Annotated Bibliography on Maternal Nutrition (1970)

Pages
205

Size
7 x 10

ISBN
0309365147

Committee on Maternal Nutrition; Food and Nutrition Board; National Research Council; National Academy of Sciences

annotated bibliography on
MATERNAL NUTRITION

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National Research Council
National Academy of Sciences

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Health Services and Mental Health Administration
Maternal and Child Health Service
1970

For sale by the Superintendent of Documents, U.S. Government Printing Office
Washington, D.C. 20402 - Price $1.50

The work of the Committee on Maternal Nutrition, of the Food and Nutrition Board, has been supported by Contract WA-CB-67-03 awarded by the Maternal and Child Health Service.

PREFACE

This bibliography is directed to physicians, nutritionists, and other persons concerned with applying the research findings reported in the literature on maternal nutrition. It is published as a supplement to Maternal Nutrition and the Course of Pregnancy (NAS-NRC Publication 1761).

The compilers scanned Index Medicus for the years 1958 to 1968, inclusive, selected relevant titles, read the papers, and prepared the annotations. Some papers that appeared before 1958 are included; these papers have historical significance or contain information essential to an understanding of the more recent literature. Only materials published in English are included. Materials that appeared in popular magazines have not been included.

There is some overlapping of subject-matter areas under the heading "References on Specific Topics," and readers may find it necessary to look under more than one topic heading for references in a given area.

GENERAL REFERENCES

American College of Obstetrics and Gynecology. 1965. Manual of standards in obstetric-gynecological practice. 2nd ed. Chicago. 120 p.

Guidelines for facilities and care for obstetric and gynecological services, including a discussion of the nutrition component, are presented.

Ashley-Montagu, M. F. 1957. The reproductive development of the female, with especial reference to the period of adolescent sterility. 2nd ed. Julian, New York. 234 p.

The book includes references to nutritional factors that relate to ovulation, fecundity, and fertility.

Assali, N. S., ed. 1968. Biology of gestation. Academic Press, New York. 2 vol.

The first volume examines the biochemical and physiological changes occurring during pregnancy, and Volume 2 focuses on the physiological and biochemical aspects of the fetus and newborn.

Assali, N. S., ed. 1968. The fetus and neonate. Academic Press, New York and London. 405 p.

An authoritative coverage of the mechanisms influencing fetal adjustment to intrauterine life and changes occurring at birth.

Barnes, A. C., ed. 1968. Intra-uterine development. Lea & Febiger, Philadelphia. 530 p.

Twenty-six authors review present knowledge about embryonic and placental development from conception to parturition. The volume undertakes to collate and interpret the most important information about intrauterine development and chromosomal abnormalities. The chapter on placental transfer provides a concise summary of current knowledge about mechanisms of transfer of nutrients from maternal blood to fetus.

Beaton, G. H., and E. W. McHenry, eds. 1964. Nutrition: A comprehensive treatise. Academic Press, New York. 3 vol.

Presents fundamental knowledge of human nutrition along with problems inherent in the interpretation and application of this knowledge to humans. Volume III discusses the application of basic knowledge to particular areas in nutrition, including pregnancy.

Brewer, T. H. 1966. Metabolic toxemia of late pregnancy. Charles C Thomas, Springfield, Ill. 127 p.

The author presents his belief that toxemia of late pregnancy is a metabolic disease caused by malnutrition.

Casida, L. E. 1963. The level of fertility in the female as influenced by feed level and energy intake. In Proc. Sixth Int. Congr. Nutr. Livingstone Ltd., London.

Ovulation rate was positively correlated with feed intake; fertilization was little affected and embryo survival (sheep and swine) often highest when feed level was limited.

Cheek, D. B. 1968. Human growth, body composition, cell growth, energy, and intelligence. Lea & Febiger, Philadelphia. 781 p.

Presents comprehensive information from multiple disciplines relating to the physiology, biochemistry, and psychology of growth in normal and abnormal children.

Chipman, S. S., A. M. Lilienfeld, B. G. Greenberg, and J. F. Donnelly. 1966. Research methodology and needs in perinatal studies. Proc. Conf., Chapel Hill, N. C. Charles C Thomas, Springfield, Ill. 309 p.

Record of a conference held in 1963 that includes nine papers and the reports of four groups. Emphasis of the conference was epidemiological. To the nutritionist, the most interesting parts may be a short review of maternal nutrition in relation to prematurity and a report of a group discussion on nutrition and outcome of pregnancy.

Committee on Adolescence, Group for the Advancement of Psychiatry. 1968. Normal adolescence: Its dynamics and impact. Charles Scribner's Sons, New York. 127 p.

A review of the physiology of adolescence, the psychological processes that normally occur during this age, and the reactions of adolescents to their environment, to their parents, and to other adults.

Davidson, S., and R. Passmore. 1967. Human nutrition and dietetics. 3rd. ed. Williams & Wilkins Co.,Baltimore. 864 p.

Written primarily for physicians, this book includes chapters on the physiology of nutrition and a general description of commonly eaten foods. The remainder deals with diseases commonly caused by defective diets and therapeutic modifications of normal diets.

Davis, J. 1960. Survey of research in gestation and the developmental sciences. Waverly Press, Baltimore. 203 p.

A critical review of gestation and development from germ cells and fertilization to special problems of gestation.

Dawkins, M., and B. MacGregor, eds. 1965. Gestational age, size and maturity. Lavenham Press Ltd., Lavenham, Suffolk. 115 p.

This volume, based on contributions to a meeting, surveys the problems surrounding premature birth of an infant and its subsequent development.

Dieckmann, W. J. 1941. The toxemias of pregnancy. C. V. Mosby Co., St. Louis. 521 p.

No relationship was found between toxemia and low-protein diet.

Dieckmann, W. J. 1952. The toxemias of pregnancy. 2nd ed., The C. V. Mosby Company, St. Louis. 710 p.

A comprehensive discussion of knowledge to 1950 of the toxemias, including sections on history, classification, incidence, pathology, the etiology of eclampsia, clinical picture, treatment, and prognosis.

Engle, E. T., ed. 1953. Pregnancy wastage. Conf. Proc. Charles C Thomas, Springfield, Ill. 254 p.

Discusses postconceptual reproductive failures and their prevention.

Hagbard, L. 1961. Pregnancy and diabetes mellitus. Charles C Thomas, Springfield, Ill. 101 p.

Discusses observations made on a series of pregnant diabetic women, including observations pertaining to maternal complications, mortality, and long-term effects of pregnancy on the disease and similar considerations in the infant.

Hansen, H. A. 1964. On the diagnosis of folic acid deficiency. Orstadius Boktryckeri AB, Göteborg, Sweden. 175 p.

A methodological and clinical study in which special attention is given to the incidence and etiology of megaloblastic anemias in pregnancy and the puerperium.

Harrell, R. F., E. Woodyeard, and A. I. Gates. 1955. The effect of mothers' diets on the intelligence of offspring. Bureau of publications, Teachers College, Columbia University, New York. 71 p.

In Norfolk, Virginia, 518 3-year-old children were tested with the Terman-Merrill Revision of the Binet test to determine the possible effect of vitamin supplements during pregnancy and lactation on subsequent intelligence of offspring. The children with the highest average IQ's had mothers who had received thiamin and B complex supplementation during pregnancy and lactation. The children with the lowest IQ's had mothers who had not received supplementation during pregnancy. The differences in IQ between children whose mothers had received supplements and those who had not was significant at the 1 percent level of confidence. When the same study design was used on a different population, no difference in intelligence was noted.

Heald, F. P., ed. 1966. Adolescent gynecology. Williams & Wilkins Co., Baltimore, Md. 163 p.

A collection of papers that discuss the adolescent. A particular interest are chapters entitled "The Psychological Impact of Pregnancy on the Adolescent Girl" and "The Reproductive Endocrinology of Adolescence."

Herzog, E., and R. Bernstein. 1964. Health services for unmarried mothers. U.S. Department of Health, Education, and Welfare; Children's Bureau. Publ. 425. U.S. Government Printing Office, Washington, D. C.

A review of research and demonstrations, based on reports published before 1962, relating to health services for unmarried mothers. Chapter headings include "Complications of Pregnancy in Births Out-of-Wedlock," "Medical Services: Availability, Use and Quality," "Efforts to Extend and Coordinate Medical and Social Services," and "Recommendations."

Horsky, J., Z. K. Stembera, I. R. Merkatz, and K. J. Dennis, eds. 1968. International Symposium Intrauterine Dangers to the Foetus, Prague. Excerpta Medica Foundation, Amsterdam, N. Y. 615 p.

Contains the papers presented at the symposium, 107 of which are in English and divided into three sections: the pathogenesis of acute and chronic fetal distress, the detection of fetal distress, and the therapy of fetal distress. A short section on the dysmature fetus is included.

Hytten, F. E. and I. Leitch. 1964. The physiology of human pregnancy. Blackwell Publications, Oxford, England. 463 p.

Book consists of 14 chapters and three appendixes. The first 12 chapters are devoted to measurements of pregnant women and the changes that take place in physiological adaptations and metabolism to delivery. The last two chapters deal with specific requirements arising from these changes.

Jackson, C. M. 1925. The effects of inanition and malnutrition upon growth and structure. P. Blakiston's Son and Co., Philadelphia. 616 p.

In animal experiments, undernutrition halted the development of ovaries, testes, and secondary sex organs, and caused cessation of estrus in adult females. Prolonged undernutrition caused irreverisble growth changes and gonadal development. The period of puberty was found most critical.

Kaltreider, F. D. 1963. Effects of height and weight on pregnancy and the newborn. Charles C Thomas, Springfield, Ill. 237 p.

Maternal height and weight are used as a diagnostic tool for predicting outcome of human pregnancy.

Keys, A., J. Brozek, A. Henschel, O. Mickelsen, and H. L. Taylor. 1950. The biology of human starvation. Univ. Minneapolis Press, Minneapolis. 2 vol.

A chapter on "Growth and Development" in Volume II is concerned with the effect of maternal nutrition on the course of human pregnancy, on certain complications, and on the size of offspring. The period covered is principally the 1920's to 1940's.

Lewis, T. L. T. 1964. Progress in clinical obstetrics and gynecology. 2nd ed. Little, Brown and Company, Boston. 753 p.

The chapter on "Toxemia of Pregnancy" contains a review of current theories of etiology and an extensive bibliography on the subject.

McCall, M. L. 1959. Present concepts in toxemia of pregnancy. (Disease-a-month series.) Year Book Publications, Chicago. 29 p.

A monograph reviewing clinical and basic data on toxemia and presentation of treatment.

Millen, J. W. 1962. The nutritional basis of reproduction. Charles C Thomas, Springfield, Ill. 125 p.

Nutrition and its effect on reproduction are discussed. Included are chapters on sterility, nutrition in pregnancy, the disorders of pregnancy, and malformations.

Mitchell, H. S., H. J. Rynbergen, L. Anderson, and M. V. Dibble. 1968. Nutrition in health and disease. 15th ed. J. B. Lippincott Company, Philadelphia. 685 p.

The principles of nutrition as they apply to needs of normal persons and to the specific dietary needs of persons suffering from pathological conditions are comprehensively reviewed. The chapter on nutrition in pregnancy discusses increased demands and stresses food selection.

Montagu, A. M. 1962. Prenatal influences. Charles C Thomas, Springfield, Ill. 614 p.

The effects of the prenatal experience on the development of the fetus and on postnatal physical and psychological development are discussed. A chapter on nutrition is included.

Pike, R. L., and M. L. Brown. 1967. Nutrition: An integrated approach. John Wiley & Sons, New York. 522 p.

The purpose of the book is to integrate scientific disciplines related to the study of nutrition. To this end, the book is divided into three parts: the first presents the historical development of nutritional science and basic information on the nutrients; the second presents basic biochemical cytology, focusing on the nutrients and their physiological and biochemical action; and the third presents concepts underlying applied nutrition.

Rhodes, P. 1960. Fluid balance in obstetrics: A critical review. Lloyd-Luke Ltd., London. 196 p.

A synopsis of fluid balance problems in obstetrics for practicing physicians.

Richardson, S. A., and A. F. Guttmacher, eds. 1967. Childbearing--its social and psychological aspects. Williams & Wilkins Co., Baltimore. 334 p.

An extensive review of the social and psychological factors that influence the course of pregnancy, delivery, and outcome. The contributors present research findings, delineate research issues, and describe and critically appraise the design and methodology of studies in their fields.

Robinson, C. H. 1967. Normal and therapeutic nutrition. 13th ed. The Macmillan Company, New York. 891 p.

A textbook written for students in nutrition and for reference use by dietitians, nutritionists, and physicians. The chapter on nutrition during pregnancy and lactation stresses desirable food practices.

Roulet, F. C., ed. 1960. Eclampsia and pre-eclampsia in pregnancy. Proc. Seventh Conf. Int. Soc. Geogr. Pathol. Karger, Basel-New York. 729 p.

Marked differences in the incidence and characteristics of toxemia are reported in a series of papers from many countries. Differences in definitions make comparisons difficult, but it appears that blood pressures in young women vary throughout the world. In Europe and North America, they are higher than in most other parts of the world, and the incidence of toxemia of pregnancy is also higher. The incidence of eclampsia and of maternal mortality due to toxemias is closely related to the quality of medical care received. The relationships with nutrition and other environmental variables are not clear.

Scrimshaw, N. S., and J. E. Gordon, ed. 1968. Malnutrition, learning, and behavior. M.I.T. Press, Cambridge, Mass. 566 p.

Proceedings of an international conference that evaluated the effects of nutritional deficiencies in young children on later mental function. Part 1 discusses philosophy; part 2, malnutrition and retarded growth in man; part 3, biological factors in central-nervous-system development; and part 4, the effects of malnutrition on learning and behavior in experimental animals.

Semmens, J. P., and W. M. Lamers. 1968. Teen-age pregnancy: Including management of emotional and constitutional problems. Charles C Thomas, Springfield, Ill. 118 p.

The authors discuss the physician's role in all aspects of management of the pregnant teenager.

Tanner, J. M. 1962. Growth at adolescence. 2nd ed. Blackwell Publications, Oxford. 325 p.

The effects of hereditary and environmental factors on growth and maturation from birth to maturity are discussed.

ten Berge, B. S. ed. 1965. Pregnancy: chemistry and management. Charles C Thomas, Springfield, Ill. 328 p.

A series of papers, written for obstetricians, that stress subjects of topical interest, such as toxemia and its management. Of special interest to nutritionists is a chapter entitled "Nutritional Status of the Mother and Its Influence on Her Milk." This chapter discusses 20 malnourished Indian women and the effect of low nutrient intake on composition of milk, particularly the whey: curd ratio.

Theobald, G. W. 1956. The pregnancy toxemias. Paul B. Hoeber, Inc., New York. 488 p.

In an examination of hypotheses for the etiology of toxemias, the main conclusions were that "eclampsia" is a generic term for a syndrome that can be provoked by more than one mechanism and that eclampsia represents a failure to adapt adequately to the "pregnancy-lactation syndrome," for which there are three main causes: (1) inherent defects in the expectant mother, (2) mechanical factors, and (3) nutritional factors.

Wohl, M. C., and R. B. Goodhart, eds. 1968. Modern nutrition in health and disease. 4th ed. Lea & Febiger, Philadelphia, Pa. 1240 p.

This volume is designed to serve as a textbook on nutrition and as a reference book for practitioners in the fields of medicine, nutrition, and public health. A chapter on nutrition in pregnancy includes theoretical discussion and practical suggestions on nutritional management in pregnancy.

World Health Organization. 1961. Public health aspects of low birth weight. WHO Tech. Rep. Ser., No. 210. Geneva. 16 p.

Report discusses prematurity and low birth weight; preventive aspects, including diet in pregnancy; and care of low-birth-weight infants.

World Health Organization. 1963. Social aspects in the teaching of obstetrics and gynecology. WHO Tech. Rep. Ser., No. 266. Geneva. 22 p.

Report points to the need for increased attention to the social aspects of obstetrics and gynecology in the education of undergraduate and postgraduate students.

World Health Organization. 1964. Biology of human reproduction. WHO Tech. Rep. Ser., No. 280. Geneva. 30 p.

Report discusses the comparative aspects and neuroendocrine aspects of reproduction, the biology of gonad and gametes, gestation, the biochemistry of steroids, and immunological and pharmacological aspects of reproduction.

World Health Organization. 1965. Health problems of adolescence. WHO Tech. Rep. Ser., No. 308. Geneva. 28 p.

Nutrition in adolescence is discussed. Accelerated growth and development following puberty causes an increased and variable demand for nutrients. Pregnancy in adolescence adds to nutritional requirements. Areas where further research is needed are delineated.

World Health Organization. 1965. Nutrition in pregnancy and lactation. WHO Tech. Rep. Ser., No. 302. Geneva. 54 p.

Report discusses the metabolic and physiological aspects of pregnancy and lactation that relate to changed nutritional needs.

Wynn, R. M., ed. 1967. Fetal homeostasis. New York Academy of Sciences. Port City Press Inc., Baltimore. 2 vol.

Proceedings of conferences focused on the factors that maintain the fetus or its antecedent germ cells in a steady state and the mechanisms that make reproduction possible in mammals. Volume I discusses implantation and fertilization, and Volume II is concerned primarily with the placenta.

Adolescence

Anderson, U. M., R. Jenss, W. E. Mosher, and V. Richter. 1966. The medical, social, and educational implications of the increase in out-of-wedlock births. Amer. J. Pub. Health 56:1866-1873.

In Buffalo, New York, tnere was more than a threefold increase in the overall percentage of illegitimacy in the 14-year study period. These unwed mothers received less prenatal care and had a higher incidence of puerperal complications and prematurity than the married controls. Infant mortality was also higher in the study group.

Anderson, W. J. R., D. Baird, and A. M. Thomson. 1958. Epidemiology of stillbirths and infant deaths due to congenital malformation. Lancet 1:1304-1306.

This study suggests that the young primigravidae living in poor social conditions are at high risk in terms of fetal death due to malformation of the central nervous system.

Aznar, R., and A. E. Bennett. 1961. Pregnancy in the adolescent girl. Amer. J. Obstet. Gynecol. 81:934-940.

A review of literature and a study of 1,137 pregnant adolescents, 16 years of age and under. The study group had an increased incidence of severe toxemia, prolonged labor, and premature labor when compared with older controls. No increased incidence of abortion was noted, although fetal abnormalities and postpartum complications were slightly increased.

Baker, H., O. Frank, S. Feingold, G. Christakis, and H. Ziffer. 1967. Vitamins, total cholesterol, and triglycerides in 642 New York City school children. Amer. J. Clin. Nutr. 20:850-857.

