Sugar. Sugar, by elevating brain levels of serotonin, would be expected to have a calming effect on behavior (Glinsmann, Irausquin, and Park 1986). Nevertheless, the suggestion that sugar consumption induces hyperactive behavior is an old one (Seham and Seham 1929; Randolph 1947; Rinkel, Randolph, and Zeller 1951), and many parents have been reported to believe that sugar is a cause of uncontrolled behavior in their children (Crook 1975). Data to support this idea are limited. One retrospective review of glucose tolerance curves of hyperactive children found that 75 percent of 261 hyperkinetic children had abnormal glucose tolerance curves following a 5-hour glucose tolerance test (Langseth and Dowd 1978). Other studies have not found abnormal glucose tolerance curves. An association was suggested between destructive agressive behavior and sugar intake in hyperkinetic children, but not in other normal 4-to 17-yearold children, based on 7-day food diaries kept by the mothers and videotaped observations of behavior by trained observers blinded to the protocol (Prinz, Roberts, and Hantman 1980). Another study noted an inverse relationship between sugar consumption by children and standardized measures of intelligence and school achievement (Lester, Thatcher, and Monroe-Lord 1982). None of these studies has shown a cause-and-effect relationship between sugar and behavior.

One way to study cause and effect, in contrast to correlation and association, is with a double-blind challenge study in which subjects are exposed to a test substance or to a placebo and the results compared. To date, however, only short-term challenges have been carried out; their results show little effect. For a group of 21 boys whose families responded to an advertisement seeking children with adverse behavioral reactions to dietary sugar loads, the ingestion of sugar produced a slight but significant *decrease* in gross motor activity (as would be expected on neurochemical grounds), and no differences were found in attentional measures (Behar et al. 1984). One hyperkinetic 5-year-old male and his mother both became frustrated, hyperactive, and difficult to control after a double-blind challenge with sugar in lemonade. However, not 1 of 50 other hyperkinetic children tested in the same way responded to a sugar challenge (Gross 1984).

The effects of acute challenges of sucrose in comparison to the artificial sweetener aspartame in grade school children were compared using 37 behavioral (playroom observation and examiner ratings) and cognitive (learning and memory tasks) measures (Wolraich et al. 1985). No differences in any measure of performance were found. No systematic change in activity was observed in eight school-aged children exposed to three differ-

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ent doses of sucrose or to an aspartame placebo, and no clear effect was found on the Continuous Performance Task (Ferguson 1984).

To date, the only double-blind challenge that has reported an increase in activity after consumption of sugar was a pilot study of 13 psychiatrically ill children given regular orange juice or juice with sugar added in the form of sucrose or fructose (Connors and Blouin 1983). A subsequent study of 37 children who were psychiatric inpatients appears to suggest that during classroom observation *less* motor activity is seen with the sucrose and fructose challenges compared with aspartame (Connors 1984a). Overall, acute challenges with dietary sugar have failed to show that sugar causes hyperactivity, inattention, or impulsiveness (Rapoport 1986; Ferguson, Stoddard, and Simeon 1986).

Caffeine. Caffeine has been a concern of consumer groups because of possible deleterious effects upon children's behavior (Rumsey and Rapoport 1983). In a series of studies of the effects of caffeine in normal grade school children, habitual dietary caffeine intake was found to be a significant predictor of behavior (Elkins et al. 1981; Rapoport, Elkins, et al. 1981; Rapoport, Jensvold, et al. 1981; Rapoport 1983; Rapoport et al. 1984). Children who habitually consumed greater amounts of caffeine became more anxious and difficult to arouse when they were deprived of caffeine. When children were challenged with caffeine, the habitually high consumers felt little effect, but children who normally consumed little caffeine experienced adverse effects. Because no caffeine was consumed for 2 weeks preceding the challenge in this study, these effects are not likely to be due to caffeine tolerance, but instead suggest a possible physiologic basis for caffeine effects in children.

Considerable research has been conducted on the possible therapeutic effects of caffeine on behavior in children. In an uncontrolled cross-over trial with hyperactive children that compared the effects of caffeine, methylphenidate, and no treatment, caffeine significantly improved behavior (Schnackenberg 1973). In one study, caffeine has also been reported to improve the reaction times of hyperactive children (Reichard and Elder 1977), but not in more carefully controlled studies (Huestis, Arnold, and Smeltzer 1975; Garfinkel, Webster, and Sloman 1975; Firestone et al. 1978; Connors 1979). Comparisons of caffeine with stimulants such as amphetamine or methylphenidate have consistently found the prescription agents to be superior (Garfinkel, Webster, and Sloman 1975; Gross 1975; Huestis, Arnold, and Smeltzer 1975; Arnold et al. 1978; Firestone et al. 1978). Thus, a therapeutic role for caffeine in hyperactivity is uncertain.

Hypoglycemia

Hypoglycemia (low blood sugar) can occur either after a fast (fasting hypoglycemia) or several hours after the consumption of a meal (reactive hypoglycemia). Many individuals believe they suffer from reactive hypoglycemia because they experience symptoms of weakness, confusion, and irritability after eating sugars or other carbohydrate foods. However, it has not been possible to document an association between these symptoms and low blood sugar or insulin levels, and most authorities believe that symptoms are due to causes unrelated to blood sugar levels (Green 1981). Some authors believe a high percentage of patients referred with symptoms of hypoglycemia have emotional disorders rather than actual hypoglycemia (Johnson et al. 1980; Ford, Bray, and Swerdloff 1976).

True reactive hypoglycemia is diagnosed by an oral glucose tolerance test in which the administration of a large dose of glucose is followed by a collection of blood samples at hourly intervals for the next 5 hours. These tests are often unreliable (Lev-Ran and Anderson 1981; Charles et al. 1981). The generally accepted dividing line between normal and abnormal blood sugar of 50 mg/dl in whole blood may be too high, because lower levels have been observed in many individuals who experience no hypoglycemic symptoms (Hofeldt, Adler, and Herman 1975; Cahill and Soeldner 1974). Only when symptoms of hypoglycemia such as sweating, tremor, anxiety, and irritability occur at the same time as the documented low blood sugar level can a valid diagnosis of reactive hypoglycemia be made. Such a diagnosis may indicate the early presence of a disease such as diabetes.

Antisocial Behavior

Before the 1970's, interest in a possible relationship between diet and criminal behavior was generally limited to food faddists, but more recently, scientists have given serious attention to this matter (Hippchen 1978, 1981; Reed 1977; Rimland 1981; Rimland and Larson 1981; Schauss 1980; Schoenthaler 1983b). Advocates of a link between diet and criminality do not agree on a single mechanism by which diet might influence criminal behavior. Among the suggested explanations are reactive hypoglycemia, food allergies, and other undocumented adverse reactions to food, to vitamin and mineral deficiencies, and to toxicities (Feingold 1979; Green 1976; Hippchen 1978, 1981; Rimland 1981; Rimland and Larson 1981; Schauss 1980; Schmidt, Brajkovich, and Asch 1981; Schoenthaler 1983a, 1983b). Experts in criminology and psychiatry have reviewed these claims and have strongly concluded that no evidence for such a connection exists. The studies that purport to show such a link are seriously flawed, lack

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appropriate controls, and are subject to bias (Gray and Gray 1983; Gray 1986; Pease and Love 1986).

