, 1978; Robbins et al., 1978;

We next present a brief overview of the structure of the brain and its operation,
explaining mechanisms that are important in producing human behavior, to clarify
how dietary factors may affect brain function and behavior.

BRAIN ANATOMY AND CHEMISTRY

The brain has three primary components: the brainstem, the limbic system, and
the cortex (see MacLean, 1976). Each of these areas specializes to a certain
extent in its function and works in concert with the others to produce behavior
(see Figure 3). The brainstem is responsible for physical survival; it controls
breathing, heart rate, blood pressure, muscle movement, and other activities that
keep a person alive without any conscious control being exerted. Disorders that
affect the brainstem may cause paralysis, muscular diseases, sudden infant
death, and other problems over which the individual has no control (Harmony,
1984).

Situated on top of the brainstem is the limbic system. This primitive area of the
brain is composed of many different structures that, in general, motivate people
to respond to their environment and increase their chances of successful sur-
vival. Specifically, moods, hunger, thirst, reproductive and sexual behaviors,
anger/aggression, memories, and many other feeling states that lead to certain
types of behaviors are regulated by this region. Disturbances that involve this
system, such as epilepsy, hormone imbalances, or low blood sugar, have been
associated with affective disorders (i.e., depression), violence, sleep problems,
memory lapses, sexual disorders, and other conditions (see Restak, 1984). This emotional center of the brain is highly vulnerable to chemical and physical traumas that can result in psychiatric or behavioral disorders. Although these disorders frequently have neurological involvement, the presence of behavioral or psychological symptoms may disguise the biological contributions and preclude accurate diagnoses.

The cortex rests above the limbic system and is highly developed in humans. This structure is responsible for higher intellectual functioning, problem solving, logic, forethought, information processing, and decisionmaking. Individuals who suffer from a cortical disturbance, depending on the region affected (see Figure 4), may experience language disorders, impulsivity, reading or comprehension difficulties, and a variety of other cognitive or intellectual impairments.
A number of studies have identified cortical deficits (particularly in the frontal lobe) in individuals with conduct disorders, antisocial behavior, hyperactivity, and other characteristics that are frequently found in individuals who engage in delinquency or criminal behavior (Hare, 1984; Luria, 1973; Nauta, 1971; Yeudall et al., 1985). Cortical deficits caused by environmental factors (i.e., head trauma) or of biological or genetic origin are associated with poor impulse control, problems anticipating consequences of one's actions, language difficulties, and irrationality.

The function of all the brain structures and regions is largely dependent upon brain chemicals. In fact, all brain activity is the result of electrochemical impulses that are evoked by both bodily processes and environmental stimuli. Brain chemicals include hormones, which are secreted by glands throughout the brain and body and travel to other locations to exert their influence. One hormone, adrenalin, produces such effects as increased heart rate and blood
pressure, feelings of anxiety, energy, stress, and aggression; insulin, another hormone, which is stimulated by carbohydrate intake, has been implicated in impulsive behavior.

All of the structural features of the brain consist of cells called neurons. Neurons communicate with each other via chemical messengers called neurotransmitters. Neurotransmitters are directly responsible for behaviors and emotions, and, like the structures mentioned above, each neurotransmitter serves a different function, although some functions do overlap.

Neurotransmitters are directly responsible for behavior, emotion, mood, and learning. They are chemical compounds that lie between nerve cells and send signals from one neuron (brain cell) to another. These systems are the basis of all thinking and body control in the brain and its neural branches. Figure 5 illustrates in more detail how neurotransmitters permit neurons to communicate.

![Neuron communication diagram](image)

Fig. 5-Neuron A communicates with neuron B via a synapse between an axon (releasing neurotransmitter toward an adjacent cell) and a dendrite (conveying information to the cell body). The expanded part of the illustration shows the synapse, which is actually a tiny gap separating the membranes of the two neurons. Transmitter chemical stored in tiny packets of the axon is released into the synapse upon the arrival of an action potential. The transmitter chemical then either excites or inhibits the activity of neuron B. (From Teyler, 1975.)
Neurotransmitter deficiencies or excesses can produce profound disruptions in physical and behavioral processes (Coppen et al., 1972; Lieberman et al., 1982; Snyder, 1980). Such changes can be observed following natural alterations or artificial manipulation of neurotransmitters or the substances responsible for their production. The production of neurotransmitters is dependent, in large part, on the availability of certain dietary constituents or “precursors,” including vitamins, minerals, carbohydrates, fats, and proteins (see Lovenberg, 1986).

Many brain chemicals are synthesized in the brain without much assistance from outside sources; the ingredients are available within the body. Others, however, require external supplies for their production. If these supplies are diminished or insufficient, disease or disruption of a mental activity may result. For example, Vitamin D comes largely from sunlight. Anemia or osteoporosis (bone thinning) may occur if sunlight is unavailable. Many brain chemicals rely on components or constituents of the foods we eat. We quite literally “eat to live.” Without these food constituents, our brains would be unable to manufacture adequate supplies of the neurotransmitters that are essential to our existence and performance.

EVIDENCE SUPPORTING THE RELATIONSHIP BETWEEN DIET AND BEHAVIOR

To evaluate studies that have attempted to demonstrate the link between diet and behavior, we must first understand the fundamental principles of research methods. Sound scientific methodology is based on the simple principle that the results of a study should be attributable only to the manipulation performed by the scientist, not to any other uncontrolled factors. For example, if a researcher finds that counselor A has more success with psychotherapy than counselor B has with behavior modification, can we conclude that psychotherapy is more effective? Absolutely not. Personalities, surroundings, circumstances, and numerous other factors come into play to determine the success or failure of a therapy. The scientist must anticipate and eliminate or control as many competing factors as possible when designing his or her study.

Simple research studies frequently contain complicated-sounding scientific jargon. Titles such as “A Double-Blind Placebo Controlled Crossover Challenge Time-Series Quasi-Experimental Test of the Effects of Diet on Behavior” suggest methodological rigor and integrity. One might conclude that an impressivesounding title indicates a high-quality research product, but this is not always the case. The terms in this illustrative title are defined briefly below.

A double-blind placebo control group design means that neither the experimenter nor the subjects (i.e., participants) know who is in the experimental group (i.e., the group receiving the treatment) and who is in the control group (i.e., the comparison group). Even an honest scientist may be so invested in supporting a particular hypothesis that he or she may unconsciously act in a manner that influences the behavior of the subjects to comply with the expected outcome of the experiment. Compounding this problem is the fact that subjects may
unconsciously try to please the experimenter by acting in the expected manner. The double-blind procedure prevents these errors from occurring by not informing participants of group assignments.

Another problem occurs when subjects are either told or discover that a program or treatment will produce a certain effect; the treatment then usually does exactly that, whether there is any active ingredient in it or not. This is called the 'placebo effect.' To avoid this result, experimenters may administer a placebo (i.e., an inert treatment or "sugar pill") to one group and the experimental treatment to another group; this enables them to determine whether any change in behavior or mood is due to the real treatment or simply to the act of receiving attention. Changes incurred over and above the placebo effect in the experimental group can be attributed to the experimental treatment. Thus, a double-blind placebo control group design means that neither the experimenter nor the subjects know who is receiving the treatment and who is receiving the placebo.

A crossover challenge design is recommended in diet studies, to control for several external influences. This design requires that one group maintain the 'experimental' diet for a specified amount of time, while the control group adheres to a conventional or other diet for the same period. Before the subjects begin the dietary program, measures are taken to evaluate existing behaviors, e.g., the incidence of disciplinary problems. After the evaluation, the behavioral assessments are repeated, and each subject "crosses over" into the opposite group. This process is repeated once more, with the subjects once again placed on their original diets, and before-and-after behavioral measures are obtained. The researcher expects (or hypothesizes) that the experimental diet will produce improvements in behavioral measures that will then deteriorate when the conventional diet is reintroduced. The same improvements in behavior should be observed when subjects later resume the experimental diet. This design controls for 'maturational effects,' i.e., natural changes that occur in behavior over time. It also controls for the placebo effect and the effects of gaining special attention as a research participant. Thus, it enables one to identify changes in behavior that can be specifically attributed to the dietary program.

These fundamental rules of research enable investigators to formulate reliable conclusions and thus have confidence in the validity of their findings. The following sections review diet/behavior studies that may be more closely scrutinized by referring to these principles of scientific inquiry.