A study of blood thiamin, biotin, riboflavin, pantothenic acid, niacin, vitamin B_6, vitamin B_{12}, folic acid, vitamin A, ascorbic acid, total cholesterol, triglycerides and

grams of high-quality protein consumed by Puerto Rican, Chinese, Negro, and Caucasian boys and girls 10 to 13 years of age whose parents were predominantly factory, service, or trade workers.

Battaglia, F. C., T. M. Frazier, and A. E. Hellegers. 1963. Obstetric and pediatric complications of juvenile pregnancy. Pediatrics 39:902-910.

In a population of 636 pregnant girls 14 years of age or less, the majority of whom were nonwhite, there was a higher incidence of toxemia, contracted pelvis, prematurity, immaturity, and perinatal mortality than in primigravidas 15 to 19 years old. This increased incidence of complications appeared to be related to age rather than race or parity.

Bochner, K. 1962. Pregnancies in juveniles. Amer. J. Obstet. Gynecol. 83:269-271.

The pregnancies of 272 girls between the ages of 12 and 16 were compared with pregnancies of 658 women aged 20 to 29. The groups were not matched with respect to parity and race. The performance of the girls was similar in most respects to the older women's. Nevertheless, there were noted increased frequency of toxemia, contracted pelvis, and prolonged labor in the cases of the girls.

Briggs, R. M., R. R. Herren, and W. B. Thompson. 1962. Pregnancy in the young adolescent. Amer. J. Obstet. Gynecol. 84:436-441.

The obstetric records of a Florence Crittenton Home were reviewed for 201 primigravidas aged 16 or less. Older primigravidas averaging 25 years of age served as controls. The great majority of both groups were Caucasian. Antepartum and postpartum complications were low for both groups, which may be the result of the excellent antepartum care received.

Burke, B. S., R. B. Reed, A. S. van den Berg, and H. C. Stuart. 1962. A longitudinal study of the calcium intake of children from one to eighteen years of age. Amer. J. Clin. Nutr. 10:79-88.

Cross-sectional distributions of the calcium intakes of 61 girls are presented for the age period one to eighteen years. Longitudinal individual patterns are also reported.

Campbell, A. A., and J. D. Cowhig. 1967. The incidence of illegitimacy in the United States. Welfare Administration, U.S. Dept. of HEW, Welfare in Review, May 1967, 1-6.

Although data are incomplete and biases exist, an upward movement in the incidence of illegitimacy has occurred since 1940. Data are also presented on changes in rate of illegitimacy for adolescent girls and older women.

Clamon, A. D., and H. M. Bell. 1964. Pregnancy in the very young teen-ager. Amer. J. Obstet. Gynecol. 90:350-354.

The course of pregnancy and labor of 224 girls under 16 years of age was reviewed. Although maternal performance was good, a significant number of the girls developed acute toxemia of pregnancy. Discusses the criteria for diagnosis of hypertension in young adolescents and the special needs of this group for skilled care.

Clough, W. S. 1958. The young primipara. Obstet. Gynecol. 12:373-381.

Review of the obstetrical performance of 175 primigravidae 16 years of age or less compared with that of a group aged 21 to 25 years.

Daly, M. J. 1966. Physical and psychological development of the adolescent female. Clin. Obstet. Gynecol. 9:711-721.

One of eight papers from a symposium on adolescent gynecology. Development is discussed under the headings Early Puberty, Menarche, Gender Role, Puberty, Sexual Development, Intellectual and Moral Development, and Inhibiting Factors.

Dibble, M. V., M. Brin, E. McMullen, A. Peel, and N. Chen. 1965. Some preliminary biochemical findings in jr. high school children in Syracuse and Onondaga County, New York. Amer. J. Clin. Nutr. 17:218-239.

Food and nutrient intake was calculated and physical examination and biochemical levels were determined on about 400 school children, age 12 to 15 years, from varied socioeconomic backgrounds. A relationship was found between investigated parameters and socioeconomic status.

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Dodge, E. F., and W. E. Brown. 1950. Effect of age upon obstetric complications in the primigravida. S. Med. J. 43:1060-1066.

A review of the obstetrical records of 3,198 indigent patients. Toxemia, eclampsia, prolonged labor, postpartum hemorrhage, fetal mortality, and maternal morbidity and mortality show higher incidence in the very young and very old primigravidae. Contracted pelvis, precipitate labor, unfavorable fetal presentation, forceps delivery, and premature labor did not correlate with age. The lowest incidence of complications was found in mothers 17 to 21 years old.

Dreizen, S., C. N. Spirakis, and R. E. Stone. 1967. A comparison of skeletal growth and maturation in undernourished and well-nourished girls before and after menarche. J. Pediat. 70:256-263.

The skeletal maturational progress of 30 undernourished and 30 well-nourished girls was followed from early childhood to early adulthood by evaluation of radiographs of the hand taken every 6 months and height measurements taken every 3 months. Chronic undernutrition slowed the rate of both skeletal growth and skeletal maturation, delayed menarche, and prolonged the growth period. Ultimate adult height was not significantly different for the two groups.

Dwyer, J. T., J. J. Feldman, and J. J. Mayer. 1967. Adolescent dieters: Who are they? Amer. J. Clin. Nutr. 20:1045-1056.

Sixty percent of the 446 white predominately middle-class or upper-middle-class female high school seniors interviewed had at some time been on a reducing diet. A larger percentage of the girls were dieting or had dieted than were classified as obese or nearing obesity.

Edmonds, E. M. 1968. A study of contraceptive practices in a selected group of urban, Negro mothers in Baltimore. Amer. J. Pub. Health. 58:263-273.

Interviews were conducted on 198 Negro mothers to determine knowledge of, use of, and attitudes toward contraception. Thirty percent of the women had knowledge of contraception before the age of 16 years, but the majority reached biological maturity several years before they had any knowledge of contraception.

Fox, R. I., J. J. Goldman, and W. A. Brumfield. 1968. Determining the target population for prenatal and postnatal care. Pub. Health Rep. 83:249-257.

Lack of recognition of the importance of care, denial of pregnancy, no one to care for other children, and lack of transportation were cited as reasons by the 27 percent of 555 mothers who did not receive prenatal care or only received care late in pregnancy. Socioeconomic as well as health factors must be considered when determining health care needs.

Fried, R. I., and E. E. Smith. 1962. Postmenarcheal growth patterns. J. Pediat. 61(4):562-565.

Girls (600) from well-to-do homes were examined to determine linear growth after menarche. In about 20 percent, the growth after menarche was more than 4 inches. In only 16 of the girls, the increased postmenarcheal growth was attributed to earlier growth impairment.

Fry, P. C. 1959. Diets of post-adolescent women. J. Amer. Diet. Ass. 35:687.

Seven-day food records were kept by 144 students 18 to 25 years of age. The percentage overweight and underweight and intakes of specific nutrients were calculated.

Garn, S. M., and J. A. Haskell. 1959. Fat changes during adolescence. Science. 129:1615-1616.

In a study of 259 children in Ohio, lower thoracic fat, as measured on serial chest plates, increased in girls 6.5 to 14.5 years of age. No evidence was found of a period of marked loss of "baby" fat in adolescence or of "waves" of fattening around the time of puberty.

Gottschalk, L. A., J. L. Titchener, H. N. Piker, and S. S. Stewart. 1964. Psychosocial factors associated with pregnancy in adolescent girls: A preliminary report. J. Nerv. Ment. Dis. 138:524-534.

A report of a survey of 131 pregnant girls, 16 years of age or younger, in which three types of interviewing techniques were used.

Hacker, E. M., J. W. W. Epperson, H. D. Priddle, and H. W. Longyear. 1952. An analysis of the adolescent obstetric patient. Amer. J. Obstet. Gynecol. 64:644-649.

An analysis of the course and outcome of 490 pregnant adolescent patients. Findings include (1) increased incidence of toxemia, prolonged labor, and prenatal diagnosis of cephalopelvic disproportion, and (2) a lower incidence of stillbirths and fetal anomalies in the adolescent girls than in older women.

Hammar, S. L. 1966. The role of the nutritionist in an adolescent clinic. Children 13:217-220.

A general discussion of the roles a nutritionist is able to assume in an adolescent clinic.

Hampton, M. C., R. L. Huenemann, L. R. Shapiro, and B. W. Mitchell. 1967. Caloric and nutrient intakes of teen-agers. J. Amer. Diet. Ass. 50:385.

A greater percentage of the Caucasian, Negro, and Oriental teen-agers studied chose diets furnishing less than two-thirds of the Recommended Dietary Allowances for calcium and iron. Those in the lowest socioeconomic group tended to have lower intakes of nutrients than others.

Harris, J. W. 1922. Pregnancy and labor in young primiparae. Bull. Johns Hopkins Hosp. 33:12-16.

In a study of 500 primigravidae, 16 years of age or less, the author concluded that these young girls usually had a shorter labor and infants of normal birth weight.

Haskins, A. L. 1964. The reproductive behavior of the adolescent female. Bull. School Med., Univ. Md. 49:3-9.

A review of 31,495 adolescent pregnancies that includes 21 white and 366 nonwhite mothers under 15 years of age, and 1,731 white and 4,951 nonwhite mothers 15 to 19 years of age. Findings included an increased incidence of uterine inertia, contracted pelvis, prolonged labor, pre-eclampsia, and prematurity, and a decreased incidence of placenta previa, premature separation, Cesarean section, and fetal anomalies in the adolescent group.

Hassan, A. M., and F. H. Falls. 1964. The young primipara--A clinical study. Amer. J. Obstet. Gynecol. 88:256-269.

A study of 159 young primigravidae 12 to 15 years of age and 78 control primigravidae 22 years of age. When the results for the group 12 to 15 years of age were tabulated by year of age and compared with the controls and the whole hospital, the young girls were found to have increased incidence of toxemia. The critical age for these complications was 14 years.

Heald, F. P., M. Daugela, and P. Brunschuyler. 1963. Physiology of adolescence. New Engl. J. Med. 268:192-198, 243-252, 299-307, 361-366.

In a series of four papers, the authors review the literature on the physiology of the adolescent. In the section on nutrition, they stress the need for nutrition studies that are based on physiological age rather than on chronological age.

Heald, F. P., and R. J. Hollander. 1965. The relationship between obesity in adolescence and early growth. J. Pediat. 67:35-38.

A restrospective study of heights and weights of 158 obese and 94 non-obese adolescent girls showed that birth weights of obese and non-obese were similar; average weight gain from birth to 1 year was significantly greater for the obese; there was no difference in height at end of the first year; and age at menarche was 12.3 years for both groups.

Hofmeister, F. J., and G. F. Burgess. 1955. Labor in young and old primiparas: Physiologic parturition or obstetric complication? Obstet. Gynecol. 6:162-168.

In a study of 332 primigravidae (136, 12 to 15 years; 196, 35 to 45 years), it was found that neither the very young nor the elderly primigravidae constitute a particular obstetric problem provided there is early and adequate prenatal care. It was felt that there is greater danger in the neglected, casually treated multigravidae.

Huenemann, R. L., M. C. Hampton, L. R. Shapiro, and A. R. Behnke. 1966. Adolescent food practices associated with obesity. Fed. Proc. 25:4-10.

The mean intakes of calcium, iron, and calories in girls from a sample of 950 Oriental, Negro, and Caucasian teenagers in the ninth to 12th grades were lower than recommended allowances. Girls from the lower socioeconomic groups had lower intakes of calories, protein, and ascorbic acid.

Huenemann, R. L., L. R. Shapiro, M. C. Hampton, and B. W. Mitchell. 1968. Food and eating practices of teen-agers. J. Amer. Diet. Ass. 53:17-24.

Four weekly food diaries were kept for 2 years by 122 junior and senior high school students, and they showed marked irregularity in eating practices in one-third of the group. Differences in eating practices were associated with ethnic and socioeconomic differences.

Hulka, J. F., and J. T. Schaaf. 1964. Obstetrics in adolescents: A controlled study of deliveries by mothers 15 years of age and under. Obstet. Gynecol. 23:678-685.

The case histories of 139 primigravidae under age 16 were compared with an adjusted stratified random sample of 119 primigravidae 19 to 21 years of age. The younger group presented no greater difficulties in pregnancy, labor, delivery, or the postpartum period than did the older primigravidae, but their babies tended to be delivered earlier, to weigh less, and to have poorer chances of survival.

Hutcheson, R. H. 1968. Iron deficiency anemia in Tennessee among rural poor children. Publ. Health Rep. 83:939-943.

Microhematocrit determinations were done routinely on children attending immunization clinics in rural areas. One-fourth of the nonwhite year-old infants were found to have hematocrits of 29 percent or less, and 10 percent of all children under age 6 were judged to be anemic.

Israel, S. L., and J. Deutschberger. 1964. Relation of the mother's age to obstetric performance. Obstet. Gynecol. 24:411-417.

Data are presented on 22,201 unselected pregnant subjects. For each subject, pregnancy terminated in a single delivery. Age of the mother did affect course and outcome of pregnancy. Women at high risk are the very young and the old. The best age for childbearing was found to be between 18 and 25 years.

Israel, S. L., and T. B. Woutersz. 1963. Teenage obstetrics: A cooperative study. Amer. J. Obstet. Gynecol. 85:659-668.

By analysis of data-processing forms from 10 institutions, 3,995 teen-age (under 20 years) pregnancies were compared with 40,709 total deliveries. The teen-age group had a higher incidence of pre-eclampsia and of iron deficiency anemia. There was essentially no difference in fetal, neonatal, and perinatal mortality.

Lee, M. M. C., K. S. F. Chang, and M. M. C. Chan. 1963. Sexual maturation of Chinese girls in Hong Kong. Pediatrics 32:389-398.

In 3,278 southern Chinese schoolgirls, 6 to 20 years of age, the mean age at menarche was 12.9 years. The highest mean age at menarche was in the group having lowest socio-economic status.

Leverton, R. M. 1968. The paradox of teen-age nutrition. J. Amer. Diet. Ass. 53:13-16.

Application of the Recommended Dietary Allowances to adolescents and methods of nutrition education for teenagers are discussed.

Lewis, B. V., and P. J. Nash. 1967. Pregnancy in patients under 16 years. Brit. Med. J. 2:733-734.

Study of pregnancy and labor in 103 patients under 16 years of age showed perinatal mortality of 1 percent. Easy spontaneous vaginal delivery was the rule. Pre-eclampsia was a hazard in this group, the incidence being 20 percent.

Lewis, R. C., A. M. Duval, and A. Iliff. 1943. Effect of adolescence on basal metabolism of normal children. Amer. J. Dis. Child. 66:396-403.

Determinations of basal metabolism were made in a study of 70 boys and 57 girls.

Marchetti, A. A., and J. S. Menaker. 1950. Pregnancy and the adolescent. Amer. J. Obstet. Gynecol. 59:1013-1020.

Review of 634 pregnancies in nonwhite girls 12 to 16 years of age showed a high incidence of pre-eclampsia and eclampsia, but short labor, few Cesarean sections, few puerperal complications, and lower than average infant and maternal mortality.

McCammon, R. W. 1965. Are boys and girls maturing physically at earlier ages? Amer. J. Pub. Health. 55:103-106.

Evidence is examined that suggests that there is a secular trend toward earlier maturation and increased physical size. The evidence is not conclusive, and further research is needed.

Merrow, S. B. 1967. Triceps skin-fold thickness of Vermont adolescents. Amer. J. Clin. Nutr. 20:978-985.

Obesity data (percentage by age) were obtained in a study of 261 girls 12 to 15 years of age. The data refer to height, weight, and skin-fold thicknesses at the triceps sites.

Metsala, P. 1966. Observations on adolescent parturients. Ann. Chir. et Gynaecol. Fenn. 55:214-218.

Three hundred and thirty-seven parturients, 19 years of age or younger, were found to have a lower age at menarche than average and a higher incidence of anemia and toxemic disturbances than the older control population. Prematurity and low birth weight were significantly higher in girls 16 to 17 years of age than in girls 18 to 19 years of age.

Morrison, J. H. 1953. The adolescent primigravida. Obstet. Gynecol. 2:297.

A review of the literature and a report of experience with 577 predominately nonwhite patients 15 years of age or younger is presented. The experimental group showed a higher incidence of toxemia and a lower incidence of Cesarean section, syphilis, tuberculosis, multiple pregnancy, abruptio, and prenatal mortality than the older controls.

Morse, E. H., S. B. Merrow, and R. F. Clarke. 1965. Some biochemical findings in Burlington (Vt.) junior high school children. Amer. J. Clin. Nutr. 12:211-217.

Hemoglobin, hematocrit, plasma ascorbic acid, vitamin A, carotene, cholesterol, thiamin, and riboflavin values were reported for 401 boys and girls in the seventh, eighth, and ninth grades.

Mussio, T. J. 1962. Primigravidas under age 14. Amer. J. Obstet, Gynecol. 84:442-444.

Fifty cases of pregnancy in girls under 14 years of age were reviewed. A high incidence of prolonged labor, anemia, and toxemia was found. Fetopelvic disproportion was not significant in this study.

Poliakoff, S. R. 1958. Pregnancy in the young primigravida. Amer. J. Obstet. Gynecol. 76:746-753.

Records of 299 obstetrical patients, 15 years of age or younger, were reviewed. The major obstetrical complication was acute toxemia. Labor and delivery were not influenced by age. Complications were greater in patients who were not married and who did not receive adequate care. The authors stress the need for sex and parenthood education.

Robinson, D. 1967. Obstetrical care and social patterns in metropolitan Boston. Pub. Health Rep. 82:117-126.

Social patterns and obstetrical care among 24,460 live births in metropolitan Boston were studied. Teen-agers accounted for 39.7 percent of all out-of-wedlock births. Births of 48 infants annually to girls under 15 years of age were reported. As many nonwhites as whites were under 20 years of age. The young girls depended primarily on hospital interns and residents for medical care. The author comments that the least experienced physicians care for the youngest patients and those with acute social problems.

Rogers, K. D., and G. Reese. 1964. Health studies--presumably normal high school students. 1. Physical appraisal. Amer. J. Dis. Child. 108:572.

Detailed medical examinations were completed on 497 white, middle-class females 14 to 19 years of age. Data on weight, hematocrits, fasting blood sugars, and other relevant physical and biochemical indices were obtained.

Russell, J. K. 1969. Pregnancy in the young teenager. Lancet 1(7590):365-366.

Four case histories of pregnancies in young girls are presented. The psychological and social problems these girls must face are illuminated.

Santow, G. S. 1965. Obstetrics in the adolescent: A clinical survey. Med. J. Aust. 2:488-491.

A comparison was made between 170 primiparas 13 to 16 years of age and a control group of primiparas 19 to 21 years of age. Pre-eclamptic toxemia occurred 1-1/3 times more often in the primiparas 13 to 16 years of age than in the older control group.