Implications for Public Health Policy

Dietary Guidance

General Public

Behavioral factors clearly influence food selection, dietary change, and chronic disease risk, but research in this area is still too preliminary to draw more than a few implications for the general public; exceptions generally apply to specific chronic disease conditions. Similarly, beyond the dietary guidance implications presented in the chapter on obesity, current evidence is insufficient to recommend specific dietary changes to prevent or treat the eating disorders—anorexia nervosa, bulimia, and pica. Sugar, certain food additives, and caffeine have been suggested as predisposing dietary factors to the development of behavioral disorders in children and adults, but evidence is weak and contradictory, and there is no reason to expect that a reduced intake of these substances would affect the incidence or severity of behavioral disorders. In addition, current evidence does not support any implications at present about the effects of amino acid precursors of neurotransmitters on behavior.

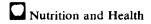
Special Populations

Studies in patients with eating disorders and other chronic disease conditions (reviewed in other chapters of this report) emphasize the importance of modification of diet-related behavior in these conditions. Such patients should receive advice from qualified health professionals on the application of dietary principles appropriate for their conditions. Although evidence linking dietary caffeine, refined sugars, and food additives to behavioral disorders is uncertain, their elimination from the diet will not impair nutritional status and can be recommended to patients on an individual trial basis.

Nutrition Programs and Services

Food Labels

Evidence related to the role of dietary factors in behavioral disorders holds no special implications for food labeling policies.



Food Services

Evidence related to the role of dietary factors in behavioral disorders currently holds no special implications for change in policies related to food programs beyond those suggested by the *Dietary Guidelines for Americans*.

Food Products

Evidence related to the role of dietary factors in behavioral disorders holds no special implications for change in policies related to food products at this time.

Special Populations

Patients with eating disorders should be provided with ready access to counseling and assistance in the development of diets that provide safe and adequate levels of energy and nutrients.

Research and Surveillance

Research and surveillance issues of special priority related to the role of behavior in the prevention of diet-related chronic disease and to the role of diet in behavioral disorders should include investigations into:

- Behavioral factors that influence food selection patterns and dietary change.
- The most effective behavioral methods to encourage appropriate dietary changes.
- Behavioral factors that increase the risk for diet-related chronic disease.
- Behavioral factors that increase the risk for obesity, anorexia nervosa, bulimia, and pica.
- The prevalence of these eating disorders among different groups.
- Behavioral techniques effective in treatment of these disorders.
- Effects of foods and nutrients on etiology and treatment of behavioral disorders.
- Behavioral interventions that increase the long-term effectiveness of health promotion and chronic disease treatment programs.



Literature Cited

Abraham, S.; Carroll, M.D.; Najjar, M.F.; and Fulwood, R. 1983. Obese and overweight adults in the United States. Vital Health Statistics 11:1-93.

Agras, W.S., and Kirkley, B.G. 1986. Bulimia: theories of etiology. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 367-78. New York: Basic.

American Psychiatric Association. 1987. Diagnostic and statistical manual of mental disorders, third edition, revised, pp. 65–69. Washington, DC: American Psychiatric Association.

Anderson, G.H., and Hrboticky, N. 1986. Approaches to assessing the dietary components of the diet-behavior connection. *Nutrition Reviews* 44(May, suppl.):42–51.

APA. See American Psychiatric Association.

Arnold, L.E.; Christopher, J.; Huestis, R.; and Smeltzer, D.J. 1978. Methylphenidate vs. dextroamphetamine vs. caffeine in minimal brain dysfunction. *Archives of General Psychiatry* 35:463–75.

Behar, D.; Rapoport, J.L.; Adams, A.J.; Berg, C.J.; and Cornblath, M. 1984. Sugar challenge testing with children considered behaviorally "sugar reactive." *Nutrition and Behavior* 1:277–88.

Boskind-White, M., and White, W.C. 1986. Bulimarexia: a historical-sociocultural perspective. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 353-66. New York: Basic.

Brownell, K.D. 1984. The psychology and physiology of obesity: implications for screening and treatments. Journal of the American Dietetic Association 84:406-14.

Brownell, K.D.; Heckerman, C.L.; Westlake, R.J.; Hayes, S.C.; and Monti, P.M. 1978. The effect of couples training and partner cooperativeness in the behavioral treatment of obesity. *Behavioral Research Therapy* 16:323.

Bruch, H. 1973. Eating disorders. New York: Basic.

_____. 1979. Eating disorders: obesity, anorexia nervosa and the person within. New York: Basic.

Cahill, G.R., Jr., and Soeldner, J.S. 1974. A non-editorial on nonhypoglycemia. New England Journal of Medicine 291:905-6.

Charles, M.A.; Hofeldt, F.; Shackelford, A.; Waldeck, N.; Dodson, L.E.; Bunker, D.; Coggins, J.T.; and Eichner, H. 1981. Comparison of oral glucose tolerance tests and mixed meals in patients with apparent postabsorptive hypoglycemia. Absence of hypoglycemia after meals. *Diabetes* 30:465-70.

Connors, C.K. 1979. The acute effects of caffeine on evoked response, vigilance and activity level of hyperkinetic children. *Journal of Abnormal Child Psychology* 7(2):145–51.

_____. 1984b. Nutritional therapy in children. In Nutrition and behavior, ed. J.R. Galler, pp. 159–92. New York: Plenum.

Connors, C.K., and Blouin, A.G. 1983. Nutritional effects on behavior of children. Journal of Psychiatric Research 17:193-201.

Crisp, A.M., and Stonehill, E. 1973. Aspects of the relationships between sleep and nutrition: a study of 375 psychiatric outpatients. *British Journal of Psychiatry* 122:379–94.

Crook, W.G. 1975. Food allergy-the great masquerader. Pediatric Clinics of North America 22:227.

Crosby, W.H. 1976. Pica. Journal of the American Medical Association 235:2765.

Curatelo, P.W., and Robertson, D. 1983. The health consequences of caffeine. Annals of Internal Medicine 98(pt. 1):641-53.

Cussler, M., and de Give, M.L. 1952. Twixt the cup and the lip: psychological and sociocultural factors affecting food habits. New York: Twayne.

Danford, D.E. 1982. Pica and nutrition. Annual Review of Nutrition 2:303-22.

Davidson, J.R.T.; Miller, R.D.; Turnbull, C.D.; and Sullivan, J. 1982. Atypical depression. Archives of General Psychiatry 39:527-34.

Desor, J.A.; Mallor, D.; and Greene, L.S. 1977. Preference for sweet in humans: infants, children, and adults. In *Taste and development: the genesis of sweet preference*, ed. J.M. Weifenbach. Fogarty International Center Proceedings no. 32. Bethesda, MD: National Institutes of Health.

Elkins, R.N.; Rapoport, J.L.; Zahn, T.P.; Buchsbaum, M.S.; Weingartner, H.; Kopin, I.J.; Langer, D.; and Johnson, C. 1981. Acute effects of caffeine in normal prepubertal boys. *American Journal of Psychiatry* 138(2):178-83.

Farb, P., and Armelagos, G. 1980. Consuming passions: the anthropology of eating. Boston: Houghton-Mifflin.

Feinberg, M., and Carroll, B.J. 1982. Separation of subtypes of depression using discriminant analysis. I. Separation of unipolar endogenous depression from non-endogenous depression. *British Journal of Psychiatry* 140:384–91.

Feingold, B.F. 1975. Why your child is hyperactive. New York: Random House.

_____. 1979. Dietary management of juvenile delinquency. International Journal of Offender Therapy and Comparative Criminology 23:73–84.