**NEUROTRANSMITTERS AND THEIR DIETARY PRECURSORS**

The best-understood transmitters, dopamine, norepinephrine, acetylcholine, and serotonin, are of particular interest, since their production and utilization are dependent upon the availability of essential dietary precursors (Fernstrom, 1981; Wurtman and Wurtman, 1979). Serotonin is a neurotransmitter which regulates psychic processes. Elevated serotonin levels have been associated with schizophrenia, certain forms of drug use, and other related behavioral disorders (Green and Costain, 1981; Woolley and Shaw, 1954). On the other hand, low serotonin levels have been related to sleep disturbances, depression, obesity, and
intellectual deficits (Coppen, 1967; Greenwood et al., 1975; Hartmann, 1977; Shopsin, 1978; Yogman et al., 1985). Of particular interest is the recent research on violence and alcohol use. Many individuals with a history of violence, including violent suicides and murders, have been found to have abnormally low serotonin levels (Brown et al., 1979; Brown et al., 1982; Linnoila et al., 1983; Muhlbauer, 1985). Moreover, alcohol ingestion lowers serotonin. Therefore, alcohol use in certain individuals may provoke violent behavior by lowering brain serotonin levels (Kent et al., 1985; Tarter et al., 1985).

Serotonin is produced in the body from tryptophan (Fernstrom and Wurtman, 1971), an amino acid found in protein foods such as eggs, dairy products, fish, and the muscle tissue of meat and poultry. Studies in laboratory animals show that brain serotonin levels can be influenced by diet and by the administration of pure tryptophan. When carbohydrate is ingested, insulin is released by the pancreas, and blood levels of other amino acids fall relative to tryptophan. As a result, tryptophan is more readily transported into the brain, where it is more available for serotonin synthesis. The administration of protein causes the blood level of tryptophan to fall relative to other amino acids, with the result that less tryptophan crosses the blood brain barrier into the brain. Given serotonin’s possible role in psychiatric and behavioral disorders, manipulation of tryptophan-containing foods, as well as carbohydrate, may help manage these disorders.

The behavioral disorders with which serotonin insufficiency is implicated may be corrected or managed with the administration of tryptophan supplements. Because tryptophan is a naturally occurring amino acid, it is not regulated by the Food and Drug Administration (FDA). It may, however, produce adverse effects (i.e., nausea, headache, dizziness, and drowsiness) when taken in large quantities for a long period of time (Leathwood, 1987). A tryptophan-rich diet or tryptophan supplements may be particularly effective in treating mania, certain types of hyperactivity, depression, alcoholism, and hyperaggression. In fact, tryptophan supplements have been shown to have antidepressant, sedative, intellectual, and anti-aggression effects (Mizuno and Yugari, 1974; Möller et al., 1976; Nyhan, 1976; Weingartner et al., 1983; Young and Sourkes, 1977).

The neurotransmitter dopamine is the precursor of the neurotransmitter norepinephrine. Imbalances in either dopamine or norepinephrine contribute to a variety of behavioral and emotional disorders including depression (Muscettola et al., 1977; Schildkraut, 1965; Snyder, 1980), psychopathy (Levander et al., 1980; Schachter, 1971), schizophrenia (Bellak, 1978; Bird et al., 1977; Van Kammen et al., 1973), and mania (Coppen, 1967; Schildkraut, 1965).

Both of these neurotransmitters rely on tyrosine, an amino acid found in most protein foods. Regulation of tyrosine supplies may aid in the remediation of some disorders (Carlsson and Lindquist, 1963; Homyckiewicz, 1978). For example, current research suggests that depression is frequently associated with depleted levels of dopamine and norepinephrine in the brain. Consequently, researchers are treating depression and related disorders by introducing concentrated forms of tyrosine to augment brain amounts of dopamine and nor-
epinephrine (Alonso et al., 1978; Gelenberg et al., 1980; Green and Costain, 1981).

The neurotransmitter acetylcholine (ACh) is largely responsible for memory, learning ability, and motor coordination (Davis et al., 1979; Green and Costain, 1981; Peters and Levine, 1977; Snyder, 1980). Diminishing brain levels of ACh are characteristic of the aging process and a form of premature senility, Alzheimer’s disease (Bartus et al., 1982; Growden et al., 1977; Marsh et al., 1985; Perry and Perry, 1980). This neurotransmitter also relies on diet for its production. It is synthesized in brain cells from the dietary compound choline. Fish, poultry, and eggs are known as “brain foods” due to their high concentration of choline. However, choline is widespread in both animal and plant products and is commonly found along with the Vitamin B complex. In humans, the amount of choline in the diet determines the amount of choline in the blood that is delivered to the brain (Barbeau et al., 1979; Davis and Berger, 1979). Pure concentrations of the choline complex are being administered to a variety of populations to examine its effects on brain functioning. Improvements in brain activity are expected in normal populations and populations with acetylcholine-related disorders (Davis et al., 1979; Peters and Levine, 1977). Benefits to Down’s syndrome patients have been documented with ACh manipulations (Cantor et al., 1986). Also of particular interest is the finding that ACh systems are directly involved in the production of aggressive and violent behaviors in animals (Ijic et al., 1970; Smith et al., 1970). Choline supplementation may be beneficial in treating learning disabilities and cognitive deficits that have been associated with antisocial behavior.

Neurotransmitter precursor therapies for the treatment of drug abuse have also been tried with some success (Kleber and Gawin, 1986; Rosecan, 1983). Several popular drugs, such as opiates, PCP, and cocaine, deplete levels of dopamine, norepinephrine, and serotonin in the brain. The use of tyrosine and tryptophan for abusers of these drugs aids in the restoration of natural neurotransmitters. Thus the symptoms of withdrawal are minimized, and cravings for the drug are reduced.

The production of powerful brain modifiers has thus been shown to be influenced by the availability of specific dietary components, namely the dietary precursors of brain neurotransmitters. (See Leathwood, 1986, for a review of studies on neurotransmitter precursor therapies with animals and their application to humans.) Studies in laboratory animals show that the administration of pure tryptophan increases serotonin production in the brain. In both animals and humans, ingestion of carbohydrate along with tryptophan enhances tryptophan’s effect and increases the ratio of tryptophan to other amino acids. It is not yet clear whether the increased brain serotonin synthesis following tryptophan administration produces an alteration in neurotransmission with related effects on behavior. There is, however, indirect clinical evidence that specific doses of tryptophan may have sedative properties (Leathwood, 1987). At any rate, it is unlikely that behavioral disorders develop in the United States from neurotransmitter precursor deficiencies, although many such disturbances that
HYPOGLYCEMIA AND REFINED CARBOHYDRATE

Several reports suggest that diets high in refined carbohydrate (e.g., white rice, white flour, sugar, cakes, candies, sodas, and potato chips) can create or aggravate behavioral and learning deficits by disrupting normal brain function in susceptible individuals (Connors et al., 1985; Fishbein, 1982; Green, 1969; Lester et al., 1982; Powers, 1974; Prinz et al., 1980; Spring et al., 1982/83). Results of case studies and quasi-experiments indicate that behavioral disturbances such as depression, irritability, violence, irrationality, hyperactivity, and impaired mental functions normalize following a change to refined-carbohydrate-free diets (Hudspeth et al., 1980, 1981; Langseth and Dowd, 1977; Powers, 1974; Schauss, 1980; Schoenthaler, 1985).

It is believed that the rapid digestion of refined carbohydrate is responsible for its drastic effect on the brain and behavior (see Figure 6). When complex car-

CARBOHYDRATE METABOLISM

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<tr>
<th></th>
<th>COMPLEX</th>
<th>Refined</th>
</tr>
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<tbody>
<tr>
<td><strong>CONVERSION</strong> to glucose</td>
<td>Digestive process slow and stable</td>
<td>Some digestive steps bypassed: predigested</td>
</tr>
<tr>
<td><strong>ABSORPTION</strong> into bloodstream</td>
<td>Glucose absorbed small amounts at a time</td>
<td>Rapid influx of glucose into bloodstream</td>
</tr>
<tr>
<td><strong>REGISTRATION</strong> by brain</td>
<td>Minimal involvement</td>
<td>Excessive increase in glucose: crisis</td>
</tr>
<tr>
<td><strong>RESPONSE</strong> of pancreas</td>
<td>Moderate insulin release to transport glucose</td>
<td>Large insulin release to transport glucose</td>
</tr>
<tr>
<td><strong>BALANCE</strong> by hormones</td>
<td>Minimal involvement</td>
<td>Increased secretion of hormones (adrenal &amp; pituitary)</td>
</tr>
<tr>
<td><strong>RESULT</strong></td>
<td>50% of glucose supplied to brain for fuel</td>
<td>Possible reactive hypoglycemia</td>
</tr>
<tr>
<td><strong>SYMPTOMS</strong></td>
<td><strong>NONE</strong></td>
<td>Physical complaints and psychopathology</td>
</tr>
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Fig. 6—Several stages are involved in the metabolism of carbohydrate. The intake of refined carbohydrate produces variations in these stages as compared with the intake of complex carbohydrate.
Carbohydrate is taken into the body, it is broken down slowly by the digestive process. As the carbohydrate passes from the stomach into the intestine, it is converted to the simpler form of glucose. Glucose is released into the bloodstream and causes the pancreas to secrete insulin. Insulin facilitates the uptake of glucose by cells, where it is used for energy or stored for later use as glycogen or fat.