Schendel, H. E., and S. S. Mills. 1968. Nitrogen balance in healthy adolescent girls. Fed. Proc. 27:726.

Nitrogen retention was studied in five healthy premenarchal adolescent girls, 11 to 12 years of age, eating a self-selected diet ad libitum and living in their normal environment.

Schlaphoff, D., and F. A. Johnston. 1949. The iron requirement of six adolescent girls. J. Nutr. 39:67-82.

On a 9-week controlled diet, six girls, 13 and 14 years of age, had an average iron retention of 1.18 mg/day on an intake of 8.6 mg/day and an average retention of 1.52 mg/day on an intake of about 11.7 mg/day. The minimum required retention for maintenance of growth and menstrual loss replacement was estimated at 1.00 mg/day. On the basis of the study, iron intake of 12 to 13 mg/day was recommended.

Shapiro, L. R., M. C. Hampton, and R. L. Huenemann. 1967. Teenagers: Their body size, shape, food, and activity. J. Sch. Health. 37:166-170.

A 4-year longitudinal study of about 1,000 girls and boys, grades nine to twelve, showed that 60 percent of the girls

wanted to lose weight but only 17 percent needed to do so. Few of the girls on modified diets succeeded in losing weight over a 3-year period. Obesity or mild obesity was found in 11 to 17 percent of the girls. The obese ate less than the non-obese, and their diets had lower nutritive value.

Sinclair, R. St. C. 1952. Pregnancy and labour in the young mother. J. Obstet. Gynaecol. Brit. Emp. 59:504-509.

Pregnancy and labor of 700 primigravidas below 16 years of age are discussed. Importance of antenatal care is stressed.

Stearn, R. H. 1963. The adolescent primigravida. Lancet 2:1083-1085.

A report on obstetric experience with 30 unmarried girls under 16 years of age who were referred from a home for adolescents. Physical maturity, good mental tolerance of pregnancy, and ease of delivery were observed.

Stine, O. C., R. V. Rider, and E. Sweeney. 1964. School-leaving due to pregnancy in an urban adolescent population. Amer. J. Pub. Health 54:1-6.

Mortality rates, frequency of low birth weights (prematures), frequency of school-age pregnancy, time of initiation of prenatal care, and geographic location of high fertility rates are reported through a review of the birth certificates of infants born in Baltimore, Maryland, to women 16 years of age and under during 1957, 1960, and 1961.

Stuart, H. C. 1946. Normal growth and development during adolescence. New Engl. J. Med. 234:666-672, 693-700, 732-738.

Changes in physiologic functions (e.g., basal metabolism and calcium and nitrogen retention) and under nutrition are treated in the concluding section.

Tanner, J. M. 1960. The development of the female reproductive system during adolescence. Clin. Obstet. Gynecol. 3:135-145.

A report on a study carried out in London, England.

Tom, F., and P. Tom. 1966. The age of the menarche in Jamaica. W. Indian Med. J. 15:83-88.

The average recorded age of onset of menses of 1,000 patients attending an antenatal clinic in the West Indies was 14.9 years; the average for 391 nurses was 13.6 years. The difference may have been due to lower socioeconomic level of the patients and poorer nutrition.

Utian, W. H. 1967. Obstetrical implications of pregnancy in primigravidae aged 16 years or less. Brit. Med. J. 2:734-736.

The course and outcome of pregnancy in 100 primigravidas 13 to 16 years of age are compared with those for 100 primigravidas 22 years of age. The younger group showed a higher incidence of hypertension, toxemia, prematurity, prolonged labor, and perinatal mortality.

Wenberg, B. G., M. T. Boedeker, and C. Schuck. 1965. Nutritive value of diets in Indian boarding schools in the Dakotas. Observations of growth and development of adolescent Sioux Indian girls. J. Amer. Diet. Ass. 46:96.

Nutritive value of diets is estimated from food inventories and from height, weight, and hemoglobin determinations completed on girls 12 to 14 years of age.

Wenzel, B. J., H. B. Stults, and J. Mayer. 1962. Hypoferraemia in obese adolescents. Lancet 2:327.

On the basis of height, weight, hemoglobin, and serum iron measurements, 36 out of 192 girls 11 to 19 years of age were found to be obese and to have low serum iron levels and normal hemoglobins.

Wharton, M. A. 1963. Nutritive intake of adolescents. J. Amer. Diet. Ass. 42:306.

Three-day dietary records were kept by 421 adolescent boys and girls 13 to 18 years of age from a depressed area. Mean daily intakes of calories, fat, protein, three minerals, and five vitamins were calculated and evaluated.

White, H. S. 1968. Iron nutriture of girls and women: I. Dietary iron and hemoglobin concentrations. J. Amer. Diet. Ass. 53:563-569.

Data on iron intake and hemoglobin concentrations from surveys conducted during a 20-year period are presented. Average iron intakes were found to be between 10 and 12 mg/day. Hemoglobin data did not indicate a high prevalence of anemia in the survey populations.

White, H. S. 1968. Iron nutriture of girls and women: II. Iron stores. J. Amer. Diet. Ass. 53:570-574.

Estimation of bone marrow iron appears to be the most reliable method for evaluating iron stores. Studies show that many healthy, non-anemic girls and women may be iron deficient.

Young, C. M., S. S. Sipin, and D. A. Roe. 1968. Body composition of pre-adolescent and adolescent girls. J. Amer. Diet. Ass. 53:25-31.

Median body densities decreased and mean skinfold thickness increased in 102 preadolescents and adolescents during sexual and physiologic development. Change in body density and skinfold thickness was more closely related to physiologic age than to chronologic age.

Zacharias, L., and R. J. Wurtman. 1969. Age at menarche. N. Engl. J. Med. 280:868-875.

Genetic and environmental influences affecting age at menarche are summarized in this report.

Birth Weight

Andersen, H., and M. Plum. 1965. Gestation length and birth weight in cattle and buffaloes: A review. J. Dairy Sci. 48:1224-1235.

Wide variance in gestation length and birth weight exists among breeds. Age does not appear to affect length of gestation, although younger cows have lighter calves. In animals of the same breed, there is a positive relationship between gestation length and birth weight.

Babson, S. G., J. Kangas, N. Young, and J. L. Bramhall. 1964. Growth and development of twins of dissimilar size at birth. Pediatrics 33:327-333.

A review of birth records of 20 pairs of twins in which the smaller twin weighed less than 2000 g and at least 25 percent less than the larger co-twin at birth. Sixteen of these pairs were examined at a median age of 8-1/2 years. The larger twins were significantly taller and heavier and had larger head circumferences, higher intelligence, and superior language ability than did their smaller co-twins.

Bacola, E., F. C. Behrle, L. de Schweinitz, H. C. Miller, and M. Mira. 1966. Perinatal and environmental factors in late neurogenic sequelae. II. Infants having birth weights from 1500 to 2500 grams. Amer. J. Dis. Child. 112:369-374.

Neonatal respiratory distress and maternal toxemia did not affect subsequent mental development of infants with birth weights of 1500 to 2500 g; however, socioeconomic and cultural factors appeared to play a significant role in the mental development of the infants.

Barker, D. J. P. 1966. Low intelligence: Its relation to length of gestation and rate of foetal growth. Brit. J. Prev. Soc. Med. 20:58-66.

Analysis of birth weights and length of gestation in 606 children with IQ's below 74. Low intelligence is associated with both slower rate of intrauterine growth and a higher incidence of birth before 38 weeks' gestation than is found in total population.

Baumgartner, L. 1962. The public health significance of low birth weight in the USA. WHO Bull. 26:175-182.

Author suggests that the state of physiological development and efficiency of an infant are more important than birth weight in determining survival and suggests factors for consideration when assessing the significance of low birth weight.

Bishop, E. H. 1964. Prematurity. Postgrad. Med. 35:185-188.

A survey of 16,000 consecutive deliveries demonstrated an

association between low birth weight and mother's race and age, hemoglobin, the interval between pregnancies, history of previous births of small babies, and maternal heart size.

Bivings, L. 1934. Racial, geographic, annual and seasonal variations in birth weights. Amer. J. Obstet. Gynecol. 27:725-728.

Average birth weights for infants born of mothers from a low social level were lower than for infants from a high social level in Atlanta, Georgia.

Churchill, J. A. 1965. The relationship between intelligence and birth weight in twins. Neurology 15:341-347.

In 50 sets of twins, lower IQ's were found in the lighter member of the identical group but not in the fraternal group. Hypothesis of intrauterine impoverishment in the etiology of low-birth-weight newborns with mental retardation is discussed. Maternal health and nutrition during pregnancy may provide a partial solution.

Churchill, J. A., J. W. Neff, and D. F. Caldwell. 1966. Birth weight and intelligence. Obstet. Gynecol. 28:425-429.

Children (51) with "undifferentiated" mental retardation had significantly lower birth weights than 51 matched controls with IQ's above 110. The IQ-birthweight relationship was independent of sociocultural factors in the middle-class-population studies.

Dann, M., S. Z. Levine, and E. V. New. 1964. A long-term follow-up study of small premature infants. Pediatrics 33:945-955.

One-hundred prematures with birth weights of 1,000 g or less were followed for varying periods up to 18.5 years. Except for a high incidence of eye defects, physical health was good. The children tended to catch up to normal height standards after the fourth year. The IQ's showed a wide range, with an average of 94.8, significantly lower than that of full-term siblings. Socioeconomic status of the families appeared to be the most important variable in determining the IQ, as there were significantly more IQ's over 100 in higher-status families.

Dean, R. F. A. 1951. Size of baby at birth and yield of breast milk. In Studies of Undernutrition. Wuppertal 1946-49. His Majesty's Stationery Office, London.

Birth weights and the yield of breast milk were studied in 22,000 women who delivered babies during 1945-46 in Germany during the period of food scarcity. Comparisons with 1937-38 births revealed that the babies were 6 percent lighter and possibly slightly shorter and that breast milk supply was up to 25 percent less than in the former period. Author concludes that the diet together with emotional factors was probably responsible.

Drillien, C. M. 1958. A longitudinal study of the growth and development of prematurely and maturely born children. Arch. Dis. Child. I. 33:417-428; IV. (1959) 34:210-217.

The incidence and severity of infections and communicable diseases during the first two years of life were highly correlated with standards of maternal care. Children of low birth weight were especially vulnerable to an adverse environment.

Fujikura, T. and W. H. Niemann. 1967. Birth weight, gestational age, and type of delivery in rhesus monkeys. Amer. J. Obstet. Gynecol. 97:76-80.

When date of conception and length of gestation were accurately known, the variability in birth weights at a specific gestational age of rhesus monkeys was wide. This suggests that fetal development is highly independent, even in the same environmental situation.

Graziani, L. J., E. D. Weitzman, and M. S. A. Velasco. 1968. Neurologic maturation and auditory evoked response in low birth weight infants. Pediatrics 41:483-494.

The nervous systems of small-for-age, low-birth-weight infants were found to be more mature than infants of similar birth weights who were not small for age.

Griswold, D. M., and D. Cavanagh. 1966. Prematurity--the epidemiologic profile of the "high risk" mother. Amer. J. Obstet. Gynecol. 96:878-882.

In terms of premature delivery, the epidemiological profile of the "high-risk" mother is: under 30 years of age; Negro; less than 5 feet 4 inches in height; nonpregnant weight of

less than 140 pounds; a gain in weight during pregnancy of less than 16 pounds; fewer than three visits to the antepartum clinic during pregnancy; and hemoglobin probably under 11 g percent when admitted in labor.

Haworth, J. C., L. Dilling, and M. K. Younoszai. 1967. Relation of blood-glucose to haematocrit, birthweight, and other body measurements in normal and growth-retarded newborn infants. Lancet II:901-905.

No significant relationships were found between blood-glucose, birth weight, and hematocrit in human infants. Infants whose birth weights are more than two standard deviations below the mean for gestational age with proportionately large heads are most prone to neonatal hypoglycemia.

Jansson, I. 1966. Aetiological factors in prematurity. Acta Obstet. Gynecol. Scand. 45:279-300.

Data from 223 mothers with 240 premature infants were compared with data from mothers of mature infants in an attempt to determine cause of prematurity. Toxemia, multiple pregnancies, placental complications, and fetal abnormalities were cited as having etiological implications in prematurity.

Jarvinen, P. A., P. Pankamaa, and O. Kinnunen. 1957. The full-term underdeveloped liveborn infant. Etud. Neonat. 6:3-9.

A report on 246 underdeveloped, underweight, full-term liveborn infants occurring in 55,625 single births in Helsinki. The incidence of toxemia was 21.7 percent. The placental weight and surface area were smaller for the underweight infants than for premature babies of equal weight and normal babies of similar length of gestation.

Klein, J. 1946. Relationship of maternal weight gain to the weight of the newborn baby. Amer. J. Obstet. Gynecol. 52:574.

In 567 cases, no correlation was found between maternal weight gain and birth weight of fetus.

Lane, E. J. 1965. Factors influencing the birth weight in normal pregnancy. Amer. J. Obstet. Gynecol. 91:342.

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Physique and weight gain of the mother correlated with birth weight of the infant. The correlation was less than that between birth weight and gestational age of the infant.

Leitch, I., F. E. Hytten, and W. Z. Billewicz. 1959. The maternal and neonatal weights of some mammalia. Proc. Zool. Soc. London 133:11-27.

When a comparison was made in 114 species between maternal weight and weight of young in mammals, the significant relation is of maternal weight to total weight of young at birth. The logarithms of maternal and newborn weights have a straight-line relation from bats to whales. Relatively, the larger mammals carry a smaller weight of young.

Love, E. J., and R. A. Kinch. 1965. Factors influencing the birth weight in normal pregnancy. Amer. J. Obstet. Gynecol. 91:342-349.

The effects of the sex and gestational age of the infant, of the weight, height, and body build of the mother, and of the maternal weight gain during pregnancy on the birth weight were assessed in over 2,000 normal pregnancies. There was a significant positive correlation between the birth weight and the gestational age for all infants. The weight, height, body build, and weight gain of the mother were significantly and positively correlated with the birth weight. However, these correlations were less than that between the birth weight and the gestational age of the infant.

Naylor, A. F. and N. C. Myrianthopoulos. 1967. The relation of ethnic and selected socio-economic factors to human birth-weight. Ann. Hum. Genet. 31:71-83.

An analysis of 20,000 births included in the collaborative study indicated a significant association of socioeconomic variables with birth weight among all racial groups, but little of the total observed differences in birth weights between whites, Negroes, and Puerto Ricans could be accounted for by socioeconomic differences. White babies appeared to be inherently about 130 g heavier than Negro babies.

Niyogi, A. K., and B. W. Gajwani. 1963. Influence of maternal factors on the weight of the human newborn. J. Indian Med. Ass. 40:64-68.

In a study of 122 villages in India a significant and positive correlation was found between birth weight of infants and the weight and height of the mother only if the mother was over 30 years, over third parity, and the interval between last and present delivery was over 2 years. Data suggest that the influence of mother's weight on infant's birth weight may have an environmental basis.

North, A. F. 1966. Small-for-dates neonates. Pediatrics 38:1013-1019.

Records of 762 infants weighing less than 2,500 g at birth and born between 37 and 44 weeks' gestation were compared with records of infants with similar birth weight born between 28 and 32 weeks' gestation and with records of infants weighing 3,000 to 3,500 g born between 37 and 44 weeks' gestation. Small-for-date infants had a higher incidence of maternal toxemia and hypertension, a greater incidence of primiparous mothers, a lower incidence of gestational bleeding, a lower incidence of low-birth-weight siblings, fewer neonatal complications and deaths, and a lower incidence of hemolytic and pulmonary disease in the newborn period than did weight-matched, preterm controls.

O'Sullivan, J. B., S. S. Gellis, and B. O. Tenney. 1965. Aspects of birth weight and its influencing variables. Amer. J. Obstet. Gynecol. 92:1023-1029.

Variables that influence birth weight are discussed. The apparent influence of parity appeared to be due to the increases in maternal weight associated with increased parity.

Perlstein, M. A., and A. Levinson. 1937. Birth weight. Its statistical correlation with various factors. Amer. J. Dis. Child. 53:1645.

Social and economic status did not have demonstrable effect on birth weight.

Reinke, W. A., and M. Henderson. 1966. Smoking and prematurity in the presence of other variables. Arch. Environ. Health 12:600-606.

Association between infant sex and birth weight and maternal smoking habits was studied in 3,156 Negro women who delivered single live infants. Maternal prenatal weight in relation to height and parity was revealed as a factor to be considered also.

Roszkowski, I., E. Janczewska, and M. Troszynski. 1964. Relative weight deficiency in the newborn. Biol. Neonat. 6:285-291.

The authors studied the characteristics of 183 low-birth-weight babies of 38 or more completed weeks of gestation with clinical traits of maturity. The low-birth-weight group was subdivided into two subgroups: those of 46 cm or longer were called "hypotrophic," and those 45 cm or less "microsomic." An association was found between "hypotrophy" and foci of infection in the mother.

Silverman, W. A., and J. C. Sinclair. 1966. Infants of low birth weight. New Engl. J. Med. 274:448-450.

Terminology for newborn of low birth weight is discussed, and the difficulties encountered in obtaining fetal growth data are described. Current etiological theories of fetal malnutrition and hypoplastic fetal growth disturbances are also discussed, and recommendations are made for prevention and care.

Van den Berg, B. J., and J. Yerushalmy. 1966. Relationship of the rate of intrauterine growth of infants of low birth weight to mortality, morbidity, and congenital anomalies. J. Pediat. 69:531-545.

The authors classified a large series of low-birth-weight infants in four categories based on both birth weight and gestational age representing different rates of intrauterine growth. In a follow-up study of 367 infants, they found differences in neonatal mortality, frequency of congenital malformations, adaptation to extrauterine environment, and certain characteristics of the placentas among infants falling into the four categories.

Wigglesworth, J. S. 1969. Pathological and experimental studies of intrauterine malnutrition. Proc. Nutr. Soc. 28:(1):31-35.

A description of the characteristics and causes of small-for-date babies is presented.

Willerman, L., and J. A. Churchill. 1967. Intelligence and birth weight in identical twins. Child Develop. 38:623-629.

Two groups of identical twins (27 sets) 5 to 15 years of age were given WISC. One, racially mixed, group was from a low socioeconomic class, and the second group was from all-white middle class. In both groups the members of pairs with lower birth weights had lower verbal and performance IQ's.

Diabetes

Barnes, P. H. 1961. Prediabetes and pregnancy. Can. Med. Ass. J. 85:681-688.

The significance of the prediabetic state is reviewed. In 41 women with abnormal glucose tolerance curves, an increased incidence of previous fetal losses, toxemia, large babies, and a family history of diabetes are noted.