Ferguson, B. 1984. The effects of sugar and aspartame on children's cognition and behavior: a challenge study. Presented at conference Diet and Behavior: A Multidisciplinary Evaluation. Arlington, VA, November 27-29.

Ferguson, H.B.; Stoddard, C.; and Simeon, J.G. 1986. Double-blind challenge studies of behavioral and cognitive effects of sucrose-aspartame ingestion of normal children. *Nutrition Reviews* 44(May, suppl.):144-50.

Fernstrom, J.D., and Wurtman, R.J. 1972. Brain serotonin content: physiological regulation by plasma neutral amino acids. *Science* 178:414-16.

Firestone, P.; Davey, J.; Goodman, J.T.; and Peters, S. 1978. The effects of caffeine and methylphenidate on hyperactive children. *Journal of the American Academy of Psychiatry* 17(3):445-56.

Ford, C.V.; Bray, G.A.; and Swerdloff, R.S. 1976. A psychiatric study of patients referred with a diagnosis of hypoglycemia. *American Journal of Psychiatry* 133:290–94.

Garfinkel, B.D.; Webster, C.D.; and Sloman, L. 1975. Methylphenidate and caffeine in the treatment of children with minimal brain dysfunction. *American Journal of Psychiatry* 132(7):723–28.

Garfinkel, P.E., and Garner, D.M. 1982. Anorexia nervosa. New York: Brunner/Mazel.

Behavior

Garfinkel, P.E., and Kaplan, A.S. 1986. Anorexia nervosa: diagnostic conceptualizations. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 266-82. New York: Basic.

Garner, D. 1986. Cognitive therapy for anorexia nervosa. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 301-27. New York: Basic.

Garvey, M.F.; Mungas, D.; and Tollefson, G.D. 1984. Hypersomnia in major depressive disorders. Journal of Affective Disorders 6:283-86.

Glanz, K. 1986. Nutrition education for risk factor reduction and patient education: a review. *Preventive Medicine* 15:721–52.

Glinsmann, W.H.; Irausquin, H.; and Park, Y.K. 1986. Evaluation of health aspects of sugars contained in carbohydrate sweeteners: report of Sugars Task Force, 1986. *Journal of Nutrition* 116(11, suppl.):S5-216.

Gold, P.W.; Gwirtsman, H.; Avgerinos, P.C.; Nieman, L.K.; Gallucci, W.T.; Kaye, W.; Jimerson, D.; Ebert, M.; Rittmast, R.; and Loriaux, D. 1986. Abnormal hypothalamic-pituitary-adrenal function in anorexia nervosa: pathophysiologic mechanisms in underweight and weight-corrected patients. New England Journal of Medicine 314:1335–42.

Goode, J.G.; Curtis, K.; and Theophano, J. 1981. Group-shared food patterns as a unit of analysis. In Nutrition and behavior, ed. S.A. Miller, pp. 19-30. Philadelphia, PA: Franklin.

Gray, G.E. 1986. Diet, crime, and delinguency: a critique. Nutrition Reviews 44(May, suppl.):89-94.

Gray, G.E., and Gray, L.K. 1983. Diet and juvenile delinquency. Nutrition Today 18:14-22.

Green, R.G. 1976. Subclinical pellagra among penitentiary inmates. Journal of Orthomolecular Psychiatry 5:68-73.

Green, T.L. 1981. Reactive hypoglycemia: current diagnosis and treatment. Journal of American Osteopathic Association 80(12):827-30.

Grivetti, L.E. 1978. Culture, diet and nutrition: selected themes and topics. *BioScience* 28(3):171-77.

Gross, M.D. 1975. Caffeine in the treatment of children with minimal brain dysfunction or hyperkinetic syndrome. *Psychosomatics* 16(1):26-27.

____. 1984. Effect of sugar on hyperkinetic children. Pediatrics 74:876-78.

Halmi, K.A.; Flak, J.R.; and Schwartz, E. 1981. Binge-eating and vomiting: a survey of a college population. *Psychology and Medicine* 11:697–706.

Harris, M. 1985. Good to eat: riddles of food and culture. New York: Simon & Schuster.

Hartmann, E. 1983. Effects of L-tryptophan on sleepiness and on sleep. Journal of Psychiatric Research 17(2):107-13.

Hartmann, E.; Spinweber, C.L.; and Ware, C. 1976. L-tryptophan, L-leucine and placebo: effects on subjective alertness. *Sleep Research* 5:57.

Hawkins, R.C., II; Frenouw, W.J.; and Clement, P.F., eds. 1984. The binge-purge syndrome: diagnosis, treatment and research. New York: Springer.

Herzog, D.B., and Copeland, P.M. 1985. Eating disorders. New England Journal of Medicine 313(5):295-303.

Hippchen, L.J., ed. 1978. Ecologic-biochemical approaches to treatment of delinquents and criminals. New York: Van Nostrand Reinhold.

_____. 1981. Some possible biochemical aspects of criminal behavior. International Journal of Biosocial Research 2:37-42.

Hofeldt, F.D.; Adler, R.A.; and Herman, R.H. 1975. Postprandial hypoglycemia: fact or fiction. Journal of the American Medical Association 233:1309.

Hopkinson, G. 1981. A neurochemical theory of appetite and weight changes in depressive states. Acta Psychiatrica Scandinavica 64:217-25.

Huestis, R.D.; Arnold, L.E.; and Smeltzer, D.J. 1975. Caffeine versus methylphenidate and damphetamine in minimal brain dysfunction: a double-blind comparison. *American Journal of Psychiatry* 132(8):868-70.

Jimerson, D. 1984. Neurotransmitter hypotheses of depression. *Psychiatric Clinics of North America* 7(3):563-73.

Johnson, C.; Thompson, M.; and Schwartz, D. 1984. Anorexia nervosa and bulimia: an overview. In *Review in pediatric psychology*, ed. W.J. Burns and J.V. Labigne. New York: Grune & Stratton.

Johnson, D.D.; Dorr, K.E.; Swenson, W.M.; and Service, F.J. 1980. Reactive hypoglycemia. Journal of the American Medical Association 243:1151-55.

Kanarek, R.B., and Orthen-Gambill, N. 1986. Complex interactions affecting nutritionbehavior research. *Nutrition Reviews* 44(May, suppl.):172-75.

Kavale, K.A., and Forness, S.R. 1983. Hyperactivity and diet treatment. Journal of Learning Disabilities 16:324-30.

Kimmens, A.C., ed. 1975. Tales of the ginseng. New York: William Morrow.

Kraepelin, E. 1921. Clinical psychiatry: a text-book for students and physicians, trans. A.R. Deifendorf. New York: MacMillan.

Kraines, S.H. 1957. The physiologic basis of manic-depressive illness: a theory. American Journal of Psychiatry 114:206-11.

Langseth, L., and Dowd, J. 1978. Glucose tolerance and hyperkinesis. Food and Cosmetics Toxicology 16:129-33.

Leckman, J.F.; Caruso, K.A.; Prusoff, B.A.; Weissman, M.M.; Merikangas, K.R.; and Pauls, D.L. 1984. Appetite disturbance and excessive guilt in major depression: use of family study data to define depressive subtypes. Archives of General Psychology 41:839-44.

Lester, M.L.; Thatcher, R.W.; and Monroe-Lord, L. 1982. Refined carbohydrate intake, hair cadmium levels, and cognitive functioning in children. *Nutrition and Behavior* 1:3–13.