In contrast, when refined carbohydrates with low fat content are eaten, they pass through the stomach rapidly, since the natural bulk found in carbohydrates has been removed during the refinement process. Consequently, large quantities of glucose pass into the bloodstream, and insulin is quickly released by the pancreas to decrease the blood sugar level. In the body’s effort to control the rapid influx of glucose, excessive amounts of insulin may be released, causing a sharp decline in blood sugar level, which seriously deprives the brain of needed glucose (see Figure 7). Hence the individual feels hungry again and/or craves sweets to raise the blood sugar level, and the process starts over again. As Wunderlich (1982) points out, refined carbohydrate requires little chewing, tastes good, and is quickly digested, causing people to be hungry sooner. Therefore, individuals tend to consume larger quantities, further exacerbating the problem.

If blood glucose falls to subnormal levels, the brain registers a crisis and causes the release of hormones such as adrenalin, prolactin, cortisol, and ACTH (Chalew et al., 1984; Knopf et al., 1977). Increases in these hormones have been associated with irritability, agitation, and anxiety. Also, the neurotransmitter...
miter dopamine rises and exacerbates behavioral and psychological disruptions. These symptoms may compromise individuals' ability to control their behavior.

It is important to note that simple and refined carbohydrates are distinguishable only by their nutrient content. Simple carbohydrate, including fruits and some vegetables that act as simple carbohydrate in the body, i.e., potatoes and carrots (Crapo and Olefsky, 1983; Jenkins et al., 1982), is metabolized in the same way as refined carbohydrate, producing the same variation in blood glucose levels. Simple carbohydrate, however, contains a full complement of vitamins and minerals that refined carbohydrate lacks. Thus, glucose metabolism may be facilitated by essential nutrients.

The chronic condition of low blood sugar is called hypoglycemia. The primary way to treat hypoglycemia is to alter the diet, increasing proteins, reducing intake of refined and simple carbohydrates, and replacing them with complex carbohydrate in the form of vegetables and whole grains (as shown in Figure 8).

The behavioral symptoms that are sometimes associated with the rapid decline in blood sugar characteristic of hypoglycemia are of particular interest to criminologists. These symptoms include fatigue, irritability, nervousness, depression, vertigo, faintness, insomnia, mental confusion, inability to concentrate, anxiety, phobias, dysperceptions, destructive outbursts, headaches, heart palpitations, muscle cramps, convulsions, digestive disturbances, allergies, blurred vision, lack of sex drive in women, impotency in men, and difficulty in performing simple physical or mental tasks (Chalew et al., 1984; Cooper and Pfeiffer, 1977; Engel and Margolin, 1941; Fishbein, 1982; Johnson et al., 1980; Rosenthal and Allen, 1978; Yaryura-Tobias, 1973). Individuals experiencing these symptoms may find it more difficult to cope with the normal stresses of life and may be more likely to act out or engage in certain maladaptive behaviors that attract the attention of the criminal justice system.

Research directly monitoring brain function as it fluctuates with blood glucose levels following a carbohydrate ingestion has shown that hypoglycemia and/or carbohydrate intolerance may produce an abnormal brain response (Chalew et al., 1984; Fishbein and Thatcher, 1986; Hudspeth et al., 1980, 1981). A recent study (Fishbein et al., 1988b) examined normal subjects who were administered one of four carbohydrates-glucose, sucrose, fructose, or corn starch-in various levels of complexity. Blood sugar levels and electrophysiological (EEG) measures of the brain's electrical activity were simultaneously obtained over a 5-hour period to determine the brain's response to carbohydrates of varying complexity. The simplest carbohydrate solutions, glucose and sucrose, caused the most dramatic rise and fall in blood sugar level and produced more substantial electrophysiological abnormalities than the other two solutions. The regions of the brain primarily affected by these glucose shifts, the left parietal and temporal regions, have been associated with learning disabilities and emotional and behavioral disturbances (Thiessen, 1976; Valzelli, 1981; Yeudall et al., 1985).

1 Corn starch is a complex carbohydrate, and fructose is a simple carbohydrate that is not processed by the body in the same way as other sugars.
<table>
<thead>
<tr>
<th>Food Item</th>
<th>Foods Allowed</th>
<th>Foods Not Allowed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beverages</td>
<td>Decaffeinated coffee, Sugar-free sodas, Diluted fruit juice, Milk and milk</td>
<td>Kool-Aid, Sugared sodas, Fruit-flavored drinks, Alcoholic drinks, Condensed milk, Milk shakes, Flavored yogurt, Chocolate milk, Cocoa</td>
</tr>
<tr>
<td></td>
<td>products, Buttermilk, Tea (to a minimum), Vegetable juices</td>
<td></td>
</tr>
<tr>
<td>Meat</td>
<td>14 oz. or more per day (minimize pork intake)</td>
<td>If overweight, less than 14 oz. per day, Breading and fried meats, Thick gravy or sauce</td>
</tr>
<tr>
<td>Meat substitutes</td>
<td>Eggs and milk products, Nuts and seeds, Legumes (beans, peas, and lentils)</td>
<td>If dairy-sensitive, eliminate dairy products</td>
</tr>
<tr>
<td>Fats</td>
<td>Butter and margarine, Unsaturated fat and oil, Light cream cheese or cream</td>
<td>Saturated fats and oils, Salad dressings with sugar, Animal lard</td>
</tr>
<tr>
<td></td>
<td>Low-calorie salad dressings</td>
<td></td>
</tr>
<tr>
<td>Breads and cereals</td>
<td>Emphasize high fiber and low sugar content, Whole grain bread and cereal,</td>
<td>White breads, rolls, muffins, White flour, rice, pasta, Cereals with added sugar, Sweet rolls, Instant cooked cereals</td>
</tr>
<tr>
<td></td>
<td>Pancakes, waffles, crackers, snacks made with whole grains</td>
<td></td>
</tr>
<tr>
<td>Vegetables</td>
<td>Any canned, fresh, or frozen starchy vegetables, such as corn, lima beans,</td>
<td>Vegetables with added sugar</td>
</tr>
<tr>
<td></td>
<td>parsnips, green peas, potatoes, pumpkins, winter squash, and yams should be</td>
<td></td>
</tr>
<tr>
<td></td>
<td>limited</td>
<td></td>
</tr>
<tr>
<td>Fruits</td>
<td>Limited to 2 to 3 servings per day of fresh, sugar-free canned or frozen fruits or juices</td>
<td>Fruits sweetened with sugar, Fruit drinks</td>
</tr>
<tr>
<td>soups</td>
<td>Broths, consomme, bouillon, Soups made with broth and allowed vegetables,</td>
<td>Soups thickened with flour or sweetened</td>
</tr>
<tr>
<td></td>
<td>Cream soups made with allowed milk</td>
<td></td>
</tr>
<tr>
<td>Desserts</td>
<td>Only dishes sweetened with artificial sweeteners and allowed milk</td>
<td>ALL OTHERS</td>
</tr>
<tr>
<td>Sweets</td>
<td>Artificial sweeteners, dietetic sugar, jelly, whole barley syrup, raw honey</td>
<td>Candy, boiled honey, jelly, jam, syrup</td>
</tr>
<tr>
<td></td>
<td>(limited), real maple syrup (limited), Polaner Apple Spread (without sugar)</td>
<td></td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>Salt, pepper, spices, herbs, flavorings, vinegar, unsweetened pickles, olives,</td>
<td>Sweetened pickles, catsup, bread crumbs, croutons, and other sugared condiments</td>
</tr>
<tr>
<td></td>
<td>nuts, mustard, and catsup</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE:** Simple sugars may be “disguised” in the form of sucrose, glucose, dextrose, corn sweetener, corn syrup, corn starch, molasses, brown sugar, and honey. For individuals who are “milk sensitive,” all milk should be eliminated, as even relatively small amounts may have adverse effects.

Fig. 8-Dietary regimen for hypoglycemia and carbohydrate intolerance. The treatment for hypoglycemia, when medication is not indicated, includes a diet high in complex carbohydrate and proteins. Simple and refined carbohydrate should be kept to a minimum.
A study by Lester et al. (1982) indicated that refined carbohydrate consumption affects learning and intelligence. Specifically, significant relations were shown between refined carbohydrate intake and brain function measures in children. As shown in Figure 9, these researchers found a negative correlation between refined carbohydrate consumption and intelligence and school achievement. In other words, children whose diets were high in refined-carbohydrate tended to have lower IQ scores than those whose diets were low in refined carbohydrate. The study controlled for such possibly confounding variables as race, sex, socioeconomic status, and age.