Breidahl, H. D. 1966. The growth and development of children born to mothers with diabetes. Med. J. Aust. 1:268-270.

Twelve-month-old infants born to diabetic mothers and fathers had a higher percentage of congenital abnormalities than infants born of normal parents.

Carrington, E. R. 1960. The effect of maternal prediabetes. Clin. Obstet. Gynecol. 3:911-920.

Transient hyperglycemia in pregnancy may produce virtually no symptoms in the mother but may be of serious consequence to the fetus. Hyperglycemia tends to increase as pregnancy advances, reverts to normal after delivery, and tends to increase in severity in successive pregnancies. Recognition and control of maternal abnormalities in carbohydrate metabolism and treatment of infants can prevent most neonatal deaths due to this disorder.

Carrington, E. R., C. R. Shuman, and H. S. Reardon. 1957. Evaluation of the prediabetic state during pregnancy. Obstet. Gynecol. 9:664-669.

Study stresses the importance of recognizing the prediabetic state early in pregnancy.

Dandrow, R. V., and J. B. O'Sullivan. 1966. Obstetric hazards of gestational diabetes. Amer. J. Obstet. Gynecol. 96:1144-1147.

In a prospective study of 188 gestational diabetic and 323 randomly selected patients with normal glucose tolerance, the gestational diabetics had a significantly higher viable fetal loss as well as a general increase in the number of complications associated with pregnancy.

Dekaban, A., and R. Baird. 1959. The outcome of pregnancy in diabetic women. J. Pediat. 55:563-576.

The outcome of 235 pregnancies in diabetic and prediabetic women was investigated and the results were compared with 249 pregnancies of matched normal controls. Fetal wastage in the diabetic women was significantly greater, and the percentage of abnormal surviving offspring (mental deficiency, congenital malformation, birth injury, and epilepsy) was 3.8 in the diabetic pregnancies and 0.4 percent in the normal controls.

Fitzgerald, M. G., J. M. Malins, D. J. O'Sullivan, and M. Wall. 1961. The effect of sex and parity on the incidence of diabetes mellitus. Quart. J. Med. 30:57-70.

The incidence of diabetes increased with increased parity, becoming twice as common in women who have had three children and six times as common in those who have had six or more.

Jackson, W. P. U. 1961. Is pregnancy diabetogenic? Lancet 7210-7218:1369-1372.

No evidence was found that pregnancy was diabetogenic in normal women except when combined with additional stress. Figures relating parity, age at diagnosis, incidence of diabetes, and glucose tolerance tests in different parity groups show no evidence that parity is important in producing overt diabetes.

Milner, R. D. G., and C. N. Hales. 1965. Effect of intravenous glucose on concentration of insulin in maternal and umbilical cord plasma. Brit. Med. J. i:284-286.

A rise in maternal blood-sugar concentrations was followed by a rise in fetal blood sugar and plasma insulin. The rise in fetal insulin could be detected within 20 minutes of a maternal injection of glucose.

Naeye, R. L. 1965. Infants of diabetic mothers: A quantitative morphologic study. Pediatrics 35:980-988.

Thirty infants, stillborn or dead in the first 2 days of life, born of diabetic mothers were examined after death and their birth weight and length were compared with normal control infants born of nondiabetic mothers. Individual tissues of organs were also compared.

Navarette, V. N., I. H. Torres, I. R. Rivera, V. P. Shor, and P. M. Gracia. 1967. Maternal carbohydrate disorder and congenital malformations. Diabetes 16:127-130.

Of 152 mothers who had delivered malformed infants, 10 percent had abnormal standard glucose tolerance tests, and 45 percent had abnormal triamcinolone glucose tolerance tests. In a control group of 60 mothers, only 3.3 percent had abnormal triamcinolone glucose tolerance tests. The study suggests a relationship between carbohydrate disturbance and malformations.

O'Sullivan, J. B., S. S. Gellis, R. V. Dandrow, and B. O. Tenney. 1966. The potential diabetic and her treatment in pregnancy. Obstet. Gynecol. 27:683-689.

A significant increased number of viable fetal losses was found in women with abnormal glucose tolerance curves as opposed to normal pregnant controls. Treatment with insulin and diet did not improve the fetal loss rate.

Pantelakis, S. N., A. H. Cameron, S. Davidson, P. M. Dunn, A. S. Fosbrooke, J. J. Lloyd, J. M. Malins, and O. H. Wolff. 1964. The diabetic pregnancy. Arch. Dis. Child. 39:334-341.

Serial estimations of serum lipoproteins were determined during pregnancy in 52 diabetic and 21 nondiabetic females. During the 12th to 16th weeks of pregnancy, total lipid, total cholesterol phospholipid, and α-lipoprotein were higher in the diabetic females; after 16 weeks, all fractions

increased but no difference was found between the two groups; after 32 weeks, α-lipoprotein decreased in non-diabetics but remained raised in diabetics. No correlation was found between lipid levels and the outcome of pregnancy.

Rubin, A., and D. P. Murphy. 1958. Studies in human reproduction. III. The frequency of congenital malformations in the offspring of nondiabetic and diabetic individuals. J. Pediat. 53:579-585.

The high mortality rate of infants of diabetic women is not due primarily to congenital anomalies.

Williger, V. M. 1966. Fetal outcome in the diabetic pregnancy. Amer. J. Obstet. Gynecol. 94:57-61.

In an analysis of 115 case histories, there was an increase in infant mortality associated with inadequate control of maternal diabetics. Patients with family or obstetrical histories suggestive of gestational diabetes should be closely supervised in pregnancy.

Wilson, R. B. (ed.) 1962. Diabetes and pregnancy. Clin. Obstet. Gynecol. 5:333-496.

Practical considerations of management of diabetes mellitus in pregnancy are presented along with a discussion of pathological physiology.

Yen, S. C. 1964. Abnormal carbohydrate metabolism and pregnancy. Amer. J. Obstet. Gynecol. 90:468-473.

In 442 Guamanian women there was an increasing incidence of maternal and perinatal complications as degrees of abnormality in glucose tolerance curves increased.

American Academy of Pediatrics, Committee on nutrition. 1961. Vitamin K compounds and the water-soluble analogues. Pediatrics 28:501-507.

The Committee recommended that vitamin K prophylaxis be administered to the infant after birth rather than prenatally.

Ammerman, C. B., L. R. Arrington, A. C. Warnick, J. L. Edwards, R. L. Shirley, and G. K. Davis. 1964. Reproduction and lactation in rats fed excessive iodine. J. Nutr. 84:107-112.

In a series of experiments, adult female rats fed zero to 2,500 ppm supplemental iodine from zero to approximately 35 days prepartum and after normal littering, survival of young was observed. Other females were killed either between 17 and 19 days of pregnancy or between 24 and 48 hours postpartum to observe ovulation rate, implantation rate, development of normal fetuses, and histology of mammary tissue.

Antonov, A. N. 1947. Children born during the siege of Leningrad in 1942. J. Pediat. 30:250-259.

Data for the starvation period during the siege of Leningrad showed a positive relationship between premature births and still births and the restriction of food intake.

Bagchi, K., and A. K. Bose. 1962. Effect of low nutrient intake during pregnancy on obstetrical performance and offspring. Amer. J. Clin. Nutr. 11:586-592.

An investigation of nutritional status, obstetrical performance, and health of offspring of 150 pregnant women in India from low socioeconomic strata who lived on a diet deficient in most nutrients, especially calories and proteins. Fifty women of higher economic strata were used for controls. Nutritional status of the women during pregnancy, the condition of the offspring, and the obstetrical performance did not differ significantly in the two groups. The authors suggest that chronic nutritional deficiency similar to that seen in the experimental groups

may bring about metabolic changes which facilitate the economic utilization of nutrients.

Balfour, M. J. 1944. Supplementary feeding in pregnancy. Lancet 1:208-211.

In a study of 11,618 pregnant women receiving food supplements containing vitamin A, vitamin D, vitamin B, calcium, phosphorus, and iron, there was a significant reduction in the stillbirth and neonatal mortality rates over that of a group of 8,095 controls receiving no supplementation.

Balfour, M. I., and S. K. Talpade. 1932. Influence of diet on pregnancy and early infant mortality in India. Indian Med. Gaz. 67:601-606.

An increased average birth weight and a lower incidence of permature births was found in women in Bombay given a diet containing whole wheat or millet compared with women whose diet consisted of polished rice.

Beaton, G. H., G. Arroyave, and M. Flores. '1964. Alteration in serum proteins during pregnancy and lactation in urban and rural populations in Guatemala. Amer. J. Clin. Nutr. 14:269-279.

Study of 175 pregnant, lactating, and nonpregnant Guatemalan women from a middle and upper economic urban center and low socioeconomic rural communities showed moderate protein intakes for all, with the rural women having a lower proportion of animal protein. Alteration in biochemical parameters in pregnancy was investigated and related to protein intake.

Black, J. A., and R.E. Bonham. 1963. Association between aortic stenosis and facies of severe infantile hypercalaemia. Lancet 2:745-748.

Presentation of five cases of severe infantile hypercalcemia. Description of vitamin D intake of mother during pregnancy described as "normal" in all cases.

Bose, A. K., and K. Bagchi. 1962. Nutritional adaptation in pregnancy. J. Indian Med. Ass. 38:334-338.

A detailed nutrition survey conducted on a group of pregnant women of low socioeconomic status living on a deficient diet did not reveal clinical or biochemical abnormalities.

Bourquin, A., and R. Bennum. 1957. The preconception diet of women who have had unsuccessful pregnancies. Amer. J. Clin. Nutr. 5:62-69.

A comparative study of the preconception dietary habits of 51 women who had histories of habitual abortion and women who had had successful pregnancies. The average dietary intake did not differ statistically except for thiamin, which was higher for the group with successful pregnancies.

Brandt, M. B. 1963. Nutrition in pregnancy. Clin. Obstet. Gynecol. 6(3):604-618.

Discussion for the practicing obstetrician of nutritional management in pregnancy.

Brewer, T. 1967. Human pregnancy nutrition: A clinical view. Obstet. Gynecol. 30:605-607.

A challenge is presented to obstetricians to provide pregnant women with adequate nutrition to meet the needs of their individual pregnancies. Explanation is given of food components of prenatal diet, and discussion of dietary supplements is included.

Brown, M. L., and R. L. Pike. 1960. Blood pressure and thiocyanate space in the vitamin deficient rat during pregnancy. J. Nutr. 70:453-458.

Changes in blood pressure and thiocyanate space are investigated in pregnant and nonpregnant vitamin B_6-deficient and control rats. Blood pressures were within the normal range for both groups. Total thiocyanate space increased significantly in deficient animals fed pyridoxine and to a lesser degree in animals fed deoxypyridoxine.

Brown, M. L., and C. H. Snodgrass. 1965. Effect of dietary level of thiamine on reproduction in the rat. J. Nutr. 85:102-105.

A study of the effect of chronic thiamin deficiency from

weaning until mating in female rats and the reproductive performance and maternal biochemical response when thiamin levels met or exceeded those generally considered adequate.

Brown, M. L., and C. H. Snodgrass. 1965. Reproduction and maternal response of the rat when thiamine intake is limited. J. Nutr. 87:353-356.

Reproduction performance, serum proteins, and hematological response were studied in female rats maintained from weaning until mating on a diet containing 2.5 mg of thiamin/kg or the same diet containing 1.0 mg of thiamin/kg. During gestation rats were fed a diet containing 5.0 mg of thiamin/kg or 0.5 mg of thiamin/kg. The 1.0 mg of thiamin/kg prepregnancy diet did not reduce fertility and affected food intake and weight gain during pregnancy only when diet in pregnancy was low in thiamin. Other variables are discussed.

Brzezinski, A., Y. M. Bromberg, and K. Braun. 1947. Riboflavin deficiency in pregnancy, its relationship to the course of pregnancy and to the condition of the foetus. J. Obstet. Gynaecol. Brit. Emp. 54:182-186.

In a study of 325 pregnant women, a relationship was found between low riboflavin excretion in the urine and prematurity, vomiting, antenatal death of the fetus, hypogalactia, and agalactia.

Burke, B. S., V. A. Beal, S. B. Kirkwood, and H. C. Stuart. 1943. The influence of nutrition upon the condition of the infant at birth. J. Nutr. 26:569-583.

A study to relate the nutritional quality of the diet of 216 women during pregnancy with the condition of their infants at birth and during the first 2 weeks of life. The diets were rated as excellent, good, fair, poor, or very poor, and the infants were classified by medical rating as superior, good, fair, or the poorest group. A statistically significant relationship was shown between the quality of the diet of the mother during the pregnancy and the group into which the infant was classified at birth and within the first 2 weeks of life.

Burke, B. S., V. A. Beal, S. B. Kirkwood, and H. C. Stuart. 1943. Nutrition studies during pregnancy. Amer. J. Obstet. Gynecol. 46:38-52.

A relationship was found between the prenatal dietary rating and the course of pregnancy, but it is not so marked as between the prenatal dietary rating and the condition of the infant at birth.

Burke, B. S., V. V. Harding, and H. C. Stuart. 1943. Nutrition studies during pregnancy. IV. Relation of protein content of mother's diet during pregnancy to birth length, birth weight, and condition of infant at birth. J. Pediat. 23:506-515.

In a study of 216 pregnancies, a positive relationship was found between protein intake of the mother and the length, weight, and general physical well-being of her infant at birth.

Burke, B. S., and H. C. Stuart. 1948. Nutritional requirements during pregnancy and lactation. J. Amer. Med. Ass. 137:119-128.

Discusses population studies that indicate a relationship between the character of the maternal diet and the outcome of pregnancy.

Cameron, C. S., and S. Graham. 1944. Antenatal diet and its influence on stillbirths and prematurity. Glasgow Med. J. 24:1-7.

Mothers with full-term infants had had better diets than mothers of stillborn or prematurely born infants.

Carlos, J. P., A. M. Gittleson, and W. Haddon. 1962. Caries in deciduous teeth in relation to maternal ingestion of fluoride. Pub. Health Rep. 77:658-660.

Little difference was found between the caries incidence in children whose mothers drank fluoridated water during pregnancy and in those whose mothers did not.

Cellier, K. M., and M. E. Hankin. 1963. Studies of Nutrition in pregnancy. I. Some considerations in collecting dietary information. Amer. J. Clin. Nutr. 13:55-62.

The validity of a 4-day diet record and changes in eating habits of women during the course of pregnancy and lactation are discussed.

Clements, F. W. 1961 Nutrition in maternal and infant feeding. Fed. Proc. 20:(1):165-168.

Discusses the effect of different levels of maternal nutrition on the infant, the significance of social and emotional factors in childbearing, and the effects of specific dietary patterns on both pregnant women and their infants.

Clements, F. W. 1962. Effect of the level of nutrition on birth weight. WHO Bull. 26:301-303.

A review of evidence of the influence of lifetime nutrition and of the diet during pregnancy on birth weight of infants. Both poor pregravid diets and poor nutrition during pregnancy may result in a higher percentage of infants of low birth weight.

Cochrane, W. A. 1965. Overnutrition in prenatal and neonatal life: A problem? Can. Med. Ass. J. 93:893-899.

The author draws attention to the fact that little information is available concerning overnutrition during prenatal and neonatal life. Discusses some clinical and biochemical disorders of the newborn that are related to excessive ingestion of calories, fat, protein, vitamins, and minerals, before and after birth.

Committee on Nutrition of the American Academy of Pediatrics. 1965. Vitamin D intake and the hypercalcemic syndrome. Pediatrics 35:46-47.

Because of evidence suggesting that the severe form of infantile hypercalcemia may arise in utero, the Committee recommends that total vitamin D intake be carefully regulated for pregnant women and not exceed 400 IU/day.

Conn, L. C., J. R. Vant, and M. M. Malone. 1936. Some aspects of maternal nutrition. Surg. Gynecol. Obstet. 62:377-383.

Recommends a diet for pregnant women which includes 2,000 to 2,500 calories, 70 to 100 g of protein, 18 mg of food iron, and 1.6 g of calcium per day.

Crump, E. P., E. Payton, and C. P. Horton. 1959. Growth and development. 4. Relationship between prenatal maternal nutrition and socioeconomic index, weight of mother, and birth weight of infant. Amer. J. Obstet. Gynecol. 77:562-572.

A nutrition index was calculated by a 7-day menu survey of 483 pregnant Negro women receiving private, hospital, clinic, or no prenatal care. No significant relation was found between nutrition of the mother and weight of the baby. There was a slight relation between maternal nutrition and the socioeconomic index.

Darby, W. J., R. O. Cannon, and M. M. Kaser. 1948. The biochemical assessment of nutritional status during pregnancy. Obstet. Gynecol. Surv. 3:704-715.

Preliminary data are presented on blood concentrations of vitamins A, E, C and the B vitamins completed on 1,300 women studied during each trimester of pregnancy and at six weeks postpartum. Reports a decrease in vitamin C, hemoglobin, vitamin A, and serum protein and compares changes with percent dilution of plasma volume. Fat-soluble factors were observed to rise. Metabolic changes and the influence of seasonal differences in dietary intake are discussed.

Darby, W. J., P. M. Densen, R. O. Cannon, E. Bridgforth, M. P. Martin, M. M. Kaser, C. Peterson, A. Christie, W. W. Frye, K. Justus, G. S. McClellan, C. Williams, P. J. Hahn, C. W. Sheppard, E. L. Carothers, and J. A. Newbill. 1953. The Vanderbilt cooperative study of maternal and infant nutrition, I, II, and III. J. Nutr. 51(4):539-563.

Describes the background, design, methodology, and population sample of a study of nutrition in pregnancy that included 2,338 pregnant white women.

Darby, W. J., W. J. McGanity, M. P. Martin, E. Bridgforth, P. M. Densen, M. M. Kaser, P. J. Ogle, J. A. Newbill, A. Stockell, M. E. Ferguson, O. Touster, G. S. McClellan, C. Williams, and R. O. Cannon. 1953. The Vanderbilt cooperative study of maternal and infant nutrition, IV. Dietary, laboratory and physical findings in 2,129 delivered pregnancies. J. Nutr. 51(4):565-597.

The nutritional characteristics are revealed by records of dietary intake, laboratory assessments, and physical examinations described for an unselected group of 2,129 pregnant white women. Findings are discussed in relation to altered physiology of pregnancy.

Davis, J. A., D. R. Harvey, and J. S. Yu. 1965. Neonatal fits associated with hypomagnesaemia. Arch. Dis. Child. 40:286-290.

A case history of a newborn infant with persistent convulsions associated with low serum calcium and magnesium levels. Magnesium deficiency in the mother and the infant's diet of cow's milk with vitamin D were suggested as factors.