Lev-Ran, A., and Anderson, R.W. 1981. The diagnosis of postprandial hypoglycemia. Diabetes 30:996-99.

Lieberman, H.R.; Spring, B.; and Garfield, G.S. 1986. The behavioral effects of food constituents: Strategies used in studies of amino acids, protein, carbohydrate and caffeine. *Nutrition Reviews* 44(May, Suppl.) :61-70.

Lieberman, H.R.; Wurtman, J.J.; and Chew, B. 1986. Changes in mood after carbohydrate consumption among obese individuals. *American Journal of Clinical Nutrition* 44:772-78.

Lipton, M., and Golden, R. 1984. Nutritional therapies in the psychiatric therapies. In *The somatic therapies*, ed. T.B. Karasu. Washington, DC: American Psychiatric Association.

Ludman, E.K., and Newman, J.M. 1984. Yin and yang in the health-related food practices of three Chinese groups. Journal of Nutrition Education 16(1):3-6.



Lustick, M.J. 1985. Bulimia in adolescents: a review. Pediatrics 76(4, suppl.): 685-90.

Marlatt, G.A., and Gordon, J.R. 1985. Relapse prevention. New York: Guilford.

Mattes, J.A. 1983. The Feingold diet: a current reappraisal. Journal of Learning Disabilities 16:319-23.

Miller, S.A., ed. 1981. Nutrition and behavior. Philadelphia, PA: Franklin.

Mitchell, J.E. 1986. Anorexia nervosa: medical and physiological aspects. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 247-65. New York: Basic.

Morley, J.E., and Levine, A.S. 1983. The central control of appetite. Lancet i:398-401.

Murphy, D.L.; Campbell, I.; and Costa, J.L. 1978. Current status of the indoleamine hypothesis of affective disorders. In *Psychopharmacology: a generation of progress*, ed. M.A. Lipton, A. DiMascio, and K.E. Killam, pp. 1223-34. New York: Raven.

National Institutes of Health, Office of Medical Applications of Research. 1982. Consensus conference: defined diets and childhood hyperactivity. *Journal of the American Medical Association* 248:290–92.

NIH. See National Institutes of Health.

Olson, R.E., ed. 1986. Diet and behavior: a multidisciplinary evaluation. Nutrition Reviews 44(May, suppl.):1-254.

Paykel, E.S. 1977. Depression and appetite. Journal of Psychosomatic Research 21:401-7.

Paykel, E.S.; Mueller, P.S.; and de la Vergne, P.M. 1973. Amitriptyline: weight gain and carbohydrate craving: a side effect. British Journal of Psychiatry 123:501-7.

Pease, S.E., and Love, C.T. 1986. Optimal methods and issues in nutrition research in the correctional setting. *Nutrition Reviews* 44(May, suppl.):122-32.

Perri, M.G.; McAdoo, W.G.; Spevak, P.A.; and Newlin, D.B. 1984. Effects of a multicomponent maintenance program on long-term weight loss. *Journal of Counsulting Clinical Psychology* 52:480.

Prinz, R.J.; Roberts, W.A.; and Hantman, E. 1980. Dietary correlates of hyperactive behavior in children. Journal of Consulting Clinical Psychologists 48(6):760-69.

Pugliese, M.T.; Lifshitz, F.; Grad, G.; Fort, P.; and Markskatz, M. 1983. Fear of obesity: a cause of short stature and delayed puberty. New England Journal of Medicine 309:513-18.

Raebel, M.A., and Black, J. 1984. The caffeine controversy: what are the facts? Hospital Pharmacy 19(4):257-67.

Randolph, T.G. 1947. Allergy, a cause of fatigue, irritability and behavior problems in children. Journal of Pediatrics 31:560.

Rapoport, J.L. 1983. Effects of dietary substances in children. Journal of Psychiatric Research 17:187-91.

_____. 1986. Diet and hyperactivity. Nutrition Reviews 44(May, suppl.):158-62.

Rapoport, J.L.; Berg, C.J.; Ismond, D.R.; Zahn, T.P.; and Neims, A. 1984. Behavioral effects of caffeine in children. Archives of General Psychiatry 41:1073-79.

Rapoport, J.L.; Elkins, R.; Neims, A.; Zahn, T.; and Berg, C.J. 1981. Behavioral and autonomic effects of caffeine in normal boys. *Developmental Pharmacology and Therapeutics* 3:74–82.

Rapoport, J.L.; Jensvold, M.; Elkins, R.; Buchsbaum, M.S.; Weingartner, H.; Ludlow, C.; Zahn, T.P.; Berg, C.J.; and Neims, A.H. 1981. Behavioral and cognitive effects of caffeine in boys and adult males. *Journal of Nervous and Mental Disease* 169:726-32.

Reed, B. 1977. Diet related to killer diseases. In *Nutrition and mental health.* U.S. Senate Select Committee on Nutrition and Human Needs, sect. V, 95th Cong., sess. no. 78–2285. Washington, DC: US Government Printing Office.

Reichard, C.C., and Elder, S.T. 1977. The effects of caffeine on reaction time in hyperkinetic and normal children. American Journal of Psychiatry 134(2):144-48.

Rimland, B. 1981. Innovative approaches to criminality, delinquency and violence. International Journal of Biosocial Research 2:43-48.

_____. 1983. The Feingold diet: an assessment of the reviews by Mattes, by Kavale and Forness and others. *Journal of Learning Disabilities* 16:331-33.

Rimland, B., and Larson, G.E. 1981. Nutritional and ecologic approaches to the reduction of criminality, delinquency and violence. *Journal of Applied Nutrition* 33:116–137.

Rinkel, H.; Randolph, T.G.; and Zeller, M. 1951. Food allergy. Springfield, IL: Thomas.

Roberts, H.R., and Barone, J.J. 1983. Biological effects of caffeine: history and use. Food Technology 37:32-39.

Rolland-Cachera, M.F., and Bellisle, F. 1986. No correlation between adiposity and food intake: why are working children fatter? *American Journal of Clinical Nutrition* 44:779–87.

Rolls, B.J. 1985. Experimental analyses of the effects of variety in a meal on human feeding. *American Journal of Clinical Nutrition* 42:932–39.

Rosenthal, N.E.; Sack, D.A.; James, S.P.; Parry, B.L.; Mendelson, W.B.; Tamarkin, L.; and Wehr, T.A. 1984. Seasonal affective disorder and phototherapy. Presented at the New York Academy of Sciences, November.

Rosenthal, N.E.; Sack, D.A.; Gillin, J.C.; Lewy, A.J.; Goodwin, F.K.; Davenport, Y.; Mueller, P.S.; Newsome, D.A.; and Wehr, T.A. 1984. Seasonal affective disorder: a description of the syndrome and preliminary findings with light therapy. *Archives of General Psychiatry* 41:72–80.

Rozin, P. 1984. The acquisition of food habits and preferences. In *Behavioral health: a handbook of health enhancement and disease prevention*, ed. J.D. Matarazzo, S.M. Weiss, J.A. Herd, N.E. Miller, and S.M. Weiss, pp. 590-607. New York: Wiley.

Rumsey, J.M., and of diet in pediatric populations. In *Nutrition and the brain*, ed. R.J. Wurtman and J.J. Wurtman, pp. 101-61. New York: Raven.

Sachar, E.J., ed. 1976. Hormones, behavior and psychopathology. New York: Raven.