There also appears to be a relationship between alcoholism and hypoglycemia. The incidence of hypoglycemia is prevalent among individuals with drinking problems or alcoholism (Williams, 1981). Alcohol is a simple carbohydrate and is absorbed and metabolized by the body in nearly the same way as refined carbohydrate. An individual prone to hypoglycemia is more likely to find relief from the condition by drinking alcoholic beverages which rapidly elevate blood sugar levels. Furthermore, excessive alcohol intake provokes hypoglycemia in the same manner as overconsumption of simple carbohydrate (Sereny et al., 1975). Drinking produces a large increase in blood glucose, thus heightening insulin release. Continuous bouts of drinking will stress the pancreas' ability to secrete insulin, and hypoglycemia, or even diabetes, may result. During periods of abstention, alcoholics are known to binge on sugary foods. This behavior serves as a substitute for drinking and worsens the metabolic condition of
alcoholism. A dietary regimen for the treatment of hypoglycemia is essential in any program designed to treat problem drinkers. This regimen will help to reduce cravings for alcohol, replenish vitamins and minerals that become deficient, and revitalize glands and organs damaged by alcohol.

Virkkunen (1986) found that male offenders who were diagnosed as violent and impulsive had abnormal insulin and blood glucose responses to a glucose tolerance test. These individuals tended to have a higher rate of hypoglycemia (as indicated by lower levels of blood glucose) than did a group of normal male controls. In addition, their insulin response to the glucose challenge was exaggerated. As discussed previously, low levels of brain serotonin have been found in habitually violent and impulsive offenders. It is possible that chronic high intake of simple sugars produces stress on the glucose metabolic system, causing an initial increase in serotonergic activity followed by a subsequent downregulation of serotonergic activity. Due to the known links among serotonin metabolism, insulin secretion, and carbohydrate intake (Femstrom and Wurtman, 1971, 1972), dietary intake of carbohydrate, particularly alcohol or sugars, may be implicated in the development of violent or impulsive behavior.

These data suggest that hypoglycemia or glucose intolerance may contribute to deviations in brain function that precipitate problematic behaviors. Behavioral disruptions observed following refined carbohydrate intake may be due to complex biochemical changes rather than simply low blood glucose levels. This would explain why only a subpopulation of individuals are affected by refined carbohydrate consumption and why low glucose levels and symptoms are not always correlated (Chalew et al., 1984; Hudspeth et al., 1980, 1981). Individuals who manifest irritability, aggression, or learning deficits and are susceptible to a metabolic condition influenced by a faulty diet may be properly diagnosed and treated with a regime that includes dietary modifications.

**DIETARY TRACE ELEMENTS**

Trace elements are another factor that may influence brain functioning and behavior. The trace elements in our diets include nutritive minerals essential for proper growth, development, and functioning of the human body, i.e., zinc, calcium, magnesium, selenium, chromium, iron, and potassium. Studies show that when diets are deficient in these nutrients, learning ability and behavioral responses are impaired (Benignus et al., 1980; Silbergeld, 1982; Tucker and Sandstead, 1982). Other trace elements, however, are toxic, and high concentrations can impede proper human development and functioning. Some of the toxic trace elements found in food are lead, cadmium, arsenic, aluminum, and mercury. Humans do not have the body mechanisms to excrete these toxins, which therefore accumulate in the body and brain, exerting their effects on individuals’ physical and mental health.

The evidence strongly suggests that toxic trace elements can directly influence behavior by impairing brain function, influencing neurotransmitter production and utilization, and altering metabolic processes (Moore and Fleischman, 1975; Otto et al., 1981; Silbergeld, 1982; Thatcher et al., 1982, 1983a, 1983b). Many
toxins are found in foods most people ingest daily. These toxins diminish levels of essential minerals and vitamins and may be closely associated with the prevalence of learning and behavioral problems (Lester and Fishbein, 1987; Thatcher and Lester, 1985).

A major source of one toxin, cadmium, is refined carbohydrate (Friberg et al., 1971; Lester et al., 1982; Sharma, 1981). Figure 10 shows that as the consumption of refined carbohydrate increases, levels of cadmium, measured in hair samples, also increase significantly.

The processing method reduces the nutritive value of the food and breaks down its molecular complexity, while introducing toxic elements into the diet. The negative relationship between intellectual development and rate of refined carbohydrate ingestion may be partly due to the effects of toxic trace elements found in highly processed and refined foods. Furthermore, cadmium affects glucose metabolism by accumulating in the liver and kidney and disrupting glucose regulation (Merali and Singhal, 1981).

Chronic exposure to even extremely low levels of both cadmium and lead has a deleterious effect on brain function. The effect is particularly insidious because it can be present without any overt symptoms. For example, the presence of lead and cadmium (body burdens) has been related to impairments in brain

![Graph showing linear regression of hair cadmium (log_{10} transformed) as a function of the proportion of refined carbohydrate foods in children's diets. The shaded area represents one standard error of estimate about the regression line. (From Lester et al., 1982.)](image)
function manifested as learning disabilities, particularly in measures of school achievement, verbal and performance I.Q., hyperactivity, and mental dullness (Lester et al., 1982; Pihl and Parkes, 1977; Thatcher and Lester, 1985). As seen in Figures 11 and 12, lead and cadmium burdens are significantly associated with deficits in performance and verbal I.Q., respectively. A large body of research has also demonstrated that lead intoxication is significantly associated with hyperactivity and violence (Benignus et al., 1980; Needleman et al., 1979; Oliver, 1976).

The presence of nutritive minerals protects the body from the insidious effects of toxic dietary trace elements. For example, calcium serves to minimize the damage lead causes to the body. Children with both high calcium and high lead levels are less likely to experience deficits in intellectual development (Thatcher and Lester, 1985). However, those with low calcium and high lead show the largest decrement in development. Similarly, zinc can counteract the deleterious effects of cadmium (Buell, 1975; Schroeder, 1973). It is well known that both zinc and calcium have a chelating function, i.e., they cleanse the body of toxins and insulate organs, including the brain, from the destructive impact of these toxins. The current diet of a substantial portion of the U.S. population is severely lacking in beneficial and protective nutrients and is high in refined carbohydrate. Therefore, large subgroups of individuals may be “malnourished” (Davis, 1983). Toxins damage organ systems, reduce the ability of the brain to function properly, delay or impair intellectual development, and interfere with the regulation of behavior.

**FOOD SENSITIVITIES AND ALLERGIES**

Reports that some children become irritable, anxious, and hyperactive after eating certain foods have appeared in the medical literature since 1908 (Alvarez, 1946; Shofield, 1908). More serious reactions to certain foods, e.g., headaches (Ghose and Carroll, 1984; Randolph, 1944), muscle pain (Randolph, 1951), arthritis (Randolph, 1959; Rinkel et al., 1951), fatigue (Crook, 1980; Randolph, 1947; Speer, 1970), aggression (Moyer, 1975, 1976), and even schizophrenic reactions (Dohan, 1978; Philpott, 1977a; Singh and Kay, 1976; Moyer, 1976), have been noted in susceptible individuals. However, these reactions have historically been considered to be idiosyncratic and quite rare.

Recent studies provide evidence that certain neuropsychological disorders provoked by foods or food constituents may be more common than previously believed (Crook, 1975; Egger et al., 1985; Feingold, 1975; Rapp, 1981; Speer, 1975). These disorders may be manifested as either allergic or pharmacologic reactions. An allergic reaction is the body’s defense against a substance that is harmless to most people. An allergy can have a relatively benign effect, e.g., a runny nose, or a more serious effect, e.g., chemical imbalances in the brain. When the brain is affected, behavioral disorders may result (Randolph, 1974; Rapp, 1981). A pharmacologic reaction to food occurs when foods act like drugs in the brain and interfere with normal neurochemical activities (Augustine and Levitan, 1980; Ghose and Carroll, 1984; Swanson and Kinsbourne, 1980).
Fig. 11—Second-order polynomial regression describing the relationship between log$_{10}$ hair lead concentration (independent variable) and full-scale I.Q. The vertical line represents two standard errors of estimate, R-square equals the amount of variance accounted for, and power (1 - beta) equals $P < .001$. (From Thatcher et al., 1983a.)

Fig. 12—Polynomial regression curves with performance and verbal I.Q. as the dependent variables and log$_{10}$ cadmium as the independent variable. R-square equals the percentage variance accounted for, and power is the probability of replication (1 - beta) at $P < .05$, given the sample size. Alpha equals $P < .001$. (From Thatcher et al., 1983b.)
Foods commonly identified as ‘allergens’ by U.S. clinicians are abundant in the Western diet. They include milk, chocolate, cola, corn, eggs, peanuts, citrus fruits, tomatoes, wheat and small grains, and artificial food colors (Feingold, 1975; Lackey, 1972; 1976; Rapp, 1981; Speer, 1975). Common reactions include swelling of extremities, swelling of the vascular system, irritability, agitation, headaches, confusion, intestinal complaints, bedwetting, fatigue, hyperactivity, seizures, and behaviors that are out of character for the affected individual. Blood glucose levels may also fall, thereby producing symptoms of hypoglycemia (O’Banion, 1981; Rapp, 1981).