Dieckmann, W. J., F. L. Adair, H. Michel, S. Kramer, F. Dunkle, B. Arthur, M. Costin, A. Campbell, A. C. Wensley, and E. Lorang. 1944. Effect of complementing the diet in pregnancy with calcium, phosphorus, iron, and vitamins A and D. Amer. J. Obstet. Gynecol. 47:357-368.

Change in biochemical or clinical indices were not observed in pregnant women receiving vitamin and mineral supplements. The authors note that if the accustomed basal diet is adequate to meet needs of both mother and fetus, additional nutrients may have no effect.

Dieckmann, W. J., D. F. Turner, and B. A. Ruby. 1945. Diet regulation and controlled weight in pregnancy. Amer. J. Obstet. Gynecol. 50:701-712.

A critical discussion of previously reported study that discusses the role of nutrition in pregnancy.

Ebbs, J. H., W. A. Scott, F. F. Tisdall, W. J. Moyle, and M. Bell. 1942. Nutrition in Pregnancy. Can. Med. Ass. J. 46:1-6.

At 6 months of age, weights of infants born of mothers who had received supplemented diets during pregnancy were higher than infants of mothers who had an unsupplemented poor diet during pregnancy.

Ebbs, J. H., F. F. Tisdall, and W. A. Scott. 1942. The influence of prenatal diet on the mother and child. Milbank Mem. Fund Quart. 20:35-46.

Pregnant women from low-income families were divided into three groups for study. Those in the first group, estimated to have a poor diet, were given food supplements during the last three to four months of pregnancy. Those in the second group had a moderately good diet and were given nutrition education only. Those in the third group, with a poor diet, served as controls. During the entire course of pregnancy, the mothers with the good or supplemented diets enjoyed better health, had fewer complications, and were better obstetrical risks than those left on poor diets. Premature babies were born to 2.7 percent of mothers on good diets and to 8 percent of mothers on poor diets; miscarriages or stillbirths to 1.2 percent and 9 percent, respectively.

Edwards, C. H., S. McDonald, Jr., R. Mitchell, L. Jones, L. Mason, and L. Trigg. 1964. Effect of clay and cornstarch intake on women and their infants. J. Amer. Diet. Ass. 44:109-115.

Analyses of diets, blood, and excreta from a group of pregnant clay and cornstarch eaters and the condition of their infants at birth was reported. Dietary intakes of protein, calcium, and iron were below recommendations, and blood hemoglobins were low. The condition of infants at birth was rated lower for mothers taking clay than for controls.

Ehrenfest, H. 1919. Can labor be facilitated by a specific diet of the mother during pregnancy? Amer. J. Obstet. Gynecol. 80:441-464.

A presentation of clinical and animal evidence that disproved the prevailing view of the time that the desirable diet for pregnant women is low in carbohydrate and water and high in protein.

Elias, H. L. 1936. A clinical study of the influence of Vitamin B supplements 2: On maternal health during gestation and labor. J. Pediat. 8:352-361.

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Enriching diets of pregnant women did not influence course and outcome of pregnancy.

El-Maraghi, N. R. H., B. S. Platt, and R. J. C. Stewart. 1966. The effect of reproduction on the interaction of dietary protein and calcium. Brit. J. Nutr. 20:733-745.

An investigation of the interaction of protein and calcium and their relative importance in rats for maintainence of the skeletal structure of mother and offspring during pregnancy and lactation.

English, R. M., and N. E. Hitchcock. 1968. Nutrient intakes during pregnancy and lactation in a group of Australian women. Brit. J. Nutr. 22:615-624.

A study of changes that took place in the quantity and quality of nutrient intakes of 26 healthy women during their first reproductive cycles. All had normal full-term babies, with an average weight gain of 10.3 kg. Caloric intakes did not increase during pregnancy but did during lactation. Intakes of iron and ascorbic acid were below recommended allowances.

Esh, G. C., T. S. Sutton, J. W. Hibbs, and W. E. Krauss. 1948. Effect of soya phosphatides on the absorption and utilization of vitamin A in dairy animals. J. Dairy Sci. 31:461.

Feeding vitamin A plus soya lecithin to pregnant cows caused an increase in vitamin A absorption and utilization in both the mother and fetus.

Fraser, D. 1967. The relation between infantile hypercalcemia and vitamin D public health implications in North America. Pediatrics. 40:115-126

Suggests that if vitamin D plays a role in the intrauterine development of the severe form of infantile hypercalcemia, it does so by placental transfer of the vitamin from the mother to a vitamin D-sensitive fetus or by producing a response in a sensitive mother which is deleterious to the fetus. Total intake of vitamin from all sources should not exceed 400 IU/day during pregnancy. There is need for studies of the vitamin D metabolism of the pregnant mothers of affected infants.

Fraser, D., B. S. Langford, S. W. Kooh, and L. Paunier. 1966. A new look at infantile hypercalcemia. Pediat. Clin. N. Amer. 13(2):503-525.

Review of a clinical aspect of infantile hypercalcemia, discussing evidence regarding causation, estimates of incidence, and prophylactic measures. In the two cases presented, mothers during pregnancy had a vitamin D intake of approximately 1,000 IU/day.

Friedman, L. V. 1926. Diet in pregnancy; attempt to control size of baby. Boston Med. Surg. J. 195:1015.

Pregnant women who gained less than one-half pound a week from the 12th to the 40th week of pregnancy had shorter first stages of labor, and the babies had a slight decrease in birth weight.

Friedman, W. F., and L. F. Mills. 1969. The relationship between vitamin D and the craniofacial and dental anomalies of the supravalvular aortic stenosis syndrome. Pediatrics 43:12-18.

A preliminary exploration in rabbits showed a relationship between hypervitaminosis D in the mother and the development in the offspring of craniofacial and dental abnormalities.

Garry, R. C., and D. Stiven. 1935-36. A review of recent work on dietary requirements in pregnancy and lactation, with an attempt to assess human requirements. Nutr. Abstr. Rev. 5:855-887.

A survey of literature to 1935, concluding that, under normal conditions, the self-selected diet of the mother had little effect on the weight of the newborn.

Garry, R. C., and H. O. Wood. 1946. Dietary Requirements in human pregnancy and lactation. Nutr. Abstr. Rev. 15:591-621.

The authors reviewed work done on the subject during 1936-1946. They point out that despite voluminous literature, the additions to scientific knowledge were meager. They note signs of development of a more critical attitude than in the period before 1936.

Gopalán, C. 1961. Maternal and infant nutrition in under developed countries. J. Amer. Diet. Ass. 39:129-131.

In a survey of 300 poor women of South India subsisting on inadequate diets during pregnancy, the mean gain in weight was 6 kg, with the women experiencing a high incidence of anemia, B-complex deficiency, low serum albumin, and low vitamin A levels. There was a high incidence of abortions, stillbirths, and low birth weight. The problems of protein malnutrition and vitamin A deficiency, which are common nutritional hazards of infancy and early childhood, appeared to have their origins in maternal malnutrition. The author discusses the need for more research on the adaptive mechanisms that enable some undernourished women to have normal pregnancies and babies.

Gopalan, C. 1962. Effect of nutrition on pregnancy and lactation. WHO Bull. 26:203-211.

Review of literature on the effect of maternal nutrition on the cause and outcome of pregnancy and on the output and chemical composition of milk in nursing mothers. Highlights areas where further research is needed.

Goss, D. A. 1962. Renal conservation of calcium during pregnancy. Obstet. Gynecol. 20(2):199-203.

From a study of the dietary intakes and urinary excretions of calcium in 205 normal primagravidae and multigravidae, 25 of whom took calcium supplements, the author concludes that routine calcium supplementation during pregnancy is superfluous for most American women.

Gotchel, R. P., and B. F. Lovett. 1960. Vitamin B_{12} absorption during pregnancy. Amer. J. Obstet. Gynecol. 79:113-116.

Plasma B12 levels were significantly elevated during pregnancy in 22 patients receiving 25 μg of vitamin B_{12} and 11 g of D sorbitol when compared with controls.

Hamil, B. M., M. Coryell, C. Roderuck, M. Kaucher, E. Moyer, M. Harris, and H. H. Williams. 1947. Thimaine, riboflavin, nicotinic acid, pantothenic acid and biotin in the urine of newborn infants. Amer. J. Dis. Child. 74:434-446.

The average maximum excretion of thiamin in the urine of newborn infants was 13 μg/100 ml, dropping breast-fed infants during the first week after birth to zero on the seventh day, indicating the low thiamin content of breast milk and low storage in the newborn. Average riboflavin excretion in the urine of newborn infants was 140 μg/100 ml the first day of life, decreasing during the first week to 2 μg on the seventh day. Greater amounts of nicotinic acid were found in the urine of infants during the first 3 days postpartum than the usual levels found in the urine of adults.

Hamil, B. M., B. Munks, E. Z. Moyer, M. Kaucher, and H. H. Williams. 1947. Vitamin C in the blood and urine of the newborn, and in the cord and maternal blood. Amer. J. Dis. Child. 74:417.

The average vitamin C value of the cord blood was double that of the infants' blood 24 hours after delivery. The average value for maternal blood was 50 percent less than that for the babies' blood. The excretion of vitamin C by the newborn was high during the first 2 days after birth and dropped to low levels by the fourth day.

Hankin, M. E., and J. K. Burden. 1964. Nutrition studies in pregnancy. 3: Influence of eating habits on nutrient intakes in pregnancy. Food Nutr. Aust. 21:25-35.

Records of food eaten and answers to questions formed the basis for a study of the food habits of 177 pregnant women before and during pregnancy. Mean intake of milk and calcium rose in the third trimester. The percentage of women eating breakfast rose during pregnancy, and mean dietary ratings and protein intake were related to the quality of breakfast. Most women reported a change in appetite during pregnancy, and 56 percent reported cravings.

Hankin, M. E., J. K. Burden, and E. M. Symonds. 1964. Nutrition studies in pregnancy. Aust. N.Z. J. Obstet. Gynaecol. 4:149-155.

The majority of 174 pregnant women were eating diets that were adequate in terms of the National Academy of Sciences-

National Research Council recommendations. No relationship was found between maternal diet and the outcome of pregnancy in this series.

Hankin, M. E., E. M. Symonds, and K. M. Cellier. 1965. Studies of nutrition in pregnancy, Aust. N.Z. J. Obstet. Gynaecol. 5:86-93.

A diet of 70 g of protein/day, 800 mg of calcium/day, and 2,500 calories/day seemed adequate for the maintenance of lactation.

Hansen, A. E., H. F. Wiese, D. M. Adam, A. N. Boelsche, M. E. Haggard, H. Davis, W. T. Newsom, and L. Pesut. 1964. Influence of diet on blood serum lipids in pregnant women and newborn infants. Amer. J. Clin. Nutr. 15:11-19.

Nutrient intake in pregnancy and serum lipid level of the pregnant woman were not correlated with serum lipid level neonate.

Hepner, R. 1958. Maternal nutrition and the fetus. J. Amer. Med. Ass. 168:1774-1777.

A discussion of the difficulties in studying the effect of nutrition on course and outcome of pregnancy. A presentation of the results of past studies and development of fundamental concepts.

Herbert, V. 1968. Nutritional requirements for vitamin B_{12} and folic acid. Amer. J. Clin. Nutr. 21:743-752.

A review of the nutritional requirements for vitamin B_{12} and folic acid. In pregnancy, folate and vitamin B_{12} requirements are increased because of an increase in the metabolism of one-carbon units.

Hillman, R. W., P. G. Cabaud, D. E. Nilsson, P. D. Arpin, and R. J. Tufano. 1963. Pyridoxine supplementation during pregnancy. Clinical and laboratory observations. Amer. J. Clin. Nutr. 12:427-430.

Effects of vitamin B_6 supplements studied in a double-blind, clinical trial in 1,532 patients. The obstetric value of administering vitamin B_6 to healthy prenatal patients was questioned.

Hillman, R. W., P. G. Cabaud, and R. A. Schenone. 1962. The effects of pyridoxine supplements on the dental caries experience of pregnant women. Amer. J. Clin. Nutr. 10:512-515.

Pyridoxine supplements were given to prenatal patients in capsule (one 20-mg capsule daily) or as lozenges (6.67 mg three times daily). The treated group had a smaller increase in diseased, missing, or filled teeth than a comparable control group of prenatal patients.

Huggett, A. St. G. 1955. Growth, pregnancy, and carbohydrate metabolism. Amer. J. Obstet. Gynecol. 69:1103.

Summarizes the work of the author and others in this field.

Hytten, F. E. 1964. Nutritional aspects of foetal growth, p. 59-65. In C. F. Mills and R. Passmore (ed.) Proc. Sixth Int. Cong. Nutr., Livingstone Ltd., London.

A review of evidence, pro and con, of the relative importance of the adequacy of maternal diet during pregnancy and prepregnancy nutritional status.

Iyenger, L. 1967. Effects of dietary supplements late in pregnancy on the expectant mother and her newborn. Indian J. Med. Res. 55:85-89.

In a South Indian population, women hospitalized during the last 4 weeks of pregnancy and given high protein and calorie diets had higher serum albumin levels, higher gain in weight, and babies with higher mean birth weights than in a general population of similar economic level.

Iyenger, L. 1968. Urinary estrogen excretion in undernourished pregnant Indian women. Amer. J. Obstet. Gynecol. 102:834-838.

After the 36th week of pregnancy, estrogen levels were found to be significantly higher in the urine of well-fed women than in those who were undernourished. When a group of the undernourished were brought into a hospital and placed on a diet containing 80 g of protein, 2,300 calories,

and vitamin and mineral supplements from 36 weeks to term, their estrogen rose to normal levels. A close correlation was observed between estrogen excretion and birth weights.

Jeans, P. C., M. B. Smith, and G. Stearns. 1952. Dietary habits of pregnant women of low income in a rural state. J. Amer. Diet. Ass. 28:27-34.

Data are presented that indicate that a large section of the pregnant population of Iowa had faulty food habits.

Johnson, O. C. 1967. Present knowledge of calories. Nutr. Rev. 25(9):257-261.

A comprehensive discussion of calorie requirements, including those needed in pregnancy. The author states that increased caloric need in pregnancy is the balance between reduced activity of pregnancy and the caloric demands of increased basal metabolic rate of the pregnant woman and the growing fetus.

Kasius, R. V., A. Randall, W. T. Tompkins, and D. G. Wiehl. 1955. Maternal and newborn nutrition studies at Philadelphia Lying-In Hospital newborn studies: I. Size and growth of babies of mothers receiving nutrient supplements. Milbank Mem. Fund Quart. 33:230-245.

No significant relationship was found between the effect of nutritional supplementation of the mothers' diet during pregnancy and the physical size and growth of their infants.

Kaunitz, H., and D. C. McKay. 1964. Food restriction and lipid metabolism in pregnancy. Metabolism 13:(9)837-842.

In an attempt to determine the effect of reduced food intake during pregnancy on tissue and serum lipids, deprived rats were given 30 percent of the daily intake of their freely eating controls.

Kerr, A., Jr. 1943. Weight gain in pregnancy and its relation to weight of infants and to length of labor. Amer. J. Obstet. Gynecol. 45:950-960.

Increased weight gains in pregnant women were associated with increasing weight of infants at birth, but the latter increase had no influence on the duration of labor.

Kirchgessner, M., H. Friesecke, and G. Kock. 1967. Nutrition and the composition of milk. In International monographs: Aspects of animal and human nutrition. Crosby Lockwood and Son, London. 273 p.

The authors outline the chemistry of the major constituents of milk and present comprehensive reviews of the influence of diet on the physical properties and content of fresh milk. It contains several thousand references, mostly to work done in the past 20 years.

Kirksey, A., and R. L. Pike. 1962. Some effects of high and low sodium intake during pregnancy in the rat. I. Food consumption, weight gain, reproductive performance, electrolyte balances, plasma total protein and protein fractions in normal pregnancy. J. Nutr. 77:33-42.

Pregnant rats on low sodium intakes showed languor and debility, ate less food, and gained less weight than rats receiving control or high sodium diets. Sodium and potassium retention and excretion were also affected. Hematocrit levels, concentrations of hemoglobin and plasma total protein, and concentrations of protein fractions were observed in rats receiving low sodium diets and compared with normal controls. Reproductive performance of both groups was also evaluated.

Kugelmass, I. N., and J. E. Tritsch. 1934. Prenatal prevention of potential hemorrhagic disease of the newborn, supplementary report. Amer. J. Obstet. Gynecol. 28:259-261.

By administration of a high protein diet, prothrombin and fibrinogen blood constituents increased and hemorrhagic disease was prevented in a pregnant woman with a history of this disease state.

Kumaresan, P., and C. W. Turner. 1968. Effect of pregnancy on feed consumption and mammary gland growth in rats. Proc. Soc. Exp. Biol. Med. 129:957-960.

Normal voluntary food intake of 18 rats increased during pregnancy.

Larson, R. H. 1964. Effect of prenatal nutrition on oral structures. J. Amer. Diet. Ass. 44:368-373.

A review of the effect of prenatal nutrition on oral structures in animals and man.

Lenkeit, W. 1964. Metabolism and nutrition during pregnancy, p. 397-409. In C. F. Mills, and R. Passmore (ed.) Proc. Sixth Int. Cong. Nutr., Livingstone Ltd., London.

A comparison of intake and excretion of nitrogen, calcium, phosphorus, and sodium from the outset of pregnancy to lactation in the sow indicated positive tolerance with a high and constant plane of nutrition with some periodic variation.

Levine, S. Z. 1948. Proteins and amino acids in nutrition and pregnancy, p. 318-348. In M. Sahyun, Proteins and amino acids in nutrition. Reinhold Publ. Corp., New York.

Extramaternal demands for protein produce a physiological lowering of plasma proteins from preconception levels of 7.0 g/100 ml to 6.2 g by the sixth month of pregnancy, despite high nitrogen balances. Little support was given to the belief that high protein intakes unrelated to pre-existing hypertension and cardiovenal disease predispose to toxemias of pregnancy.

Lewis, J. M., O. Bodansky, M. C. C. Lillienfeld, and H. Schneider. 1947. Supplements of vitamin A and of carotene during pregnancy. Amer. J. Dis. Child. 73:143-150.

The drop in vitamin A content of the mother's blood in the last trimester of pregnancy was prevented by giving 10,000 IU of vitamin A daily. The vitamin A and carotene values of cord blood were no higher in the infants of those mothers who received the vitamin A than in the cord blood of infants whose mothers received no supplements.

Liu, S. H., H. T. Chu, H. C. Hsu, C. H. Chav, and S. H. Chu. 1941. Calcium and phosphorus metabolism in osteomalacia. XI. The pathogenetic role of pregnancy and relative importance of calcium and vitamin D supply. J. Clin. Invest. 20:255-271.