Schauss, A.G. 1980. Crime and delinquency. Berkeley, CA: Parker House.

Schmidt, K.; Brajkovich, W.R.; and Asch, M. 1981. Clinical ecology treatment approach for juvenile offenders. International Journal of Biosocial Research 2:15-20.

Schnackenberg, R.C. 1973. Caffeine as a substitute for schedule II stimulants in hyperactive children. American Journal of Psychiatry 30:796-98.

Schoenthaler, S.J. 1983a. Diet and crime: an empirical examination of the value of nutrition in the control and treatment of incarcerated juvenile offenders. *International Journal of Biosocial Research* 4:25–39.

_____. 1983b. Diet and delinquency: a multistate replication. International Journal of Biosocial Research 5:70-78.

Behavior

Schotte, D.E., and Stunkard, A.J. 1987. Bulimia vs. bulimic behaviors on a college campus. Journal of the American Medical Association 258:1213–15.

Seham, M., and Seham, G. 1929. The relation between malnutrition and nervousness. American Journal of Diseases of Children 37:1-38.

Seltzer, S.; Stoch, R.; Marcus, R.; and Jackson, E. 1982. Alteration of human pain thresholds by nutritional manipulation and L-tryptophan supplementation. *Pain* 13:385–93.

Slochower, J.A. 1983. Excessive eating. New York: Human Sciences.

Smith, S.L., and Sauder, C. 1969. Food craving, depression and premenstrual problems. *Psychosomatic Medicine* 31:281-87.

Sobotka, T.J. 1986. The regulatory perspective of diet-behavior relationships. *Nutrition Reviews* 43(5, suppl.):241-45.

Somers, A.R., and Weisfield, V.D. 1986. Individual behavior and health. In *Maxcy-Rosenau* public health and preventive medicine, 12th ed., ed. J.M. Last, pp. 983-97. New York: Appleton.

Sprague, R.L. 1981. Measurement and methodology of behavioral studies: the other half of the nutrition and behavior question. In *Nutrition and behavior*, ed. S.A. Miller, pp. 269–75. Philadelphia, PA: Franklin.

Story, M., and Brown, J.E. 1987. Do young children instinctively know what to eat? The studies of Clara David revisited. New England Journal of Medicine 316:103-6.

Striegel-Moore, R., and Rodin, J. 1986. The influence of psychological variables in obesity. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 99–121. New York: Basic.

Strober, M. 1986. Anorexia nervosa: history and psychological concepts. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 231-46. New York: Basic.

Stunkard, A.J., and Berthold, H.C. 1985. What is behavior therapy? A very short description of behavioral weight control. *American Journal of Clinical Nutrition* 41:821-23.

Stunkard, A.J., and Rush, J. 1974. Dieting and depression re-examined: a critical review of reports of untoward responses during weight reduction for obesity. *Annals of Internal Medicine* 81:526-33.

Stunkard, A.J.; Sorenson, T.I.A.; Hanis, C.; Tensdale, T.W.; Chakraborty, R.; Schull, W.J.; and Schulsinger, F. 1986. An adoption study of human obesity. *New England Journal of Medicine* 314(4):193-98.

Syme, S.L. 1986. Strategies for health promotion. Preventive Medicine 15:492-507.

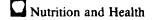
Vermeer, D.E., and Ferrell, R.E. 1985. Nigerian geophagical clay: a traditional antidiarrheal pharmaceutical. *Science* 227:634-36.

Wadden, T.A., and Stunkard, A.J. 1985. Social and psychological consequences of obesity. Annals of Internal Medicine 103(6, pt. 2):1062-67.

Weiss, B.; Williams, J.H.; Margen, S.; Abrams, B.; Caan, B.; Citron, L.J.; Cox, C.; McKibben, J.; Ogar, D.; and Schultz, S. 1980. Behavioral responses to artificial food colors. *Science* 207:1487–89.

Weissenburger, J.; Rush, J.; Giles, D.E.; and Stunkard, A.J. 1986. Weight change in depression. *Psychiatry Research* 17:275-83.

Williams, P., and King, M. 1987. The "epidemic" of anorexia nervosa: another medical myth? *Lancet* i:205-7.



Wilson, G.T. 1986. Cognitive-behavioral and pharmacological therapies for bulimia. In Handbook of eating disorders: physiology, psychology, and treatment of obesity, anorexia, and bulimia, ed. K.D. Brownell and J.P. Foreyt, pp. 450–75. New York: Basic.

Wilson, G.T., and O'Leary, K.D. 1980. Principles of Behavior Therapy. Englewood Cliffs, NJ: Prentice Hall.

Winston, D.H. 1987. Treatment of severe malnutrition in anorexia nervosa with enteral tube feedings. Nutritional Support Services 7(6):24-26.

Wolraich, M.; Milich, R.; Stumbo, P.; and Schultz, F. 1985. Effects of sucrose ingestion on the behavior of hyperactive boys. *Journal of Pediatrics* 106(4):675-82.

Wurtman, J.J., and Wurtman, R.J. 1983. Studies on the appetite for carbohydrates in rats and humans. Journal of Psychiatric Research 13:213-21.

Wurtman, R.J., and Wurtman J.J., eds. 1977-86. Nutrition and the brain, 7 vols. New York: Raven.

Wurtman, R.J.; Hefti, F.; and Melamed, E. 1981. Precursor control of neurotransmitter synthesis. *Pharmacologic Review* 32:315–35.

Wurtman, J.J.; Wurtman, R.J.; Growdon, J.H.; Henry, P.; Lipscomb, A.; and Zeisel, S. 1981. Carbohydrate craving in obese people: suppression by treatments affecting serotoninergic transmission. *International Journal of Eating Disorders* 1:2–11.

Young, S.N. 1986. The clinical psychopharmacology of tryptophan. In Nutrition and the brain, vol. 7, ed. R.J. Wurtman and J.J. Wurtman, pp. 49–88. New York: Raven.

Zifferblatt, S.M., and Wilbur, C.S. 1977. Dietary counseling: some realistic expectations and guidelines. Journal of the American Dietetic Association 70:591–95.

Zuckerman, D.M.; Colby, A.; Ware, N.C.; and Iazerson, American Journal of Public Health 76(9):1135-37.



Further it must be known that the children must be fed only milk and pap until they have grown the front teeth. Then one can give them somewhat stronger food—bread softened with milk or in a meat or pea broth. Bartholomaus Metlinger Kinderbuch (1473)

Introduction

The health and productivity of our society have roots in the quality of the reproductive experience and the subsequent nurturing strategies of parents and childhood caretakers. Much can be done to enhance the well-being of mothers and infants, and adequate nutrition during pregnancy and lactation is an important component of this process. Societies throughout history have recognized the unique needs of pregnant and lactating women and have made special provisions for their health care and nutritional needs.

This chapter reviews key issues related to the nutrition of pregnant women and lactating mothers and their infants, children, and adolescents and to the improvement of their health and survival. It also discusses aspects of chronic disease prevention that especially concern these younger age groups.

Historical Perspective

Although the unique health status of pregnant women has long been recognized, dietary recommendations for this group have reflected incomplete knowledge about human reproduction and growth and nutrition. Likewise, while a healthy, vigorous infant has always been the desired outcome of pregnancy, only recently has nutrition been recognized as influencing that outcome.