Additional clinical reports suggest that food allergies may influence neurotransmitter systems (Buckley, 1972; Crayton, 1986; Ghose and Carroll, 1984; Rapp, 1981; Weiss and Kaufman, 1971; Williams and Kalita, 1977). The activity of serotonin may be reduced in persons experiencing allergy-type reactions. If this anecdotal evidence becomes scientifically verified, it will be noteworthy in light of research indicating that low serotonin is related to violent behavior and depression (Brown et al., 1979, 1982; Linnoila et al., 1983; Muhlbauer, 1985).

Food constituents that may elicit a pharmacological reaction include phenylethylamine (as found in chocolate), tyramine (as found in aged cheese and Chianti), monosodium glutamate (a flavor enhancer often found in packaged and Chinese food), xanthines (e.g., caffeine), and aspartame (an artificial sweetener known as NutraSweet). Each of these has been associated with excitatory changes in the brain, contributing to hyperactivity and/or learning difficulties. Aspartame, which has been approved for use by the Food and Drug Administration, has been shown to block the synthesis of serotonin (Wurtman, 1983). As a result, some individuals experience mania, shortened attention span, distractibility, and impaired problem-solving ability (Drake, 1986; Walton, 1986). Phosphates, another class of additives that aid in the processing of foods and control their acid balance, are found pervasively in beverages, oils, baked goods, soft drinks, and fruit products. Phosphates have been associated with hyperactivity in certain children; the removal of phosphates from the diets of those children has resulted in improvements in behavioral symptoms (Walker, 1982).

Two case studies reported by Rapp (1981) illustrate the relationship between food allergies and antisocial behavior. The first subject, Donald, was an eleven-year-old black male. His teachers complained of his disruptive and rude behavior. He had no friends. He was moody and always angry. He could not fall asleep at bedtime and went to the bathroom several times each night. He was irritable, hyperactive, unhappy, restless, and hostile. His physical complaints included backaches, muscle aches, cracked skin, stuffy nose, and puffy eyes. He had been treated with drugs intermittently for five years, without benefit.

To evaluate the role of foods in Donald’s hyperactivity, he was placed on a diet that contained no milk, wheat, eggs, cocoa, corn, sugar, or dyes (foods which are commonly associated with allergies). In 24 hours, he was slightly better; a week later, he had responded so dramatically that his parents were astonished. He was more manageable and more pleasant, His speech was more coherent,
and he seemed calmer. His physical symptoms disappeared, although a stuffy
nose reappeared occasionally. His scores on activity level, reflective of hyperac-
tivity, normalized.

After six months, each of the omitted foods was reintroduced into his diet, one
at a time. He had no difficulty until he ate eggs and artificially dyed foods,
which resulted in the onset of symptoms his parents described as “violent.” He
became uncontrollable; he talked rapidly; his eyes were glassy and puffy; his
face was flushed. The tension reappeared and he complained of a headache.
He was given capsules containing milk powder and he did not react. He was
given egg-containing capsules and the symptoms returned. He has since been
maintained on an egg- and dye-free diet, and two and one-half years later, his
activity scores remain normal.

The second case study was that of Stephen, a four-year-old who manifested
‘nasty, violent, aggressive behavior.” He had temper tantrums continuously,
and he hit his mother, punched others, and screamed and cried frequently. He
did not sleep well, and he woke up repeatedly throughout the night. He
became fatigued easily and experienced drastic mood changes. The physical
symptoms from which he suffered included a chronic cough, hives and asthma,
sinus congestion, frequent infections, and daily vomiting. In addition, he craved
milk and cheese. This history is typical of individuals experiencing food aller-
gies.

Stephen was placed on a milk-free diet for two weeks, and noticeable improve-
ments occurred. He no longer screamed and yelled. He slept through the night
and seemed relaxed. The vomiting and coughing ceased, and the violent out-
bursts disappeared. When milk products were mistakenly reintroduced, both
the behavioral and physical symptoms returned. To confirm the source of these
symptoms, a double-blind case study was conducted, and the finding that milk
was associated with Stephen’s behavioral problems was supported. Two years
later, Stephen was able to ingest small amounts of milk products without
adverse reactions. However, larger amounts still caused the symptoms to return.

Another area of recent interest in behavioral nutrition is the relationship
between sucrose sensitivity and hyperactivity. Gross (1984), for example, identi-
fied an irritable and hyperactive patient who was hypersensitive to sucrose. To
investigate the prevalence of this problem, Gross studied 50 hyperactive chil-
dren whose mothers reported that the children’s behavior was associated with
sugar intake. None of the children showed any consistent response to sucrose.
Gross concluded that although a hypersensitivity to sucrose can exist and lead to
behavioral problems, sucrose-induced hyperactivity is not common and may
affect only a subpopulation of hyperkinetic children.

Similarly, Kruesi (1986) studied the effect of carbohydrate intake on children’s
behavior. He concluded that “thus far in children, little if any cognitive or
behavioral effect of an acute dose of sugar has been demonstrated. Although
rare isolated individuals may show adverse, behavioral responses to sucrose,
acute doses seem to have little effect upon behaviors thus far examined.” (See
Crayton, 1986, for a review of food sensitivities, immune function, the brain, and behavior.)

Detection of food allergies and pharmacological reactions is a difficult task, and identification of the responsible food or constituent can be tedious. Important clues to isolating the offending food are food cravings, pulse changes, the time of day symptoms appear, and where meals are eaten (e.g., at school or at home) before the onset of symptoms. Other laboratory tests involve challenges with the suspected food allergen, i.e., sublingual (under the tongue) and subcutaneous (under the skin) tests and the RAST (Radio AllergoSorbent Test). The RAST is an immunological assay for the presence in a person’s blood of antibodies against specific test antigens. These tests should be conducted under the supervision of an allergist or medical professional for safety and accuracy.

An “elimination diet” is considered to be the most accurate method of detecting food allergies. A food diary is kept for at least two weeks. Foods in the diet that are associated with the onset of symptoms and thus may be possible sources of allergy or sensitivity are then eliminated from the diet for a period of time, usually three weeks.

Onset of symptoms following a one-at-a-time reintroduction of the food or foods that were eliminated signals a sensitivity. The individual should then avoid the food that produced the reaction for at least six months, at which time it may be reintroduced in small quantities to determine whether a sensitivity still exists (see Hippchen, 1978; Rapp, 1981). Before measures such as elimination diets are employed in attempts to isolate food allergens, immunological tests should be performed to exclude the possibility of endocrine or other systemic disorders.

**FOOD DYES AND OTHER FOOD ADDITIVES**

Benjamin Feingold’s studies are perhaps the best known and most controversial work on food sensitivity and hyperactivity (Feingold, 1975, 1976, 1977). Feingold has asserted that between 30 and 60 percent of childhood hyperactivity is due primarily to central nervous system reactions to artificial food coloring, artificial flavors, and natural salicylates. He received considerable press coverage and publicity when he claimed that the disorder could be cured with a special diet devoid of these additives. His findings were hailed as the new panacea for hyperactivity.

Subsequent research has produced conflicting findings, indicating that the excitement generated by Feingold’s theory and cure may have been premature (Connors et al., 1976; Harley and Matthews, 1980; Swanson and Kinsbourne, 1980). It appears that the “cure” was transitory and may have been due to the placebo effect of ritualized, special food-buying and cooking techniques associated with treatment. A number of controlled, double-blind challenge experiments in which neither experimenter nor patient knew whether food additives
or placebos had been administered did not support the Feingold hypothesis (Connors et al., 1976; Lipton et al., 1979; Sobotka, 1978; Wender, 1977).

On the other hand, the results of some more recent studies have strongly supported the food-additive/hyperactivity hypothesis (Egger et al., 1985; Swanson and Kinsbourne, 1980; Weiss et al., 1980). Several methodological differences between these more recent experiments and earlier studies provide insight into some of the reasons for the conflicting experimental results.

The Swanson and Kinsbourne (1980) study differed in three respects from previous controlled double-blind, crossover challenge experiments to test the relationship between hyperactivity and food-dye ingestion. First, Swanson and Kinsbourne used a 100-mg or 150-mg dose of food dye as a challenge, whereas previous studies used food-dye challenge doses of 1 to 26 mg. Swanson and Kinsbourne speculated that such low dosages may have been below the sensitivity threshold for the children who participated in the study, particularly since the 1976 estimate of average daily intake of synthetic food dyes by children between 5 and 12 years of age was 76.5 mg (Sobotka, 1978). Second, most previous experiments used behavioral rating scales as the assessment instrument, but Swanson and Kinsbourne used a paired-associate learning task, which they maintained was more sensitive to food-induced effects, since attention/learning deficits are a central attribute of the hyperactivity syndrome (Rosenthal and Allen, 1978). Third, Swanson and Kinsbourne categorized their hyperactive subjects into two physiologically similar groups for comparison of food-dye effects on cognitive function. Specifically, 20 children were identified as hyperactives who responded positively to amphetamine treatment; another 20 children demonstrated adverse or no positive behavioral effects in response to the drug. In the dye challenge experiment, 85 percent of the positive drug responders showed adverse effects after eating food dye, whereas only 25 percent of the nonresponders showed evidence of food-dye sensitivity.