In Chinese women with an adequate supply of vitamin D,

the same degree of calcium retention was maintained on a somewhat lower intake of calcium, while a high level of calcium would not maintain the individual in balance if severe vitamin D depletion was present. A greater mineral conservation seemed to be present in the Chinese women than in other pregnant women. An individual appears to adapt to low dietary intakes, and the requirement for reproductive activity is reduced correspondingly.

Lund, C. J., and M. S. Kimble. 1943. Plasma vitamin A and carotene of the newborn infant with consideration of fetal-maternal relationships. Amer. J. Obstet. Gynecol. 46:207-221.

In human beings, the carotene blood plasma level of infants varied with the level in the mothers' blood. Vitamin A levels were independent of maternal values.

Macomber, D. 1934. The effect of changes in the amount of protein upon pregnancy and lactation. Amer. J. Obstet. Gynecol. 27:483-492.

Protein at a level of 20 percent of calories in the diet of pregnant rats was found to be superior for fertility and for success in pregnancy to all lower levels. This level for rats was calculated as representing 100 to 125 g of protein daily for women.

Mainland, D. 1963. X-ray bone density of infants in a prenatal nutrition study. Milbank Mem. Fund Quart. 41(1):1-106.

No evidence was found of a relationship between four types of prenatal dietary supplement and 286 infants' bone density in the first postnatal week or its change in density between the first and fourth weeks. A discussion of bone densitometry is included.

Mayer, J. 1963. Some aspects of the relation of nutrition and pregnancy. Postgrad. Med. 33:277-282.

Quality and quantity of diet during pregnancy may vary widely without demonstrable impairment of health of mother or infant.

McCance, R. A., and E. M. Widdowson. 1962. Nutrition and growth. Proc. Roy. Soc. (Biol.). 156:326-337.

Studies on the effects of the plane of nutrition on growth and form in rats, pigs, and cockerels are presented and interpreted.

McCance, R. A., E. M. Widdowson, and C. M. Verdon-Roe. 1938. A study of English diets by the individual method; pregnant women at different economic levels. J. Hyg. 38:596-622.

In the diets of 120 pregnant women at different economic levels, the intakes of calories, fat, and carbohydrate were not affected by income. The intakes of protein, animal protein, calcium, phosphorus, iron, and vitamin B_1 rose with income. Women on better diets were taller and less anemic.

McGanity, W. J., R. O. Cannon, E. B. Bridgforth, M. P. Martin, P. M. Densen, J. A. Newbill, G. S. McClellan, A. Christie, J. C. Peterson, and W. J. Darby. 1954. The Vanderbilt study of maternal and infant nutrition. V. Description and outcome of obstetric sample. Amer. J. Obstet. Gynecol. 67:491-500.

An outline of purpose and plan of study with a description of the 2,046 pregnant women included in the sample is presented. The prenatal and obstetric courses were reported and compared with previously reported incidences of complications.

McGanity, W. J., R. O. Cannon, E. B. Bridgforth, M. P. Martin, P. M. Densen, J. A. Newbill, G. S. McClellan, A. Christie, J. C. Peterson, and W. J. Darby. 1954. The Vanderbilt cooperative study of maternal and infant nutrition VI. Relationship of obstetric performance to nutrition. Amer. J. Obstet. Gynecol. 67:501-527.

The only positive correlation found between obstetric performance, fetal complications, and adequacy of nutrient intake was found in women during the third trimester with dietary intakes of less than 1,500 calories and 50 g of protein. These women had increased complications of pregnancy including pre-eclampsia and eclampsia. The abnormalities were judged to be the cause of the lowered intake, not vice versa.

Metz, J., H. Festenstein, and P. Welch. 1965. Effect of folic acid and vitamin B_{12} supplementation on tests of folate and vitamin B_{12} nutrition in pregnancy. Amer. J. Clin. Nutr. 16:472-479.

The effect of folic acid and vitamin B_{12} supplements was studied in 175 presumably well fed, white pregnant subjects. FIGLU excretion, L. casei folate activity, vitamin B_{12} concentration, hematocrit, and hemoglobin were determined. The results suggest routine supplementation of folic acid during pregnancy.

Mitchell, H. H. 1962. The nutrient requirements for mammalian reproduction, p. 526-570. In H. H. Mitchell, Comparative nutrition of man and domestic animals, Vol. I. Academic Press, New York.

A comprehensive discussion of nutrition in human reproduction.

Moore, H. C. 1960. The effect of a choline deficient diet in young pregnant rats. J. Obstet. Gynaecol. Brit. Emp. 67:297-298.

No renal damage was produced in young pregnant rats.

Moore, M. C., M. B. Purdy, E. J. Gibbons, M. E. Hollinger and G. Goldsmith. 1947. Food habits of women during pregnancy. J. Amer. Diet. Ass. 23:847-853.

In a study of 159 pregnant women, 90 white and 69 Negro, the average level of ascorbic acid of plasma was 0.45 and 0.31 mg/100ml, respectively. Considering 0.60 or above as "normal," 26 percent of the white and 12 percent of the Negro group showed normal values.

Mueller, P. S., F. Solomon, and J. R. Brown. 1964. Free fatty acid concentration in maternal plasma and fetal body fat content. Amer. J. Obstet. Gynecol. 88:196-203.

Rats were fed freely throughout pregnancy a commercial diet with 13 percent Crisco added. Three times daily one group of 20 was given subcutaneous injections of heparin; the remainder served as controls. Litters obtained 19.5 to 21.5 days' gestation. Little difference was noted in

weight gain or protein content; however, the plasma-free fatty acid composition of the fetuses of mothers receiving heparin was 3 times higher than the controls at 19.5 days.

Naismith, D. J. 1966. The requirement for protein and the utilization of protein and calcium during pregnancy. Metabolism. 15:582-595.

The influence of semisynthetic diets of high, medium, and low protein value on the course and outcome of pregnancy in rats was investigated. Concept of storage of nutrients was re-examined and an estimate made of the requirement for protein.

Oldham, H., B. B. Sheft, and T. Porter. 1950. Thiamine and riboflavin intakes and excretions during pregnancy. J. Nutr. 41:231-245.

An increased need for thiamin and riboflavin during a normal pregnancy was not indicated by intakes, excretions, and test dose determinations of 15 pregnant women on self-selected diets. The course of the pregnancies, the condition of the infants at birth, and the postnatal development of the infants did not appear to be correlated with the quality of the diets of the mothers.

Payton, E., E. P. Crump, and C. P. Horton. 1960. Growth and development. VII. Dietary habits of 571 pregnant southern Negro women. J. Amer. Diet. Ass. 37:129-135.

Dietary records of 571 pregnant Negro women were calculated and compared with Recommended Dietary Allowances (1958 edition). The intake of all nutrients, except vitamin A, fell below recommended levels. Customs, education, and economic circumstances influenced dietary intake.

People's League of Health. 1946. The nutrition of expectant and nursing mothers in relation to maternal and infant mortality and morbidity. J. Obstet. Gynecol. Brit. Emp. 53:498-509.

Supplementary vitamins and minerals given to 50 percent of about 5,000 English women were associated with reduction in the incidence of toxemia and of prematurity in the supplemented group, especially in primigravidas.

Pike, R. L. 1964. Sodium intake in pregnancy. J. Amer. Diet. Ass. 44:176-181.

The author discusses previously reported data that suggest that low sodium intakes in pregnant rats produce signs of sodium deficiency, evidenced by lethargy and debility, in significant reductions in plasma and tissue sodium, and in increased width in the zona glomerulosa in the adrenal cortex. It is suggested that, in humans, lowering of plasma sodium and reduction of the quantity of exchangeable sodium during pregnancy may be undesirable.

Pike, R. L., J. Nelson, and M. J. Lehmkuhl. 1962. Some effects of high and low sodium intakes during pregnancy in the rat. J. Nutr. 78:325-329.

Adrenals and hearts from nonpregnant and pregnant rats fed three levels of dietary sodium were analyzed for sodium, potassium, and water. The combination of pregnancy and low sodium intake led to a significant reduction in sodium concentration in maternal tissues and fluids. An increase in water content was associated with a decrease in sodium concentration of the adrenals. The heart was not similarly affected. Cardiac potassium concentration was significantly decreased with decreased sodium.

Posner, L. B., C. M. McCottry, and A. C. Posner. 1957. Pregnancy craving and pica. Obstet. Gynecol. 9:270-272.

A study of 600 consecutive women in the third trimester of pregnancy indicated 394 had cravings, 196 no craving, and 10 had pica. A common craving was for Argo starch.

Robinson, M. 1958. Salt in pregnancy. Lancet 1:178-181.

In a group of 2,077 pregnant women similar in age, parity, and time of first attendance at the clinics, half were told to take more salt, and half were instructed to take less salt. There were 38 cases of toxemia in the women advised to take more salt, and 97 cases in the group advised to take less salt. The women taking more salt had a lower incidence of edema, perinatal death, antepartum hemorrhage, and bleeding.

Roderuck, C. E., H. H. Williams, and I. G. Macy. 1945. Human milk studies. XXIII. Free and total thiamine contents of colostrum and mature human milk. Amer. J. Dis. Child. 70:162-170.

The maternal diet has been shown to be the main source of the amount of thiamin transferred in breast milk.

Rowe, R. D., and R. E. Cooke. 1969. Vitamin D and craniofacial and dental anomalies of supravalvular stenosis. Pediatrics 43:1-2.

A commentary on the status of idiopathic hypercalcemia of infancy and a discussion of pathogenesis are included.

Schumacher, M. F., M. A. Williams, and R. L. Lyman. 1965. Effect of high intakes of thiamine, riboflavin and pyridoxine on reproduction in rats and vitamin requirements of the offspring. J. Nutr. 86:343-349.

During gestation and lactation, female rats received a control diet containing thiamin 0.4, riboflavin 0.4, and pyridoxine 0.25 mg/100 g, or a diet high in thiamin, riboflavin, or pyridoxine with 25 times the amount of vitamin. The effect of maternal vitamin intake on vitamin requirements was tested by comparing the rate of depletion of the young of control females, and by growth response of the depleted young to graded levels of the vitamin given in excess of the maternal diet. It was concluded that high intakes of the vitamins had no effect during gestation or lactation or in the young.

Scrimshaw, N. S. 1950. Evaluation of nutrition in pregnancy. J. Amer. Diet. Ass. 26:21-24.

Determining nutritional status in pregnant women from dietary history must be done cautiously because pathological requirements may be unrecognized; optimum requirements for some nutrients are unknown; requirement of one nutrient related to intake of other nutrients and individual variation may alter effects of diet. The influence of nutritional status in an individual is the sum of environmental factors, including nutrition acting on genetic makeup.

Scrimshaw, N. S. 1963. Factors influencing protein requirements. Harvey Lect. 58:181-216.

Protein requirements are discussed, and it is suggested that allowances should be set which ensure that all normal metabolic requirements are met and tissues are repleted for times of stress. The nutritional stress of repeated pregnancies and lactation is discussed.

Seifrit, E. 1968. Changes in beliefs and food practices in pregnancy. p. 79-90. In L. J. Roberts, Award essays, Amer. Diet. Ass., Chicago.

Beliefs and practices from earliest recorded history to 1960 are reviewed.

Sveringhaus, E. L. 1957. Pregnancy and nutrition. Bull. Margaret Hague Matern. Hosp. 10:15-27.

Literature was reviewed in an attempt to determine which nutrients are important in pregnancy, why they are important, when they are critical, and how much of each nutrient is optional for reproductive efficiency.

Slobody, L. B., R. A. Benson, and J. Mestern. 1947. A comparison of vitamin C in mothers and their premature newborn infants. J. Pediat. 31:333-337.

In a study of 30 premature infants and their mothers, it was found that the average intradermal test time for the prematures was 4.9 minutes and for their mothers 9.1 minutes. In a previous study of 77 mothers and their normal newborn, the average time for full-term newborns was 5.0 minutes. The premature and the full-term newborn have a good vitamin C level at birth even when the mother is depleted.

Smith, C. A. 1947. Effects of maternal undernutrition upon the newborn infant in Holland (1944-1945). J. Pediat. 30: 229-243.

During 1944-1945 in Holland, general undernutrition was severe enough to interfere with the prenatal growth of infants, as shown by fetal weight and, less definitely but significantly, by fetal length. Menstruation ceased in about 50 percent of urban women and became highly irregular in almost 50 percent of the others. An

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inadequate maternal diet before the sixth month of pregnancy was not associated with reduced birth weight. When maternal calories were reduced to one half the normal consumption in the last trimester, infants were about 8 ounces lighter than normal, but prematurity did not account for the decreased weight.

Smith, C. A. 1947. The effect of wartime starvation in Holland upon pregnancy and its product. Amer. J. Obstet Gynecol. 53:599-608.

A period of generalized undernutrition for 6 or 7 months in urban areas preceded the liberation of northwestern Holland in 1945. During this period, 50 percent of the women became amenorrheic. There was a reduction in the incidence of toxemia, and birth weight of infants decreased. No significant changes in prematurity, stillbirth, neonatal mortality, or percentage of mothers' breast feeding were found. It was noted that the populace involved in the study had been reasonably well fed before the period of undernutrition.

Smith, C. A. 1962. Prenatal and neonatal nutrition. Pediatrics 30:145-156.

Review article.

Sontag, L. W. 1941. Significance of fetal environmental differences. Amer. J. Obstet. Gynecol. 42:996-1003.

X-ray studies of a group of newborn infants revealed that the severity of round bone scars resulting from the birth process was much greater in children born of mothers whose diets were defective.

Sontag, L. W., and L. M. Harris. 1938. Evidences of disturbed prenatal and neonatal growth in bones of infants aged one month. II. Contributing factors. Amer. J. Dis. Child. 56:1248-1255.

A close relationship was found between the torsal striae noticed in roentgenograms and the diet and condition of the mothers during pregnancy.

Sontag, L. W., S. I. Pyle, and J. Cape. 1935. Prenatal conditions and the status of infants at birth. Amer. J. Dis. Child. 50:337-342.

In a study completed during the depression, no relation was found between prenatal nutritional factors and fetal growth.

Sontag, L. W., and J. Wines. 1947. Relationship of mothers' diets to status of their infants at birth and in infancy. Amer. J. Obstet. Gynecol. 54:994-1103.

In a study of the diet histories of 203 pregnant women, no relationship was found between weight, height, and ossification of infants at birth and protein intake during pregnancy.

Speert, H., S. Graff, and A. M. Groff. 1951. Nutrition and premature labor. Amer. J. Obstet. Gynecol. 62:1009-1019.

No convincing evidence could be found in an analysis of the relevant literature (30 studies reviewed) for the prevailing view that nutritional inadequacy is a common cause of premature labor. Seventy mothers of liveborn premature infants and 67 control mothers of term infants were also studied, and no significant differences in intake of nutrients, as revealed by diet history, or in hematological and blood biochemical determinations between the two groups was found.

Stearns, G. 1958. Nutritional state of the mother prior to conception. J. Amer. Med. Ass. 168:1655-1659.

A review of evidence indicated that long-term poor nutrition of the mother is one of the most important causative factors in pregnancy wastage. The author concludes, "the best insurance for a healthy infant is a mother who is healthy and well nourished throughout her entire life, as well as during the period of pregnancy itself."

Stevens, H. A., and M. A. Ohlson. 1967. Nutritive value of the diets of medically indigent pregnant women. J. Amer. Diet. Ass. 50:290-296.

The mean values of dietary intake of 129 healthy, medically indigent, predominantly white, randomly selected pregnant

women were compared with Recommended Dietary Allowances (1958 edition). Values met or exceeded recommendations except for calcium and iron for all age groups and ascorbic acid for the youngest and oldest groups. Comparison with a study of a similar group 13 years earlier revealed higher intake of all nutrients except thiamin for the present group.

Stuart, H. C. 1945. Findings on examinations of newborn infants and infants during the neo-natal period which appear to have a relationship to the diets of their mothers during pregnancy. Fed. Proc. 4:271-281.

A relationship was found between the protein content of the mother's diet during pregnancy and the osseous development in infants at birth.

Swartwout, J. R., W. G. Unglaub, and R. C. Smith. 1960. Vitamin B_6, serum lipids and placental arteriolar lesions in human pregnancy: A preliminary report. Amer. J. Clin. Nutr. 8(4):434-443.

In a small group of pregnant women, those receiving a placebo had a higher xanthurenic acid output throughout pregnancy and more placental arteriolar changes than the women receiving 25 mg of pyridoxine hydrochloride daily.

Taggart, N. 1961. Food habits in pregnancy. Proc. Nutr. Soc. 20:35-40.

Differences in food habits between socioeconomic groups in Britain, spontaneous changes which occur during pregnancy, and the effect of diet education on food habits are discussed.

Taylor, R. D., and J. R. Swartwout. 1967. Biochemical survey of protein sufficiency during pregnancy in urban women. Obstet. Gynecol. 29:244-246.

Protein sufficiency was determined in three groups of pregnant women by urea nitrogen/creatinine ratios. Ratios varied directly with expenditures for food.

Teel, H. M., B. S. Burke, and R. Draper. 1938. Vitamin C in human pregnancy. I. Studies during pregnancy. Amer. J. Dis. Child. 56:1004-1010.

With a relatively constant intake of vitamin C, the amount of the vitamin in maternal blood plasma decreased markedly as pregnancy advanced, whether the dietary intake of vitamin C was optional, suboptional, or deficient. The average amount of vitamin C in maternal plasma at the time of delivery was only slightly more than one half that present during the first 28 weeks of pregnancy. The mean amount of the vitamin in plasma from cord blood was 2 to 4 or more times greater than that in maternal plasma taken at the time of delivery.

Theobold, G. W. 1966. Nutrition and pregnancy. Med. Times 94:474-484.

Evidence is reviewed on the relation of nutrition to animal and human reproduction. The author postulates that stillbirth, low birth weight, and neonatal death are largely attributable to imperfect nutrition of the mother or of her parents and grandparents rather than to the diet taken during pregnancy.

Thomson, A. M. 1956. The diagnosis of malnutrition in well-nourished communities. Amer. J. Clin. Nutr. 4:647-653.

The author notes that the nutritional status of a pregnant woman is the product of many years of nutritional experience.

Thomson, A. M. 1957. Technique and perspective in clinical and dietary studies of human pregnancy. Proc. Nutr. Soc. 16:45-51.

In populations where the average level of nutrition is reasonably high, correlations between food intake during pregnancy and outcome of pregnancy are elusive when survey methods are used. Difficulty of survey techniques and necessary perspectives are discussed.