Infants were weighed at birth during Talmudic times, but it was not until the 17th century that the French obstetrician Mauriceau recognized the health

significance of adequate birth weight (IOM 1985). Mauriceau's ideas did not gain currency, perhaps because his estimate of 15 lb as a normal birth weight was obviously incorrect. It was not until the 19th century that birth weights, on the basis of their variability, were used as indicators of nutritional status and physical growth (IOM 1985).

Beliefs about the effects that various foods might have on the mother or child influenced dietary recommendations for pregnant women in the 19th century. For example, to prevent a child from having an unpleasant disposition, pregnant women were cautioned against eating salty, acidic, or sour foods. Obstetrical problems also influenced dietary recommendations. During the time of the Industrial Revolution, rickets commonly impaired normal pelvic bone formation. As a result, women who had had rickets in childhood faced major risks in childbirth, and both maternal and infant mortality were high. To combat this problem, Prochownick, a German physician, prescribed a fluid-restricted, low-carbohydrate, high-protein diet for the last few weeks of pregnancy. Because the infants of women who followed this diet were smaller and more easily delivered, it became a standard recommendation for women throughout pregnancy, one that persisted even when rickets, the original reason for it, was no longer a problem (Worthington-Roberts, Vermeersch, and Williams 1985).

In the early 20th century, scientists began to study the relationship of low birth weight (LBW) to shortened gestation and to infant mortality. After World War I, attempts to relate food shortages to reduced birth weights were inconclusive. In 1930, the Finnish pediatrician Yllpo suggested that infants were at high risk below a birth weight of 2,500 g (about 5.5 lb). The World Health Organization subsequently adopted this standard, despite recognition that birth weight below this level is not synonymous with prematurity (IOM 1985).

Although research on diet and pregnancy did not begin until the 1930's, obstetric practitioners voiced strong opinions about what pregnant women should—and should not—eat. Obstetrical authorities commonly warned the pregnant woman against accepting either advice to ". . . eat largely because she is eating for two" or to ". . . eat sparingly because if she does not her baby will be too large" (Danforth 1933), holding the view that the weight of the unborn child could not be influenced by diet except within very narrow limits. At the same time, pediatricians argued that the mother's health and nutritional status did indeed influence the size of the newborn child. Studies conducted by the Research Laboratory of the Children's Fund of Michigan demonstrated that the mother's nutritional state before and at the time of conception and the adequacy of her diet during pregnancy

influence the well-being of the infant. There was an upswing of research in maternal nutrition in the 1940's, influenced by the impact of wartime food shortages on maternal health (Egan 1987). Some of the famine studies during World War II showed a marked fetal impact from extreme food deprivations of the mother, but correlational studies were inconsistent in relating nutritional intake to birth outcomes (IOM 1985). There was sufficient interest and concern in the United States about the relationship of maternal nutrition to problems of prematurity, congenital malformations, and infant mortality to stimulate the National Academy of Sciences to appoint a Committee on Maternal Nutrition and Child Feeding in 1946. Its charge was to "implement improved health of the nation through better coordination of scientific advances in the field of obstetrics and pediatrics." The Committee's 1950 report, *Maternal Nutrition as It Relates to Child Health—An Interpretive Review*, served as the chief reference for maternal and child health nutrition programs for many years (Egan 1987).

Uncertainties about the role of nutrition in the management of pregnancy led to discussion between the service community and the research community in 1966 that resulted in formation of a Committee on Maternal Nutrition of the National Academy of Sciences. In 1970, the Committee issued a hallmark research report, *Maternal Nutrition and the Course of Pregnan*cy, stimulating the following:

- Significant changes in clinical practices related to the routine restriction of weight gain and sodium intake during pregnancy.
- Reaffirmation of the priority needs of pregnant women and infants in supplemental food programs.
- Development, adoption, and dissemination of policy statements and guidelines related to nutrition and maternal health by professional organizations.
- Increased emphasis on training of nutrition personnel as well as on education in nutrition for physicians.
- Increased funds for research on maternal and perinatal nutrition, and renewed interest of the National Institutes of Health in this area (Egan 1987).

Maternal and infant nutrition was a focus of the White House Conference on Food, Nutrition, and Health in 1969, and its Conference Panel on Pregnant and Nursing Women and Young Infants identified adequate diet as one factor "known to be necessary for favorable outcomes of pregnancy" (White House Conference 1969). The panel recognized the increased and special needs of pregnant and nursing women and infants and con-

cluded that optimal nutrition for women throughout life is the best way to promote infant health. In 1970, the White House Conference on Children called for expansion and improvement of existing food programs as well as promotion of nutrition education in the schools. In 1971, the White House Conference on Youth demanded Federal food assistance for all young Americans in need, and a National Nutrition Education Conference later that year also focused attention on the nutritional needs of adolescents (Egan 1972).

Federal Maternal and Child Nutrition Programs

Despite scientific uncertainties about the relation among nutrition and human reproduction, growth, and development, Federal programs have addressed the nutritional needs of mothers and children since early in this century (Egan 1977). The Children's Bureau issued dietary advice to parents and teachers and conducted nutritional surveys of low-income children (Egan 1977). School feeding programs began in the early 1900's when free, compulsory, and universal education was instituted. These programs were also supported by philanthropic organizations, local school districts, and private donors. Several States authorized schools to serve meals at cost, and a few States even served meals at reduced or no cost to needy children (CBO 1980). Increasing Federal involvement in these programs resulted from the burden on local and State entities during the 1930's. An amendment to the Agricultural Act of 1933 established a fund to purchase surplus agricultural commodities for donation to needy families and to child nutrition programs, including school lunch programs. The Social Security Act of 1935 authorized grants to the States for health services for mothers and children and established the basis for a national program of nutrition services (including assessment, counseling, referral, and followup) for this population (Select Panel 1981).

When the supplies of surplus commodities declined during World War II, Federal involvement in child feeding programs shifted to general income assistance. To avoid the uncertainties of congressional appropriation of cash subsidies, the National School Lunch Act, authorizing permanent grants-in-aid to States, was enacted in 1946. To receive cash and commodity assistance, the States had to operate school lunch programs on a nonprofit basis, provide free or reduced-price lunches for needy children, and serve lunches that would meet specified Federal standards (CBO 1980).

The Child Nutrition Act of 1966 enlarged the scope of Federal efforts by establishing numerous programs to expand food assistance year round to children of all ages and to provide assistance to pregnant women and



infants. For the first time, high-risk families enrolled in comprehensive maternity and infant health projects and children and youth projects received direct nutrition services as an integral part of their health care (Egan 1977). These programs and policies led to authorization of the Special Supplemental Food Program for Women, Infants, and Children (WIC) by Congress in 1972. Modified versions of many of the Federal food assistance programs that developed during the 1960's and 1970's remain in place today.

U.S. Department of Agriculture Programs. Current food assistance programs of the U.S. Department of Agriculture (USDA) include:

- School Breakfast and Lunch Programs: help schools to serve nourishing, low-cost meals to children and to provide free or reduced-price meals to children from low-income families.
- Summer Food Service Program: helps communities serve meals to needy children when school is not in session. Operates in areas where the concentration of low-income families is high.
- Child Care Food Program: subsidizes meals and snacks for children up to age 18 in nonresidential child care programs.
- Special Supplemental Food Program for Women, Infants, and Children: provides selected foods rich in specific nutrients, nutrition education, and health care referrals to pregnant, lactating, and postpartum women and children up to age 5. Participants must be certified as nutritionally at risk and of low income.
- Commodity Supplemental Food Program: distributes certain agricultural commodities at no cost to supplement the diets of low-income infants, children under 6 years, and pregnant and postpartum women who are vulnerable to malnutrition.
- Special Milk Program: makes it possible for school children to purchase milk at reduced price or receive it free; subsidizes milk served to children in schools, day care institutions, and summer camps.
- Food Stamps: provide eligible individuals and households with coupons free of charge that can be exchanged for food at authorized food stores. The redemption value of the food stamps depends on the household's size and financial circumstances. Food stamps are meant to supplement what a family spends on food.