This finding is particularly pertinent in evaluating previous experiments that based their conclusions of a no-dye effect on small samples of hyperactive children. According to the Feingold hypothesis, not all hyperactive children should be expected to respond to a synthetic-dye challenge. The statistical implications of the hypothesis that only subpopulations are sensitive to dyes and additives have recently been described (Weiss, 1983). To expect a statistically significant effect from a small sample is inappropriate when only 30 percent of a heterogeneous group of hyperactive children are expected to respond. “A population containing 30% responders would require a sample of 265 subjects to be certain of finding a statistically significant difference 90% of the time, even if the susceptible members of the group, on the whole, responded as much as 1 standard deviation . . . beyond the control mean” (Weiss, 1983:35).

Egger et al. (1985) took a different experimental approach in their study, which is of major interest. The Egger et al. study demonstrates the complexities of identifying specific provoking food constituents for affected children. The study involved several phases. The first was a four-week baseline nonreactive diet phase; the second introduced provocative food constituents to test their effects;
and the third employed a controlled double-blind, crossover trial in which an active constituent and a placebo were administered for a subgroup of subjects who evidenced a sensitivity reaction in the second phase.

Since the group of children had been originally referred for suspected food-based behavior problems, the fact that 81.5 percent of them demonstrated one or more food reactions in the second phase is probably not surprising. The results of the controlled trial phase of the experiment were based on 28 subjects. The cognitive and behavioral rating-scale results for these children showed statistically superior performance during the placebo trials, as compared with the active-agent trials. (For only one measure of generalized motor activity was there no difference between trials.) Over 40 foods or food constituents were found to evoke neuropsychological reactions in the children. Significantly, neither dyes nor preservatives alone were offensive, but in combination (as they often exist in the food supply), they produced an adverse behavioral reaction in 79 percent of the children.

**DIET AND BEHAVIOR STUDIES IN THE JUSTICE SYSTEM**

The studies described above strongly indicate the need for further investigation into the link between diet and behavior. There is ample evidence that dietary factors do influence behavior in known ways. The evidence is not clear, however, when these findings are applied to antisocial behavior and criminality. Nevertheless, inadequate dietary habits do appear to act as risk factors in the development of maladaptive behavior. The extent to which diet affects this developmental process has yet to be determined. Studies of the effects of dietary changes on institutionalized offenders may help to identify individuals whose behavior is influenced by diet and to assess the prevalence and severity of the problem. Some of the ongoing research efforts to evaluate the diet-behavior phenomena in a correctional setting are described below.

D’Asaro et al. (1975) recruited 44 jail inmates for an experiment in which the inmates were placed on modified diets (reduced refined carbohydrate) and given either vitamin supplements or a placebo. The subjects were also given classroom instruction on nutrition. The authors concluded that the vitamin-supplemented diets resulted in an overall improvement on most psychological test scores. Unfortunately, 25 of the subjects (57 percent) dropped out of the study, leaving only seven inmates in the control group. The small number of cases (16 experimental and 7 control) precluded any definitive conclusions. Further, the differential dropout rate between experimental and control groups raises the possibility of extensive confounding of effects.

Most of the psychological measures showed similar gains for the vitamin and placebo groups, both of which received nutrition courses and ritualized procedures associated with the administration of pills. Only the no-treatment group, which did not receive the pills or nutritional program, failed to show any gains. These data suggest a positive testing response to the demand

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Portions of this section appeared previously in Pease and Love, 1986.
characteristics of the experiment, including nutritional training, and gains on the test scores cannot be clearly attributed to the vitamin treatment. A competing interpretation of these findings may be that both the treatment and placebo groups were responding to the reduction of refined carbohydrate in the subjects' diets. Moreover, the statistical analysis compared changes in test scores within groups but not among groups. The conclusions regarding differential performance of vitamin and placebo groups, then, were not supported by the statistical analysis. Finally, the study covered an eight-week period, offering only a limited observation of the possible effects of a new dietary regimen.

Fishbein (1982) reported a well-controlled, if limited, study, using 104 volunteer inmates at a Florida institution for offenders with drug dependency. The participants were prescreened (by self-reported data) to determine the extent of refined carbohydrate in their diet and their symptoms of hypoglycemia. The resulting two groups, “hypoglycemic” and “nonhypoglycemic,” were each randomly divided into two additional groups, experimental and control. A propensity for perceptual deficits and maladaptive behavior was measured by the Hoffer-Osmond Diagnostic Test (Osmond et al., 1975) before and one month after the implementation of a diet with a greatly reduced level of refined carbohydrate.

The results showed a significant reduction in indices of maladaptive behavior for the experimental group, which previously had diets high in refined carbohydrate and exhibited symptoms of hypoglycemia. The experimental group without symptoms of hypoglycemia and low refined-carbohydrate-intake histories did not show improvement, indicating that the dietary change itself did not produce differences. As noted by Fishbein, the study suggested, but did not clearly demonstrate, a change in behavior because the dependent variables were limited to psychological tests rather than physiological measures of glucose tolerance. There were also problems in ascertaining the degree to which inmates in the experimental groups maintained the dietary regimen.

Schoenthaler has conducted a majority of the studies on the impact of dietary changes on the behavior of institutionalized offenders. Two reports deal with a study conducted at a Virginia juvenile center (Schoenthaler, 1982, 1983a). The composition of the meals served at the center, which houses 12 to 15 offenders at one time, was purportedly modified to reduce the level of refined carbohydrate. In addition, vended soda and candy were replaced by fresh fruit, fruit juice (which contains simple sugars), and vegetables. Schoenthaler (1982) compared the performance of juvenile inmates at the facility before the revised diet was implemented with that of another group of juveniles who resided in the facility afterwards. The use of two separate intact groups was necessitated by the short incarceration periods of the inmates in the center (an average of one month).

In this study, the major dependent variable was behavior (institutional infractions) rather than psychological test scores, a clear improvement over the D'Asaro et al. (1975) and Fishbein (1982) experiments. However, as in the D'Asaro study, there was no way to differentiate the effects of diet modification
from the possible demand characteristics associated with the implementation of the study. Modification of their diet and available snacks was certainly a significant change in the inmates' environment. Prison inmates are sensitive to any change in their daily routine. Thus, Schoenthaler's claim of having conducted a "double-blind" experiment was unfounded—as previously noted, in a double-blind experiment, neither the experimenter nor the subjects know who is in the experimental group and who is in the control group, although both groups usually know that an experiment is being conducted. Schoenthaler was actually referring to his attempt to conduct a deception experiment, where subjects may not know they are in an experiment and/or they may not know the "real" purpose of the experiment.

Schoenthaler attempted to deceive both inmates and staff by stating that changes in diet were due to financial considerations, i.e., that the new food was cheaper. It is also important to note that the detention facility's director, Frank Kern, stated that other changes in the institution took place simultaneously with the changes in diet (Kern, 1987). New special education and counseling programs were implemented, with particular emphasis on improving the residents' self-concept.

Schoenthaler used a within-groups t-test\(^3\) to compare the effects of the two treatments. However, inferential analysis of this type was inappropriate because the groups were not randomly assigned. They were two intact groups whose members were in the institution before, during, or after the treatment was implemented. The two groups were not clearly defined, in that both treatments included overlapping and nonoverlapping memberships in the two groups. The t-test procedure does not have provisions for such circumstances, so the correlated t-test results are nullified because a majority of inmates did not experience both treatments.

The short observation periods in the experimental treatment (2 to 51 days) also did not allow sufficient time for the experimental diet to generate observable effects. Nearly one-third (7) of the 24 experimental inmates were housed in the youth center for 8 days or less. Over half (13) were in the center 2 weeks or less. But at least 2 weeks are required for the body to adapt to dietary modifications of that magnitude; predicted improvements in behavior should not occur in less time.

The fact that the experimental program was implemented with a majority of inmates who entered the institution after the control group was discharged suggests the possibility that the groups may have differed in other ways, beyond the independent variable. Campbell and Stanley (1966) have described several threats against the validity of experiments under these circumstances. For example, the fact that the control group was incarcerated longer (mean = 28.2 days) than the experimental group (mean = 18.9 days) suggests the possibility that behavioral differences may have resulted from the characteristics of juveniles given longer sentences (i.e., the long-term offenders presumably committed

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\(^3\) A t-test is a method for determining whether there is a statistically significant difference between the mean scores of two groups, e.g., an experimental group and a control group.
more serious offenses and had more exposure time in which to engage in infractions). Schoenthaler was unable to provide any data regarding offenses or other criminal history data to address this problem but argued that, according to Goffman (1961), adjustment to institutional life improves over time. By this reasoning, the control group results should be "biased against" the study. However, the control group showed a weak positive relationship between time at the center and number of infractions-contrary to Schoenthaler's expectation. Further, Goffman's assertion refers to an adjustment period of several months, while Schoenthaler's control subjects were institutionalized for an average of one month.