Thomson, A. M. 1958. Diet in pregnancy: Dietary survey technique and the nutritive value of diets taken by primigravidae. Brit. J. Nutr. 12:446-461.

A home-weighed, 1-week dietary survey was completed by 489 primigravidas from all social classes during the

seventh month of pregnancy. Methods used and reliability of methods were discussed. The mean nutritive value of diets showed a downward gradient from highest to lowest social class. Within each social class variation in nutritive value of diets was also noted.

Thomson, A. M. 1959. Diet in pregnancy. Brit. J. Nutr. 13:509-525.

Detailed analysis of the nutritive values of the diets of 489 primigravidas during pregnancy revealed little or no association with duration of gestation, birth weight, antepartum hemorrhage, operative delivery, fetal malformation, perinatal mortality, or failure to breast feed. Pre-eclampsia was associated with a relatively raised intake of most nutrients and of calories. Diets of pregnant women can vary widely in quantity as well as quality without clinically obvious impairment of the reproductive process. Although importance of the diet in pregnancy is usually inconspicuous in the individual, this is not necessarily true in large population groups.

Thomson, A. M. 1959. Diet in pregnancy. II. Assessement of nutritive value of diets, especially in relation to differences between social classes. Brit. J. Nutr. 13:190-204.

Correlations between levels of calories and nutrients in the diets of Aberdeen primigravidas were calculated. Differences in the intakes of a given nutrient correlated with similar differences in other nutrients.

Thomson, A. M. 1965. Diet in pregnancy. Practitioner 194:743-750.

A concise review is presented of what is and is not known about the diet during pregnancy; based on studies of the physiology of pregnancy.

Thomson, A. M., and W. Z. Billewicz. 1961. Height, weight and food intake in man. Brit. J. Nutr. 15:241-252.

The relationship between height and weight and diet was investigated in 489 pregnant women. The intake of all nutrients increased with increased stature, and taller

and heavier women were, generally, from a higher social class than the shorter, lighter women. The caloric intakes of overweight and underweight women were similar.

Tompkins, W. T. 1949. The objective evidences of nutritional deficiencies in pregnancy. Proc. Ann. Conf. Milbank Mem. Fund p. 82-99.

By using pictorial material, it was demonstrated that observable tissue areas (tongue) react to the stress of pregnancy and that these clinical signs are alleviated by intensive nutritional supplementation.

Tompkins, W. T., R. Mitchell, and D. Wiehl. 1954. Maternal and newborn studies at Philadelphia Lying-In Hospital. Maternal studies: II. Prematurity and maternal nutrition. Proc. Ann. Conf. Milbank Mem. Fund 1954:25-61.

The influence of maternal nutrition on the incidence of premature births was evaluated in 1,570 prenatal patients. The incidence of premature birth for patients classified by their pregravid weight increased sharply for underweight patients and was low for obese patients. Patients receiving both protein and vitamin supplements had the lowest incidence of premature births. A significant association was noted between gain in weight in the first and second trimesters and rate of prematurity.

Toverud, G. 1938. Preventive dentistry in the pre-school period and particularly during foetal life. Dent. Mag. Oral Top. 55:299-310.

Calcification of teeth before birth is determined by nutritional status and general condition of the mother before and during pregnancy. Not only the temporary teeth but also subsequent formation of tooth substance is affected.

Toverud, G. 1950. The influence of nutrition on the course of pregnancy. Milbank Mem. Fund Quart. 28:7-24.

A report of 6 years of experience involving over 1,000 pregnant women in the Sagene Health Station in Oslo (1939-1944) showed a decrease in the incidence of anemia, of hypocalcemia, of low serum ascorbic acid levels, and of low vitamin B excretion. There was no eclampsia, and stillbirth, prematurity, and neonatal death rates declined

sharply. These favorable outcomes were in contrast to the poorer outcomes found in the population of the city of Oslo. The good results were attributed to prenatal medical supervision, careful monitoring of the diets of the women, and nutrient supplements of iron, vitamins A and D, brewer's yeast, and vitamin K in late pregnancy. The use of refined carbohydrates, sugar, and sweets was kept as low as possible.

Toverud, K. U. 1933. Nutritional condition of new-born infants. Amer. J. Dis. Child. 46:954-962.

The average birth weight was 3,587 g for full-term infants born to mothers who had received a good diet and resided in a maternity home for 2 months or more before delivery as compared with an average birth weight of 3,347 g for mothers who had resided in the home for 6 weeks or less before delivery.

Turner, E. R., and M. S. Reynolds. 1955. Intake and elimination of vitamin B_6 and metabolites by women. II. Pregnant women. J. Amer. Diet. Ass. 31:1119-1120.

The mean daily intake of vitamin B_6 during pregnancy of four women during 5 days each month of the last trimester of pregnancy was 1.39 mg. The total urinary excretion of B_6 and 4-pyridoxic acid was 2 to 4 times the intake; over 90 percent of the total was 4-pyridoxic acid.

Venkatachalam, P. S. 1962. Maternal nutritional status and its effect on the newborn. WHO Bull. 26:193-201.

The low birth weight, high incidence of prematurity, and high fetal and infant mortality among the poor in India may be partially attributable to the poor nutritional status of pregnant women. The infants who survive probably possess little or no nutritional reserves and become early candidates for development of nutritional deficiency diseases. The unanswered question is how, in spite of poor nutritional status, a large number of these women are able to deliver normal babies.

Venkatachalam, P. S., B. Belavady, and C. Gopalan. 1962. Studies on vitamin A nutritional status of mothers and infants in poor communities of India. J. Pediat. 61:262-268.

Vitamin A and carotene content in serum of pregnant women, neonates, and lactating women and vitamin A content of breast milk were estimated in a poor Indian community. The effect of supplements was discussed.

Wachstein, M. 1956. Evidence for abnormal vitamin B_6 metabolism in pregnancy and various disease states. Amer. J. Clin. Nutr. 4:369-385.

The author suggests that routine supplementation of vitamin B_6 in pregnancy may be desirable.

Wideman, G. L., G. H. Baird, and O. T. Bolding. 1964. Ascorbic acid deficiency and premature rupture of fetal membrane. Amer. J. Obstet. Gynecol. 88:592-594.

In 288 white and nonwhite pregnant women there appeared to be a relationship between the adequacy of ascorbic acid intake, measured by plasma levels, and premature rupture of the membranes. The authors raise several questions.

Wiehl, D. G., and W. T. Tompkins. 1954. Maternal and newborn nutrition studies at Philadelphia Lying-In Hospital; maternal studies: I. Method of study and description of sample. Proc. Ann. Conf. Milbank Mem. Fund pp. 1-24.

It is hypothesized that high levels of intake of proteins, of vitamins, or of both would prevent toxemia and other symptoms and result in superior infants. Population, research design, composition of supplements, and usual dietary habits are described.

Williams, P. F., and F. G. Fralin. 1942. Nutrition study in pregnancy. Dietary analyses of seven-day food intake records of 514 pregnant women, comparison of actual food intakes with variously stated requirements, and relationship of food intake to various obstetric factors. Amer. J. Obstet. Gynecol. 43:1-20.

In a study of 514 pregnant women, a positive relationship between dietary adequacy and the occurrence of toxemia, stillbirth, prematurity, or birth weight of the infants could not be found.

Wiltse, H. 1967. Hypercalcemia and mental retardation. Proc. Conf. Drugs and Poisons, Pub. Health Serv., Publ. No. 1791 p. 212-218.

The association of mental retardation with hypercalcemia was examined in the context of three clinical syndromes: vitamin D intoxication, idiopathic infantile hypercalcemia, and the syndrome of supravalvular aortic stenosis with hypercalcemia.

Wise, G. H., M. J. Caldwell, and J. S. Hughes. 1946. The effect of the prepartum diet of the cow on the vitamin A reserves of her newborn offspring. Science 103:616.

Vitamin A fed to cows late in gestation significantly increased the vitamin A content of the blood and livers of the newborn calves over values found in newborn of mothers on a regular pasture diet.

Wishik, S. M. 1959. Nutrition in pregnancy and lactation. Fed. Proc. 18(2):4-8.

Nutritional needs of pregnant and lactating women are discussed; the interrelationship between the mother and the fetus is stressed.

Folic Acid and Megaloblastic Anemia

Ainley, N. J. 1961. Megaloblastic anaemia of pregnancy and the puerperium. J. Obstet. Gynaecol. Brit. Commonw. 62:254-260.

Megaloblastic anemia was diagnosed in one in every 92 deliveries during a 4-year period in an industrial area in England. The highest incidence was between May and November, and iron deficiency was frequently present. This disease was associated with increasing age, parity, and twin pregnancies. Megaloblastic anemia responded well to folic acid, but vitamin B_{12} was found ineffective.

Alperin, J. B., H. T. Hutchinson, and W. C. Levin. 1966. Studies of folic acid requirements in megaloblastic anemia of pregnancy. Arch. Intern. Med. 117:681-688.

Results of a study of two pregnant women with megaloblastic anemia demonstrated that folic acid requirements are increased during pregnancy because of demands of the growing

fetus. It suggests that demands of pregnant women during the third trimester with folate deficiency may be 400 μg or more.

Baker, S. J., E. Jacob, K. T. Rajan, and S. P. Swaminathan. 1962. Vitamin-B_{12} deficiency in pregnancy and the puerperium. Brit. Med. J. i:1658-1661.

In a study of nine women in the obstetric service of a South Indian hospital, all mothers were anemic and had a megaloblastic bone marrow, while the babies had no anemia. Maternal serum vitamin B_{12} levels ranged from 20 to 90 μμg/ml (mean 49.7 μμg/ml), and levels in the babies from 100 to 560 μμg/ml (mean 220 μμg/ml). Breast-milk levels were similar to serum levels in the mothers. The accumulation of B_{12} in fetal liver reached a minimum of 20 to 25 μg at birth.

Ball, E. W., and C. Giles. 1964. Folic acid and vitamin B_{12} levels in pregnancy and their relation to megaloblastic anaemia. J. Clin. Pathol. 17:165-174.

In a study of 247 pregnant women, there was a significant fall in serum folic acid levels during pregnancy. In twin pregnancies, this fall was more pronounced. In pregnant women with megaloblastic anemia, vitamin B_{12} and folic acid levels are lower than in normal pregnancies.

Booth, K. 1967. Folic-acid-deficient megaloblastic anaemia associated with child-bearing in Papua. Med. J. Aust. 1:640-642.

In a study of 21 pregnant women with megaloblastic anemia, it was found that serum vitamin B_{12} levels were not depressed. Over one half of the women were found to have complicating diseases (e.g., hookworm, dysentery, or tuberculosis). A complicating disease was present in all cases that failed to respond to the administration of folic acid.

Bunting, W. L. 1966. Megaloblastic anemia of pregnancy. Ariz. Med. 23:837-842.

A review of the literature and report of a case study.

Butterworth, C. E., and C. W. Scott. 1964. Megaloblastic anemia. Gen. Pract. 30:134-141.

Discusses common causes and features of diagnosis and treatment of megaloblastic anemia.

Chanarin, I. 1967. Diagnosis of folate deficiency in pregnancy. Acta Obstet. Gynecol. Scand. 46:39-46.

Folate deficiency in pregnancy is discussed in terms of detection of megaloblastic hemopoiesis and interpretation of biochemical evidence.

Chanarin, I., B. B. Anderson, and D. L. Mollin. 1958. The absorption of folic acid. Brit. J. Haematol. 4:156-166.

Study suggests that there may be some impairment of folic acid absorption during apparently normal pregnancies.

Chanarin, I., and D. A. Davey. 1964. Acute megaloblastic arrest of haemopoiesis in pregnancy. Brit. J. Haematol. 10:314-319.

Four cases of megaloblastic anemia in pregnancy are described.

Chanarin, I., B. M. MacGibbon, W. J. O'Sullivan, and D. L. Mollin. 1959. Folic acid deficiency in pregnancy: The pathogenesis of megaloblastic anaemia of pregnancy. Lancet 2:634-639.

An intravenous dose of 15 μg/kg of folic acid cleared from the serum more rapidly in pregnant than in nonpregnant women. Rate of clearance increased as pregnancy advanced and was greater in patients with twin pregnancies. Suggests rapid clearance is due to fetal uptake of folic acid.

Chanarin, I., D. Rothman, S. Ardeman, and V. Berry. 1965. Some observations on the changes preceding the development of megaloblastic anaemia in pregnancy with particular reference to the neutrophil leucocytes. Brit. J. Haematol. 11:557-562.

Serial observations of blood count, serum "folate" estimation, urinary formiminoglutamic acid excretion, and

neutrophil lobe counts were made on a group of 154 normal pregnant women. No significant differences were noted between those women who developed megaloblastic anemia and those who did not. The difficulty of diagnosing megaloblastic anemia without adequate bone-marrow samples was emphasized.

Chanarin, I., D. Rothman, and V. Berry. 1965. Iron deficiency and its relation to folic-acid status in pregnancy: Results of a clinical trial. Brit. Med. J. 1:480-485.

Clinic patients in early pregnancy were randomly assigned to three supplementation groups: iron, iron and folic acid, and a placebo. The latter group showed the highest incidence of iron-deficiency anemia and the greatest proportion of megaloblastic changes. Authors conclude that iron deficiency produces additional stress on folate metabolism in pregnancy.

Chanarin, I., D. Rothman, J. Perry, and D. Stratfull. 1968. Normal dietary folate, iron, and protein intake, with particular reference to pregnancy. Brit. Med. J. 2:394-397.

Lactobacillus casei assay of 24-hour food samples (111) of home consumption showed a mean folate content of 160 μg of free folate, 676 μg of total folate, a mean iron content of 14.2 mg/day, and a mean protein of 69 g. Folate intake was correlated with red-cell folate levels.

Chanarin, I., D. Rothman, A. Ward, and J. Perry. 1968. Folate status and requirement in pregnancy. Brit. Med. J. 2:390-394.

The addition of 100 μg of folate to a normal diet elevated red-cell folate levels, which were maintained at higher levels after the 30th week of pregnancy. A folate supplement of 100 μg of pteroglutamic acid in pregnancy was suggested. Megaloblastic hemopoiesis was found in 13 percent of 105 pregnant women receiving iron and in 5 percent of those receiving iron and 100 μg of folate. Women with megaloblastic features late in pregnancy had significantly lower red-cell folate values early in pregnancy than those who remained normoblastic. Megaloblastic changes were found to be more frequent in the winter. No relation was found between red-cell folate levels and spontaneous abortion or birth weight.

Chisholm, M. 1966. A controlled clinical trial of prophylactic folic acid and iron in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:191-196.

Presentation of the results of a controlled therapeutic trial of 360 pregnant women beginning at the 28th week of pregnancy, designed to test the effectiveness of a large dose (5 mg) and a small dose (5 μg) of folic acid in maintaining a normal level of folic acid activity in the blood and to determine if folic acid was effective in raising the hemoglobin in the absence of megaloblastic erythropoiesis.

Chisholm, M., and A. A. Sharp. 1964. Formimino-glutamic acid excretion in anaemia of pregnancy. Brit. Med. J. 5421:1366-1369.

Study of 102 pregnant women to determine if formimino-glutamic acid (FIGLU) in urine could be used instead of bone-marrow biopsy to determine folic acid deficiency. The Figlu test was not a useful test to determine megaloblastic anemia or transitional erythropoiesis during pregnancy.

Coyle, C., and F. Geoghegan. 1962. The problem of anaemia in a Dublin maternity hospital. Proc. Roy. Soc. Med. 55:764-765.

Accidental hemorrhage occurred in 13 of 95 women with megaloblastic anemia of pregnancy, a rate of 14 percent as compared with a normal incidence of 2.5 percent. These investigators advocate a marrow biopsy in all cases of accidental hemorrhage.

Dawson, D. C. 1962. The bone marrow picture of folic acid deficiency in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 69:38-46.

One hundred and fifty-one marrow smears were reviewed to determine the pattern of changes attributable to folic acid deficiency in pregnant and postpartum patients.

Dawson, D. W. 1966. Microdoses of folic acid in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:44-48.

Twenty women attending an antenatal clinic at 28th week of pregnancy were given 105 mg of elemental iron and 150 μg of folic acid daily. Interval estimations of total serum folate activity, labile folate, hemoglobin and mean corpuscular hemoglobin concentration were completed and compared with those of pregnant women not taking supplementary folic acid and iron. Further trials starting earlier in pregnancy and using other methods of assessing folic acid deficiency are recommended.

Edelstein, T., K. Stevens, N. Baumslag, and J. Metz. 1968. Folic acid and vitamin B_{12} supplementation during pregnancy in a population subsisting on a suboptimal diet. J. Obstet. Gynaecol. Brit. Commonw. 75:133-137.

From the 28th week of pregnancy and for 8 weeks, pregnant Bantu women subsisting on a diet low in folate were given 5 mg of folic acid and 200 mg of iron, and 72 patients were given 50 μg of vitamin B_{12} in addition to 5 mg of folic acid and 200 mg of iron. Administration of folic acid resulted in improvement in the indices of folate nutrition, but mean hemoglobin and hematocrit values were not changed. Vitamin B_{12} administration did not affect the serum vitamin B_{12} level, hemoglobin concentration, and hematocrit value.

Edelstein, T., K. Stevens, B. Brandt, N. Baumslag, and J. Metz. 1966. Tests on folate and vitamin B_{12} nutrition during pregnancy and the puerperium in a population subsisting on a suboptimal diet. J. Obstet. Gynaecol. Brit. Commonw. 73:197-204.

Tests on folate and vitamin B_{12} nutrition were completed in 235 randomly selected pregnant Bantu women in an attempt to assess the incidence of megaloblastic anemia associated with pregnancy. Hemoglobin and hematocrit estimations were also performed to determine the relationship between folate and vitamin B_{12} deficiency and the incidence of anemia.

Edelstein, T., S. S. Zail, G. E. Faulding, and J. Metz. 1967. Iron, folate and vitamin B_{12} nutrition in Bantu patients with postpartum anaemia. S. Afr. Med. J. 41:300-303.

In a study of 100 patients, bone-marrow iron stores were found to be diminished in 90 patients, with the percentage saturation of transferrin less than 20 in 92 percent of the 90 patients. Morphologic evidence of megaloblastic change

in bone marrow was found in 78 patients, and this change was associated with deficiency of folate rather than with deficiency of vitamin B_{12}.

Fraser, J. L., and H. J. Watt. 1964. Megaloblastic anemia in pregnancy and the puerperium. Amer. J. Obstet. Gynecol. 89:532-534.

Seventeen cases of megaloblastic anemia of pregnancy are presented along with a discussion of the teratogenic effect of this dyscrasia. Administration of 5 mg of folic acid daily from the beginning of pregnancy to all pregnant women is recommended.