Although there are intrinsic limitations in methods, evaluations of the school feeding and WIC programs have generally shown that they improve the health and nutritional status of the target populations (see Systems Development Corporation 1983; Rush 1985).

Nutrition Programs of the U.S. Department of Health and Human Services (DHHS). Nutrition services are an important component of many of the health and social service programs administered by DHHS. These programs include:

- Title V Maternal and Child Health Program: provides for nutrition assessment, dietary counseling, nutrition education, and referral to food assistance programs to women of childbearing age, infants, preschool and school-aged children, adolescents, and children with special health care needs served through a wide range of health care programs. In addition, it supports training in nutrition for nutrition personnel and other health professionals who lead the development of nutrition services. It supports nutrition projects of regional and national significance to advance and improve nutrition services for mothers and children.
- Medicaid-Early Periodic Screening, Diagnosis, and Treatment: requires assessment of the nutritional status of eligible children and provision of appropriate referral and treatment services.
- Head Start: provides preschool children from low-income families with nutritious meals and snacks; provides nutrition education for the children and their families; and provides training and technical assistance in nutrition for Head Start staff.
- Primary Care-Community Health Centers, Migrant Health Programs: provide nutrition services as an integral component of these comprehensive health care programs.

Nutrition Education Programs. Both USDA and DHHS sponsor nutrition education programs targeted to parents and children. Major programs are the USDA Extension Service's Expanded Food and Nutrition Education Program and Food and Nutrition Program, the Nutrition Education and Training Program of the Food and Nutrition Service, and the educational component of the WIC program. DHHS funds maternal and child nutrition education programs through the programs administered by its various agencies: the health care programs of the Health Resources and Services, Administration and the Indian Health Service of the Public Health Service, and the Head Start Program of the Administration on Children, Youth, and Families of the Office of Human Development Services (Select Panel 1981).

National Goals and Recommendations

Within the past decade, Federal reports and conferences have established goals and produced recommendations designed to improve the nutrition and health of mothers and children. Some of these goals and reports are listed below.

Healthy People: The Surgeon General's Report on Health Promotion and Disease Prevention. This report defines broad goals for the improvement of health and reduction of mortality rates among infants, children, and adolescents. Nutrition is included as a key health promotion strategy to achieve these goals (DHEW 1979).

1990 National Health Objectives. At least 35 of the 226 specific objectives to achieve the goals defined by Healthy People are related to nutrition and the health of pregnant women, infants, children, or adolescents (DHHS 1980). Table 15-1 presents a selection of these objectives.

Table 15-1 Selected National Objectives to be Achieved by the Year 1990 Related to Maternal and Child Nutrition*

Pregnancy and Infant Health

- Reduce the national infant mortality rate (deaths for all babies up to 1 year of age) to no more than 9 deaths per 1,000 live births
- Ensure that no county nor racial or ethnic group of the population has an infant mortality rate in excess of 12 deaths per 1,000 live births
- Reduce the maternal mortality rate to no more than 5 per 100,000 live births for any county or for any ethnic group
- Reduce to no more than 5 percent of all live births the percentage of low birth weight babies (2,500 g and under)
- Ensure that 85 percent of women of childbearing age can state the special nutritional needs of pregnancy and can understand the hazards of alcohol use during pregnancy and lactation
- Ensure that virtually all infants receive adequate primary health care, including nutrition services, when needed

Fluoridation and Dental Health^b

- Eliminate cariogenic foods from school vending machines and school breakfast or lunch programs
- Ensure that at least 95 percent of school children and their parents are able to identify the principal risk factors related to dental diseases and be aware of the importance of fluoridation in controlling these diseases

Misuse of Alcohol^b

- Reduce the proportion of adolescents 12 to 17 years old who report acute drinking-related problems during the past year to below 17 percent
- Increase the proportion of women of childbearing age who are aware of the risks associated with drinking during pregnancy to greater than 90 percent
- Increase the proportion of high school seniors who recognize the risk of alcohol intoxication to 80 percent or more

Table 15-1 (continued)

Nutrition

- Increase the proportion of women who breastfeed their babies to 75 percent at hospital discharge and to 35 percent at 6 months of age
- Increase the proportion of school cafeteria managers who are actively promoting the USDA/DHHS *Dietary Guidelines* to more than 50 percent
- Ensure that all States include nutrition education as part of required school health education at elementary and secondary levels

Physical Fitness and Exercise^b

• Increase the proportion of children and adolescents ages 10 to 17 participating regularly in appropriate physical activities to more than 90 percent

^a Some objectives apply to more than one category.

^b These issues are discussed further in the chapters on dental diseases, alcohol, and obesity, respectively.

Source: U.S. Department of Health and Human Services 1980.

Better Health for Our Children: A National Strategy. A chapter of the report of the Select Panel for the Promotion of Child Health emphasizes the importance of nutrition in maternal and child health and gives specific recommendations for improved information and education, health care services, and research in nutrition for this population (Select Panel 1981).

Surgeon General's Workshop on Maternal and Infant Health. This report recommends that nutrition services become integrated into all maternal and child health programs, that maternal and child health professionals receive improved training in nutrition, and that further research be conducted on infant feeding patterns, the development of dietary patterns and tastes, and the role of social and cultural factors in human nutrition (DHHS 1981).

Surgeon General's Workshop on Breastfeeding and Human Lactation. This report recommends specific strategies to promote breastfeeding through improved public and professional education, health care systems and support services, and research (DHHS 1984).

Significance for Public Health

Although the precise contribution of nutrition to maternal and infant health cannot yet be distinguished from genetic, environmental, or behavioral factors that affect risk, an inadequate diet during pregnancy increases the probability of a LBW infant, who, in turn, has an increased risk of mor-



bidity and mortality. Well-nourished mothers who gain appropriate amounts of weight during pregnancy generally give birth to heavier, healthier babies. Poorly nourished mothers who eat well and gain adequate weight during pregnancy can greatly improve their chances of giving birth to healthy infants. Diet is also critical in the control of diabetes, hypertension, and gastrointestinal disorders when such conditions are present during pregnancy.

Infant Mortality

In the United States, the infant mortality rate (the number of deaths of infants under 1 year of age per 1,000 live births) has declined rather steadily throughout this century, largely due to improvements in control of infectious diseases, health care, and nutrition. In 1900, the rate was about 100 deaths per 1,000 births; by 1978 it had declined to 13.8 and by 1985 to 10.6 (NCHS 1987c). Recent information suggests that the rate of decrease is slower than it has been in the past, as indicated by a provisional rate of 10.4 for 1986 (Kleinman 1987). Infant mortality rates in several industrialized countries are considerably lower—for example, 6.5 and 6.6, respectively, for Finland and Japan in 1983 (NCHS 1986). However, rates among countries are not strictly comparable due to differences in definitions.