In his second study, Schoenthaler (1983a) increased the number of participants to 276 juveniles by expanding the time frame in which data were collected. However, the other problems of the first study were not resolved. An additional confounding variable in the later study is the presence of females, who were admitted to the institution after the experimental treatment was begun. It is generally accepted in the correctional community that the presence of female staff members and female inmates helps reduce the likelihood of male inmates exhibiting aggressive behavior. But Schoenthaler reported that even male inmates who were in the institution before it became coeducational evidenced an improvement in behavior.

Schoenthaler attempted to differentiate effects of diet by type of offense committed by the juvenile, i.e., violent, property, or status. The group receiving the experimental diet showed lower levels of antisocial behavior, regardless of type of offense. Unfortunately, he did not address the issue of differences in exposure time, and he thus failed to resolve the problems raised in the first study.

The two Virginia juvenile detention studies were later reanalyzed by Schoenthaler (1984). This time, the statistical analyses were greatly improved by using a time-series procedure. However, the specificity of the independent variable remains in question. It has already been noted that the diet modification represents such a substantive change in the inmates' daily routine that it is difficult to conduct a true double-blind study. Schoenthaler noted that the superintendent of the detention center was a diet enthusiast, suggesting that the administrator had a particular interest in finding significant results. Further the study does not provide an independent assessment of changes in actual dietary intake. The shortcomings of the Virginia studies, with the exception of statistical fallacies, remain.

Other replications of the diet/behavior relationship (Schoenthaler, 1983b-e) suffer from similar difficulties. A study conducted in Alabama (Schoenthaler, 1983e) was based on a multiple interrupted time-series design. A group of 488 incarcerated juveniles was measured under baseline conditions over a 6-month period; the group was then given a 6-month low-carbohydrate experimental diet; this was followed by a 6-month period at the baseline (control) condition. There was no change in the rate of reported incidents from the initial baseline condition during the experimental diet condition. However, there was a slight increase in incidents after return to the baseline condition. Schoenthaler
reported an increase from a mean of 0.039 to 0.061 incidents per day, which represents an increase in mean annual rates from 14.6 to 22.3 incidents per year. These fluctuations fall within the normal variations in a prison population.

Another problem in the experimental design is a failure to control for seasonal changes in rates of reported incidents (McCleary and Hay, 1980). While the Alabama study is a step in the right direction, it remains to be seen whether the changes observed are a function of the diet or of other competing variables. Schoenthaler indicated that there was, for example, an increase in the number of violent offenders during the treatment phase.

**RECOMMENDATIONS TO JUSTICE PRACTITIONERS AND ADMINISTRATORS**

Although the evidence supporting the efficacy of dietary intervention in reducing antisocial behavior among incarcerated populations is weak, it is still possible that the effects of diet are real and that antisocial behavior may, indeed, be reduced in the institutional setting. A second possibility is that only certain individuals who are susceptible to the effects of dietary trace elements, hypoglycemia, food allergies, etc., will be helped by dietary modifications. A third possibility is that diet has little or no observable effect on the behavior of incarcerated offenders. The determination of which of these possibilities is correct warrants serious scientific investigation. The research would present a minimum of risk to inmates or institutions, and if behavior could be shown to be significantly affected by diet, the benefits would certainly outweigh the costs.

The research should be conducted by an interdisciplinary team that includes a psychologist or criminologist, a nutritionist, a physician, a correctional research specialist, and a neurophysiologist. The ideal research study would involve a medical examination of all inmates to identify individuals who are hypoglycemic, who suffer from nutrient imbalances or food allergies, and who have high body burdens of toxic trace elements. The independent variable (i.e., change of diet) must be clearly defined and operationalized, and the intake of food must be more reliably measured. Dependent measures of behavior should include both institutional variables and individual variables. Institutional variables include incident reports, sick calls, and a measure of the residents' view of the institutional milieu. Individual measures include test scores on psychological tests, IQ scores, behavioral measures, and physiological evaluations. Other variables that must be controlled for their influence on behavior include criminal/delinquent history, age, sex, race, educational level, achievement level, and competing institutional policies.

The design recommended for this study is the time series a-b-a-b design conducted over a four-year period. This interrupted time-series with multiple replications (Cook and Campbell, 1979) provides a very powerful demonstration of the effects of the independent variable. Individuals would receive in sequence a standard diet currently in use in the institution (referred to as treatment a) and a special diet that is low in refined carbohydrate and sugars (treatment b). The standard diet would be presented for one year, followed by the special diet for
one year. The same sequence would be repeated over the second two years. The one-year observation period in each diet condition is required because both physical and behavioral measures fluctuate as a function of seasons. The annual cycles of treatments, for example, provide a means to control for differences in incident rates in the summer and winter. If the dietary changes result in consistent improvements in overall inmate behavior under administrations of the special diet condition and consistent deterioration in behavior during the administration of standard diets, then it can be concluded that diet has an effect on overall institution performance. If, on the other hand, identified categories of inmates show improvement and deterioration of behavior during the special and standard diets, respectively, and other groups of inmates do not, then the second proposed possibility is supported—that is, only certain individuals are influenced by changes in diet. Finally, changes in diet not accompanied by consistent changes in overall inmate performance or in identified categories of inmates (e.g., medical categories such as hypoglycemic) support the third possibility. According to the last scenario, the behavior of inmates would not be found to be substantially influenced by the food they eat.

Recommendations to administrators must await the results of such a study. A few recommendations, however, are warranted on the basis of the limited information currently available:

1. A state-level task force comprising physicians, nutritionists, economic planners, and correctional specialists should critically evaluate the diets offered inmates in prisons, adult and juvenile detention facilities, and other care facilities. Appropriate adjustments to the foods offered inmates should be geared to ensure balanced diets.

2. Facilities housing inmates should provide nutritional education to ensure individual investment in the intake of nutritionally balanced meals.

3. Intake screening of all individuals should include assessments of nutritional status during a general medical checkup. This portion of the intake physical should be conducted by a certified nutritionist or at least under the guidance of a trained nutritionist who serves as a consultant. When the results of nutritional screening indicate a need, remedial dietary interventions should be planned and conducted by staff or contract nutritionists.

4. Court-ordered psychiatric assessments to determine offender responsibility and/or competence should include nutritional assessments.

5. The treatment regime for alcoholics should include training and assessment of dietary intake and the incidence of hypoglycemia.

CONCLUSION

This overview of diet/behavior phenomena is not exhaustive; however, the studies reviewed are the most prominent in the fields of nutrition and the behavioral sciences. We have attempted to include those research reports that potentially affect the criminal justice system, in our efforts to understand and manage antisocial behavior.
Interest in this relationship is burgeoning, as is the research. New findings will eventually replace the old, and the field will gain more sophistication and recognition. We cannot at this point draw definitive conclusions concerning the effects of diet on antisocial or criminal behavior, particularly with respect to causality. The studies mentioned herein have not provided adequate data to warrant such conclusions. Nevertheless, we do feel that current findings justify further attention to the possibility of a diet/behavior link that is relevant to the criminal justice system.

Many of the studies reviewed here limited their inquiries to individuals who have already exhibited problem behaviors (e.g., prison inmates). These individuals were then retrospectively determined to possess certain characteristics that are less prevalent in a control group of “normals.” For example, Virkkunen (1986) examined aggressive males and concluded that they had a higher rate of glucose intolerance than nonaggressive individuals. But this does not provide a basis for determining whether aggressive behavior is caused by glucose intolerance.

It would be instructive to conduct a longitudinal study to determine what percentage of a large group of hypoglycemics evidenced aggressive behavior and to compare their performance with that of an equally large group of nonhypoglycemics. If the difference were significant, we might be able to generalize to the relationship between hypoglycemia and maladaptive behavior. Other aspects of nutrition, such as the effects of toxins, neurotransmitter imbalances, additives, etc., should also be investigated in correctional settings.

The policy initiatives delineated above reflect the present limitations and shortcomings in this topical area. These initiatives emphasize the need for future research and substantial conclusions. The criminal justice system is particularly vulnerable to “quick and dirty” solutions that have not been adequately tested; thus, we suggest that history not be repeated. Indeed, the puzzle is too complex at this point for us to present easy solutions. It is hoped that more sophisticated research that adequately controls for the complexities of human bodily functions and the multifaceted aspects of behavior will provide more useful information about the contributions of biological variables to behavior.
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You are what you eat. This bromide has normally been applied to those who are concerned about their weight and body appearance or their general physical health. In this monograph, Dr. Diana Fishbein, of the University of Baltimore, and Dr. Susan Pease, of North Carolina Central University at Durham, examine other implications of diet and food intake. They have studied the literature and past research projects directed at the notion that food directly affects emotional, psychological, and deviant behavior.

Their study includes a review of the physiological dynamics of the human body and its reaction to food substances, including dyes. It provides a brief review of the brain’s operation and its reaction to toxic trace elements, as well as reactions to carbohydrates, glucose, etc. This research would indicate that while there is scientific documentation and evidence of measurable reaction in the brain to these substances, most of the research projects fall short of providing solid evidence that proves dietary elements have an effect on deviant or observable illegal behavior.

In the much-publicized and now famous “Twinkies” defense used at the trial of the accused murderer of San Francisco’s mayor and a city councilman, the court was presented with evidence purporting to prove that the killing was directly related to diet and the physiological imbalance of the killer. For the most part, the public scoffed at this as a legitimate defense.

Drs. Fishbein and Pease, after an exhaustive review of previous experiments and research projects, essentially draw two conclusions:

1. Most of the experiments have been basically flawed, both in their implementation and in the lack of adequate data.
2. Research has not been done on a sufficiently massive scale to develop solid public or correctional policy.

The authors, however, feel that the available documentation indicates that further exploration and experimentation into the real implications of diet and nutrition as they relate to development of criminal behavior are warranted.

The theory of a relationship between diet and criminal or delinquent behavior is supported by nonclinicians and the public. Parents are concerned, usually because of what they have read in popular magazines, that their children will act differently if they consume what is commonly referred to as “junk food.”
They readily provide anecdotal observations regarding the correlation between their children’s behavior and consumption of foods with high sugar content.

Food service administrators at correctional institutions also support this non-scientific observation. The food service director of a large maximum security facility in Minnesota claims that he has personally observed that a reduction in the disproportionate use of sugar in inmate diets has had a noticeable effect on inmate behavior. He says that following his hiring and prior to his intervention, inmates were using sugar by the cupful on even small amounts of breakfast cereal. This excessive use of sugar, he feels, provided an artificial “high” and was a constant stimulant to aggressive, loud, and inappropriate behavior by a significant proportion of the population. He decided to reduce the consumption of sugar to the use of small packets, and he feels that this has had a dramatic impact upon behavior at the facility.

Fishbein and Pease make a number of recommendations regarding implementation of projects as a result of the research. Basically, their suggestions revolve around providing nutritional education, making counseling available for offenders if dietary intervention is necessary, and incorporating information regarding sugar and its relationship to alcohol addiction into programs for alcoholics. They further suggest that when the courts order a psychological assessment, a nutritional assessment should also be done.

None of these recommendations are particularly significant, however, primarily because the authors feel that further research is needed before basic policies can be changed. Unfortunately, it is difficult to do research in a correctional institution, especially research relating to food intake or dietary regimen. Even if inmates volunteer to participate in a research project, most of them have access to a canteen, visitors, and other sources of food beyond the institution dining rooms or food service programs.

One area in which inmates do not have the usual access to outside interference is the segregation unit. It is possible that in this very controlled environment, an experimental diet could be developed that would still meet the ethical standards of the American Correctional Association. Deprivation of food or the use of diets as punishment is never acceptable, but good, adequate, and controlled food programming could be experimented with within a segregation unit. With the increasing acceptance of holistic medicine, a significant number of inmates could be willing to become involved in research projects that might provide significant and useful data. As the authors indicate, this research must involve not only a large enough sample but enough time for valid results to occur. There are enough specialized programs in institutions today—e.g., chemical dependency programs, sex offender programs—whose subjects are not in the general population that it would probably be possible to recruit an experimental group for whom the research could be linked to past behavior.

It is ironic that, the placebo effect mentioned by a number of researchers can only help the correctional administrator, since dietary experimentation itself generally causes behavior to become more positive. Thus, no correctional adminis-
Corrections should be hesitant to join in experiments with clinicians or nutritionists. Significant long-term changes in human values, attitudes, etc., may be very difficult to quantify, but the observed behavioral change may be enough to justify undertaking such a project.

One of the areas that certainly should be explored further is that of the impact of nutritional education and intervention, especially in juvenile facilities. Here again, the positive aspects far outweigh the negative insofar as long-term benefits can result from juveniles learning proper diet and nutrition, with the obvious attendant physical health advantages. While the emotional or behavioral changes are paramount in terms of the research with which this study is concerned, this kind of education can affect the long-term health of a young person. For the relatively short lengths of incarceration in the researchers’ studies, few would argue that any of these activities will have lifelong attitudinal or value changes. However, feeling better about one’s self, improved alertness, and a feeling of greater well-being can all contribute to a more positive self-perception, which certainly can then affect one’s behavior. Thus, whether one can prove a direct relationship between the ingestion of certain dyes, yeasts, or carbohydrates and behavioral change many be less important than understanding that individuals care enough to attempt to assist people in better utilization of food.

Correctional administrators should not hesitate to experiment or introduce projects within their systems, both to add to the body of knowledge regarding the effects of diet and to add another dimension to their efforts to provide alternative, positive lifestyles for inmates and offenders. Food service managers should be encouraged to read this paper and to implement its recommendations regarding nutritional education and awareness. Correctional administrators should be encouraged to make the information available to inmate populations and, with voluntary concurrence, to develop and implement larger research projects.

While there is no magic pill or quick fix in any area of correctional reformation or rehabilitation, the information provided in the Fishbein and Pease presentation can become part of an overall attempt to make the correctional experience more meaningful for staff and inmates alike.
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Diana Fishbein received her Ph.D. in Psychobiological Criminology at Florida State University. In 1981, she was awarded a fellowship from the National Institutes of Health to conduct research on the effects of nutritional components, toxins, and neurotransmitter precursors at the Applied Neuroscience Laboratory, University of Maryland School of Medicine. Dr. Fishbein has been an Assistant Professor of Criminal Justice at the University of Baltimore for six years, teaching research methods and the biological foundations of human behavior. In addition, she is a researcher at the Addiction Research Center, National Institute on Drug Abuse, where she directs studies on the psychobiological correlates of aggression, antisocial behavior, alcoholism, and substance abuse.

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Susan Pease completed her Ph.D. in Criminology at Florida State University in 1981 and is currently an Assistant Professor of Criminal Justice at North Carolina Central University in Durham, North Carolina. She has published studies on copycat crime, nutrition and behavior, and the reunification of inmates with their families. Additional areas of interest include the prediction of dangerousness, juvenile delinquency, violence and addictions, and policy development in the criminal justice system. Dr. Pease recently completed a project with the North Carolina Department of Corrections which involved prerelease inmate-family counseling in the city of Durham's War on Drug and Alcohol Abuse, and she also works with Durham's Rape Crisis Center.

ORVILLE B. PUNG

Orville B. Pung, a graduate of St. Cloud State University, has worked in the Minnesota Department of Corrections since 1958. He became Deputy Commissioner for the nonmetropolitan region in 1974, assuming administrative responsibility for five state correctional institutions. In 1976, he was appointed Assistant Executive Commissioner-Programs, in charge of all state correctional programs and correctional institutions. Three years later, he was appointed Deputy Commissioner-Institutional Services, responsible for all correctional facilities and institution support programs. In 1982, Mr. Pung was appointed Commissioner of Corrections. He provides administrative leadership for the 1900 employees of the Department of Corrections and also serves as a member of the Minnesota Sentencing Guidelines Commission and the State Criminal Justice Policy Task Force.
THE ROBERT J. KUTAK FOUNDATION

After the death in 1983 of Robert J. Kutak, one of the founding partners of the law firm of Kutak Rock & Campbell in Omaha, Nebraska, the partners of the firm and other friends and colleagues established the Robert J. Kutak Foundation to honor his memory and to continue support of the activities in which he had been personally and professionally involved. Among those interests was the field of criminal justice, with special emphasis on corrections. As a staff member of the U.S. Senate, Mr. Kutak helped draft the legislation that established the National Institute of Corrections and served as the first chairman of the NIC Advisory Board. He also served on the President’s Task Force on Prisoner Rehabilitation and on the American Delegation to the Fourth and Fifth United Nations Congresses on the Prevention of Crime and Treatment of Offenders.

THE NATIONAL INSTITUTE OF CORRECTIONS

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As established by the enabling legislation, the Institute’s policy is determined by an active 16-member nonpartisan Advisory Board appointed by the Attorney General of the United States. The Board is composed of six federal officials serving ex-officio, five correctional practitioners, and five individuals from the private sector who have demonstrated an active interest in corrections. Through public hearings, the Advisory Board regularly solicits the opinions of correctional practitioners and others involved in the criminal justice process prior to targeting the Institute’s fiscal year funds.
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