Although marked improvements in infant survival have occurred among all racial and ethnic groups, the infant mortality rate among black Americans remains nearly twice as high as that among white Americans. From 1978 to 1985, the infant mortality rate among whites fell from 12.0 to 9.3 per 1,000, and that among Native Americans fell from 13.7 to 9.1 per 1,000. The rate among black Americans, however, was 23.1 in 1978; it declined, but in 1985 it was still 18.2—nearly twice the rate of whites (NCHS 1987b).

Low Birth Weight

In the United States, the most important factor contributing to the infant mortality rate is a low birth weight—less than 2,500 g, or 5.5 lb. Low birth weight occurs as a result of birth prior to 37 weeks' gestation, intrauterine growth retardation, or both. Infant deaths and illnesses increase sharply as birth weight declines within the normal weight range and even more sharply below 2,500 g. They are highest among infants of very low birth weight (VLBW), below 1,500 g. LBW infants are at increased risk for developmental handicaps, birth defects, respiratory and other infectious diseases, behavior problems, and complications of medical interventions. These conditions greatly increase the emotional and financial burden to the infant's family and to the Nation (IOM 1985).

Although overall rates of infant mortality have decreased greatly, the prevalence of LBW has declined more slowly. In 1971, babies with weights below 2,500 g accounted for 7.6 percent of all live births; in 1985, they accounted for 6.8 percent. Infants of moderately low birth weight (1,500 to 2,500 g) accounted for over 5.5 percent of all live births in 1985 and those of VLBW for over 1.2 percent. Together, the 6.8 percent of infants born at weights less than 2,500 g are responsible for 67 percent of all infant deaths during the first month of life and approximately 60 percent of all infant deaths (NCHS 1987a).

Much of the higher infant mortality among black Americans is explained by the high proportion of LBW infants born to this group. LBW infants comprised 12.4 percent of all black births in 1985. For Americans of Puerto Rican descent, the percentage of LBW infants was 9.1. It ranged from 6.2 to 6.9 percent for Americans of Japanese, Filipino, other Asian and Pacific Island, and American Indian ancestry, and from 5.6 to 5.8 percent for Mexican, Cuban, and other white Americans. The lowest percentage of LBW (5.3 percent) occurred among Chinese Americans (Secretary's Task Force 1985). Of special concern in these figures is the continuing racial disparity in birth weight. Between 1973 and 1983, rates of moderately low birth weight decreased more among whites than among blacks. Overall rates of VLBW decreased, but they actually increased among blacks (Kleinman and Kessel 1987).

Risk Factors for Low Birth Weight

Medical, social, behavioral, and dietary factors before and during pregnancy contribute to the risk for LBW. Medical risk factors include a previous reproductive history that includes many pregnancies, anemia, hypertensive disorders of pregnancy, inadequate weight gain, or delivery of a LBW infant; low prepregnancy weight; chronic illnesses such as diabetes or hypertension; and poor weight gain during pregnancy. Social, demographic, and behavioral risk factors have been identified as low socioeconomic status, low educational level, minority race, single marital status, adolescence, inadequate prenatal care, and use of drugs, alcohol, or cigarettes.

Dietary risk factors include an inadequate intake of calories or essential nutrients such as protein, vitamins, and minerals. Evidence indicates that the more of these risk factors present, the greater the risk to mother and child (IOM 1985). Because these risk factors interact and affect one another, it is difficult to determine the role of nutrition separate from these other risk factors.

Estimates of Cost

The personal cost of the death or severe illness of a LBW infant is incalculable. Quantitative estimates of the economic costs of care of LBW infants have been published. In 1987, the average hospital cost was estimated at \$12,000 to \$39,000. Costs increase, however, as birth weight falls; the average cost for a VLBW surviving infant is \$31,000 to \$71,000. For infants born at under 750 g, the average hospital stay was 98 days, and the costs ranged as high as \$150,000 (OTA 1987). In 1984, the rehospitalization rate during the first year of life was 38.3 percent for VLBW infants and 19 percent for moderately LBW infants. Such infants remained rehospitalized an average of 16.2 and 12.5 days, respectively. In addition, nearly 20 percent of LBW infants who survive will have long-term morbidity during the first year that requires medical care (IOM 1985). Even taking into consideration the increased costs of providing adequate prenatal care, the savings to the Nation from prevention of even a small proportion of LBW infants would be considerable.

Scientific Background

Pregnancy and Lactation

Normal pregnancy is accompanied by anatomical and physiologic changes that are necessary to promote fetal growth and development and prepare the mother for labor, birth, and lactation. Many of these changes are apparent in the early weeks of pregnancy.

Physiologic. During both pregnancy and lactation, hormonal changes affect retention, utilization, and excretion of nutrients (Hytten and Leitch 1971). These changes lead to physiologic adjustments that result in expansion of blood volume and accumulation of fluid. They include an increase in cardiac output, heart rate, and basal metabolic rate. Preparation of the mammary glands for lactation begins during pregnancy, when the duct system enlarges and the alveolar cells proliferate. Lactation is initiated and maintained by hormonal changes that occur in response to the infant's sucking stimulus. Milk proteins, lipids, and lactose are synthesized by the alveolar cells. Stored fats are secreted into the milk, as are a variety of nutrients and other compounds. Although recent evidence suggests that lactation is associated with physiologic changes that conserve energy and reduce the need for increased energy intake (Butte, Garza, Stuff, et al. 1984), additional studies are needed to evaluate this matter (Lawrence 1985).

Biochemical. During pregnancy, decreases in plasma concentrations of albumin, most minerals, and most water-soluble vitamins occur as a result of the dilution effect of the expanded plasma volume. Because expansion in red cell mass is not as great as the expansion in plasma volume, the hematocrit, which is the percentage of red blood cells in a sample of centrifuged blood, typically drops during pregnancy. Other nutrients, however, increase in plasma concentration, perhaps as a result of improved intestinal absorption.

Physical. During pregnancy, body weight, lean body tissue, and fat increase. Increases in tissue fluid levels are most significant in the third trimester, but women vary substantially in the timing and degree of fluid accumulation. After childbirth, blood volume and extracellular fluids return to prepregnant levels. The uterus also returns to normal size, but breast size remains enlarged throughout lactation. Loss of the body fat stores accumulated during pregnancy occurs gradually and is usually complete by the time the nursing infant is about 6 months old (Lawrence 1985).

Normal Growth and Development

In humans, intrauterine growth and development require about 40 weeks of gestation. From the third month until term, fetal weight increases nearly 500-fold, from about 6 g (0.2 oz) to 3,000 to 3,500 g (6.5 to 7.6 lb) at birth.

Infants. Immediately after birth, weight is lost, but birth weight is usually regained by the 10th day. After this time, weight increases at a rapid but decelerating rate. Most infants double their birth weight by the age of 4 months and triple it within 1 year. Length increases by 50 percent during the first year. These changes are accompanied by changes in body composition. Fat accumulates rapidly; by 6 months, it makes up about 25 percent of the total body weight. During the second 6 months, the relative increase in lean body mass is much greater than the increase in fat (Formon et al. 1982).

Children and Adolescents. The very rapid rate of growth in infancy is followed by slower growth during the preschool and early school-age years. Weight gain approximates 2.5 kg/year until 9 to 10 years of age. Length increases by an average of 11 to 12 cm in the second year, about 7 cm during each of the next three. Children become leaner between 6 months and 6 years, after which a gradual increase in fat thickness occurs in both males and females until puberty; females have a relatively greater body fat content than males at all stages of development (Fomon et al.