Gatenby, P. B. B., and E. W. Lillie. 1960. Clinical analysis of 100 cases of severe megaloblastic anaemia of pregnancy. Brit. Med. J. i(5206):1111-1114.

Incidence, clinical features, and obstetrical aspects of 100 cases of severe megaloblastic anemia in pregnancy and the puerperium are reported.

Giles, C. 1966. An account of 335 cases of megaloblastic anaemia of pregnancy and the puerperium. J. Clin. Pathol. 19:1-11.

The incidence of megaloblastic anemia in pregnancy and the puerperium in North Staffordshire has steadily declined as a result of prophylaxis with folic acid. Microscopic examination of marrow was the most reliable method of diagnosis, although serum folate estimations gave a 95 percent correlation with marrow findings. Factors operating in pathogenesis are depletion of folic acid stores, mainly because of the demands of the growing fetus and insufficient intake of folic acid due to poor diets and absorption defects.

Giles, C., and E. Shuttleworth. 1958. Megaloblastic anaemia of pregnancy and the puerperium. Lancet 2(7061):1341-1347.

In England, an incidence of megaloblastic anemia of 2.8 percent or one in 39 hospital confinements in pregnant women was found. All cases responded to folic acid. In 42 percent of the women with megaloblastic anemia, the diet was poor compared with 12 percent of cases of normoblastic anemia and 4 percent of healthy controls.

Goodall, H. B. 1961. Megaloblastic anaemia of pregnancy and the puerperium. Pathol. Microbiol. 24:682-686.

The corrected incidence of megaloblastic anemia at Dundee Royal Infirmary was found to be 1.6 percent. The buffy coat technique was used. Hypertensive toxemia was found in 23 percent of these cases, and 13 percent had edema or albuminuria or both.

Grossowicz, N., J. Aronovitch, M. Rachmilewitz, G. Izak, A. Sadovsky, and B. Bercovici. 1960. Folic and folinic acid in maternal and foetal blood. Brit. J. Haematol. 6:296-302.

Folic and folinic acid was measured in paired maternal and fetal blood samples obtained from 25 nonanemic women at delivery. Higher folic acid values were found in fetal blood than in the corresponding maternal blood. The difference was striking in folinic acid. These data presumably indicate great fetal demand for folic acid and its active metabolite.

Grzesiukowicz, H., R. F. Jennison, and A. H. Gowenlock. 1965. Enzymatic release of folate activity from the red cells in megaloblastic anaemia of pregnancy. J. Clin. Pathol. 18:599-604.

Subjects with megaloblastic anemia of pregnancy had a significantly reduced folate activity precursor content, and 14 subjects (58 percent) had significantly low plasma factor activity. Impaired activity of the plasma factor may be part of the etiology of megaloblastic anemia of pregnancy.

Hansen, H. A., and H. v. Klewesahl-Palm. 1963. Blood folic acid levels and clearance rate of injected folic acid in normal pregnancy and puerperium. Scand. J. Clin. Lab. Invest. 15(69):78-99.

The authors found no correlation between fetal growth and folic acid clearance rates.

Hibbard, B. M. 1964. The role of folic acid in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 71:529-542.

Defective folate metabolism was found in 10 percent of the pregnancies studied. The author notes that there is a constant relationship between folic acid deficiency and

387-735 O - 70 - 6

the occurrence of abruptio placentae, and prophylaxis with folic acid should be started early in pregnancy to prevent invisible damage to the placenta. True prophylaxis should begin before conception, particularly for high-risk women.

Hibbard, B. M. 1967. Defective folate metabolism in pathological conditions of pregnancy. Acta Obstet. Gynecol. Scand. 46:47-59.

Discussion of folates in relation to needs in pregnancy, causes and effects of defective folate metabolism, manifestation of defective folate metabolism, and megaloblastic anemia and its effect on course and outcome of pregnancy.

Hibbard, B. M., and E. D. Hibbard. 1963. Aetiological factors in abruptio placentae. Brit. Med. J. 5370:1430-1436.

In a study of the etiological factors in abruptio placentae, all except one of the 73 cases showed a positive FIGLU, and that exception had received folic acid.

Hibbard, B. M., and E. D. Hibbard. 1966. Recurrence of defective folate metabolism in successive pregnancies. J. Obstet. Gynaecol. Brit. Commonw. 73:428-430.

In a prospective investigation of 200 women who in previous pregnancies had shown a positive FIGLU, the authors found that in 146 cases in subsequent pregnancies the positive FIGLU recurred. It was suggested that inadequate dietary folate intake may not be the only causative factor.

Hibbard, B. M., and E. D. Hibbard. 1968. Folate metabolism and reproduction. Brit. Med. Bull. 24:10-14.

Assessment of folate status, requirements and sources, factors leading to defective folate metabolism, clinical manifestations, treatment, and prophylaxis are included in this discussion.

Hibbard, B. M., E. D. Hibbard, and T. N. A. Jeffcoate. 1965. Folic acid and reproduction. Acta Obstet. Gynecol. Scand. 44:375-400.

Defective folate intake and metabolism, as determined by FIGLU excretion test, was observed in pregnant women who showed an increased incidence of abruptio placentae, repeated abortion, and birth of infants with congenital malformations. Folic acid deficiency is more likely to be found in women of high parity and those who have multiple pregnancies, in whom the demand for folic acid exceeds intake.

Hibbard, B. M., and T. N. A. Jeffcoate. 1966. Abruptio placentae. Obstet. Gynecol. 27:155-167.

Analyses of 506 cases of abruptio placentae in Liverpool. Etiology and management are discussed, and results are reported in relation to maternal and fetal condition.

Hibbard, E. D. 1964. The FIGLU-excretion test and defective folic-acid metabolism in pregnancy. Lancet 2:1146-1149.

The urinary excretion of FIGLU and end product of folic acid metabolism was found to be of great value during pregnancy in diagnosing the deficient state.

Hibbard, E. D., and R. W. Smithells. 1965. Folic acid metabolism and human embryopathy. Lancet i:1254.

Women (98) who had babies with severe malformations were matched by age, parity, and time of conception and gestation controls. The FIGLU test was made as soon as the malformation was detected before birth, or within 3 days of birth, to determine if a defect in folate metabolism or absorption may be the cause of central nervous system malformations. The women with positive FIGLU's had a higher incidence of malformed infants than women with negative FIGLU's.

Hourihane, B., C. V. Coyle, and M. I. Drury. 1960. Megaloblastic anaemia and pregnancy. J. Irish Med. Ass. 47:1-6.

In Dublin Maternity Hospital, routine investigation of anemia revealed an incidence of megaloblastic anemia of 4.2 percent (95 cases in 2,226 deliveries). A high incidence of twins and accidental hemorrhage was found in this group when compared with controls. The pathogenesis of megaloblastic anemia is discussed.

Husain, O. A. N., D. Rothman, and L. Ellis. 1963. Folic acid deficiency in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 70:821-827.

In an investigation of 168 pregnancies in the last trimester, 2 to 3 percent true megaloblastic anemias were found and about 4 percent probable folic acid deficiencies. A further 9 percent gave a positive FIGLU test around term, and as many gave a doubtful test suggesting a possible terminal folic acid deficiency.

Izak, G., M. Rachmilewitz, A. Sadovsky, B. Bercovici, J. Aronovitch, and N. Grossowicz. 1961. Folic acid metabolites in whole blood and serum in anemia of pregnancy. Amer. J. Clin. Nutr. 9:473-477.

In 64 pregnant women, low folic acid values were frequently associated with low serum vitamin B_{12} and low serum iron concentration. The mechanism of development of folic acid deficiency is discussed.

Johnson, E. M. 1964. Effects of maternal folic acid deficiency on cytologic phenomena in the rat embryo. Anat. Rec. 149:49-55.

A purified diet deficient in folic acid was fed to rats during eighth and ninth days of pregnancy. Embryos were removed on the tenth day and compared with those of normal controls. Mitotic activity was depressed, and a marked reduction in RNA was found in the deficient embryos.

Karthigaini, S., D. Gnanasundaram, and S. J. Baker. 1964. Megaloblastic erythropoiesis and serum vitamin B_{12} and folic acid levels in pregnancy in South Indian women. J. Obstet. Gynaecol. Brit. Commonw. 71:115-122.

The incidence of megaloblastic erythropoiesis, serum vitamin B_{12}, and folic acid levels in the third trimester of pregnancy and its effects on the course and outcome of pregnancy were studied in 50 less-privileged South Indian women.

Kershaw, P. W., and R. H. Girdwood. 1964. Some investigations of folic-acid deficiency. Scot. Med. J. 9:201-212.

In an attempt to compare two methods of investigation, Lactobacillus casei level and FIGLU excretion level, 11 pregnant women with megaloblastic anemia and 36 other pregnant women were included in the study population. No significant correlation was found between the two

measurements. In pregnant women with megaloblastic anemia, the FIGLU excretion test is of dubious value and may be misleading.

Kitay, D. Z., and J. S. Marshall. 1968. Remission of folic acid deficiency in pregnancy. Amer. J. Obstet. Gynecol. 102:297-303.

The presentation of a case of severe folate deficiency was used to demonstrate the range of abnormal laboratory findings associated with the disease and the time required for remission of biochemical and morphological abnormalities during specific therapy.

Laurence, C., and F. A. Klipstein. 1967. Megaloblastic anemia of pregnancy in New York City. Ann. Intern. Med. 66:25-34.

A study of 24 patients with megaloblastic anemia, 50 nonanemic controls in ninth month of gestation with hematocrit readings greater than 32 percent and taking iron medication, and 50 healthy nonpregnant female controls receiving no medication. Normal blood counts early in pregnancy did not preclude subsequent development of megaloblastic anemia. Folate deficiency was responsible in the majority of cases for megaloblastic anemia, and deficiency of vitamin B_{12} probably was not a factor. On the basis of this study, the routine administration of a folic acid supplement to all pregnant women in the last trimester is recommended.

Lowenstein, L., L. Brunton, and Y. S. Hsieh. 1966. Nutritional anemia and megaloblastosis in pregnancy. Can. Med. Ass. J. 94:636-645.

The incidence of megaloblastic changes in bone marrow of anemic women attending a public clinic in Montreal was 25 percent. Serum folate values of 4 ng/ml or less were indicative of folate deficiency in pregnancy and may precede development of bone-marrow changes. Mild to moderate folate deficiency developed commonly in the population that was studied. Greek women whose diets were rich in protein had a lower incidence of megaloblastic anemia.

Lowenstein, L., G. Cantlie, O. Ramos, and L. Brunton. 1966. The incidence and prevention of folate deficiency in a pregnant clinic population. Can. Med. Ass. J. 95:797-806.

A study of 311 nonanemic pregnant women. Each woman was given a daily multivitamin tablet containing 78 mg of elemental iron, and one group also received supplementary 0.5 mg of folic acid and 0.005 mg of vitamin B_{12}. The incidence of megaloblastic bone-marrow change in the unsupplemented group was 26 percent; it was sharply reduced in the supplemented group. The incidence of low folate levels and megaloblastosis suggests insufficient intake, excessive requirements, or altered folate metabolism in pregnancy.

Lowenstein, L., C. Pick, and N. Philpott. 1955. Megaloblastic anemia of pregnancy and the puerperium. Amer. J. Obstet. Gynecol. 70:1309-1337.

Clinical and hematological studies on 19 cases of megaloblastic anemia of pregnancy and the puerperium are reported.

Martin, J. D., and R. E. Davis. 1964. Serum folic acid activity and vaginal bleeding in early pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 71:400-403.

In a study of serum folate activity in 19 threatened and 35 inevitable abortions, higher levels of activity were found when vaginal bleeding terminated in abortion, suggesting that folic acid demands were reduced following abortion.

Martin, R. H., T. A. Harper, and W. Kelso. 1965. Serum-folic-acid in recurrent abortions. Lancet 1:670-672.

The results of this study indicated an association between spontaneous isolated abortion of unknown etiology and a deficiency of folic acid and its derivatives.

Matoth, Y., A. Pinkas, and C. Skroka. 1965. Studies on folic acid in infancy. III. Folates in breast fed infants and their mothers. Amer. J. Clin. Nutr. 16:356-359.

Total folate activity in breast-fed infants was significantly higher than in a comparable group of artificially fed infants. The mothers of the breast-fed infants had lower folate levels than their infants or normal adults.

Menon, M. K., M. Sengupta, and N. Ramaswamy. 1966. Accidental haemorrhage and folic acid deficiency. J. Obstet. Gynaecol. Brit. Commonw. 73:49-52.

In retrospective and prospective studies, pregnant women with megaloblastic anemia or folic acid deficiency are not more prone to accidental hemorrhage.

Perry, J., and I. Chanarian. 1968. Absorption and utilization of polyglutamyl forms of folate in man. Brit. Med. J. 4:546-549.

A study indicating that in man the polyglutamate forms of folate are absorbed and utilized to about one third the extent of monoglutamate forms.

Pritchard, J. A., R. A. Mason, and M. R. Wright. 1962. Megaloblastic anemia during pregnancy and the puerperium. Amer. J. Obstet. Gynecol. 83:1004-1018.

Studies of 20 cases of megaloblastic anemia during pregnancy or the puerperium are presented. Hematological changes in order of appearance are described.

Solomons, E., S. L. Lee, M. Wasserman, and J. Malkin. 1962. Association of anaemia in pregnancy and folic acid deficiency. J. Obstet. Gynaecol. Brit. Commonw. 69:724-728.

The relationship between anemia megaloblastosis and folic acid status was determined by serum folic acid level in a population of 81 pregnant women of low economic status.

Stone, M. L., A. L. Luhby, R. Feldman, M. Gordon, and J. M. Cooperman. 1967. Folic acid metabolism in pregnancy. Amer. J. Obstet. Gynecol. 99:638-648.

The authors studied blood and marrow morphology, urinary FIGLU excretion, fasting serum, and whole blood L. casei folic acid activity in normal and complicated pregnancies and nonpregnant controls. Results indicate that significant folic acid deficiency may be present without overt megaloblastic anemia; the incidence of deficiency was 22 percent, tended to increase near term, and was markedly increased in pregnancy complications, particularly toxemia. Abnormal folic acid metabolism, the hazards for mother and infant,

and the role of nutritional deficiency as an etiological factor are discussed.

Streiff, R. R., and A. B. Little. 1967. Folic acid deficiency in pregnancy. New Engl. J. Med. 276:776-779.

Women at Boston City Hospital with third trimester bleeding and abruptio placentae showed a 44 and 94 percent incidence of folate deficiency, respectively, as compared with 18 percent in women at term in normal pregnancy, and 15 and 16 percent in the second and third trimesters. Nonpregnant women chosen at random as controls showed a 5 percent incidence of folate deficiency. Disturbance of appetite and assimilation, metabolic demands of pregnancy, and urinary tract infections favor development of folate deficiency. Dietary management in pregnancy should take into account the increased need for folic acid.

Thamber, J., and D. Llewellyn Jones. 1966. Bone marrow studies in abruptio placentae. J. Obstet. Gynaecol. Brit. Commonw. 73:930-933.

The state of bone marrow was studied in 112 consecutive patients with antepartum hemorrhage. The bone marrow smears did not show an increased incidence of megaloblastic erythropoiesis in patients with abruptio placentae.

Vanier, T. M., and J. F. Tyas. 1966. The effect of prophylactic folic acid on serum and whole blood levels during the last trimester of pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:934-939.

The effect of 90 to 150 mg prophylactic folic acid on serum and whole blood levels during the last trimester of pregnancy was studied in two groups of 27 normal pregnant women comparable in age and parity. The women receiving folic acid maintained a more favorable serum folate and whole blood folate level than did the control group.

Varadi, S., D. Abbott, and A. Elwis. 1966. Correlation of peripheral white cell and bone marrow changes with folate levels in pregnancy and their clinical significance. J. Clin. Pathol. 19:33-36.

Findings in 222 women with megaloblastic anemia of pregnancy are reviewed.

Willoughby, M. L. N. 1967. An investigation of folic acid requirements in pregnancy. II Brit. J. Haematol. 13:503-509.

From the findings in 3,599 pregnant women randomly allocated to one of five groups receiving iron or iron plus differing levels of folic acid per day, it was concluded that, in a population where dietary deficiency is common, 300 μg per day of folic acid is close to the minimal daily requirement late in pregnancy.

Willoughby, M. L. N., and F. G. Jewell. 1968. Folate status throughout pregnancy and in postpartum period. Brit. Med. J. 5627:356-360.

Serial trends of whole blood folate levels in two groups of women were followed throughout pregnancy and to 6 weeks postpartum. Half of those women who received iron alone were in the deficient range at 6 weeks postpartum. Those who received iron plus 330 μg of folic acid per day maintained whole blood folate levels in the normal range. The authors suggest that a supplement of 300 μg of folic acid per day will prevent deficiency in late pregnancy and the puerperium.

Willoughby, M. L. N., and F. J. Jewell. 1966. Investigation of folic acid requirements in pregnancy. Brit. Med. J. 2:1568-1571.

The incidence of megaloblastic anemia in pregnancy and the puerperium and serum folate levels in the puerperium in 350 pregnant patients divided into five groups according to iron and folic acid intakes. It was concluded that minimal oral requirements of folic acid in late pregnancy are about 300 μg a day.

Iron Status and Iron-Deficiency Anemia

Benjamin, F., F. A. Bassen, and L. M. Meyer. 1966. Serum levels of folic acid, vitamin B_{12}, and iron in anemia of pregnancy. Amer. J. Obstet. Gynecol. 96:310-315.

Among prenatal patients served by a large city hospital and a voluntary hospital in New York, 80 percent of the patients considered to be anemic had low serum iron, 65 percent had folic acid deficiency (serum folate bioassay), and 31 percent had vitamin B_{12} deficiency. In 66 percent of the anemias there was more than one deficiency, and the most common deficiency was associated with low levels of both iron and folic acide.

Benstead, N., and G. W. Theobold. 1952. Iron and the physiological anemia of pregnancy. Brit. Med. J. i:407-410.

In 2,000 consecutive antenatal patients less than 16 weeks pregnant, 59 percent had hemoglobin values below 12 g percent, with 6.5 percent below 11 g percent. An iron-supplemented group maintained nonpregnant values of hemoglobin and mean corpuscular hemoglobin concentration. Physiological anemia became evident in the nonsupplemented group.

Bethell, F. H., S. H. Gardiner, and F. MacKinnon. 1939. The influence of iron and diet on the blood in pregnancy. Ann. Intern. Med. 13:91-100.

The cause of "true anemia of pregnancy" may be impaired utilization of iron and low dietary levels of animal protein. The authors recommend 50 g of animal protein per day and iron supplementation in pregnancy.

Chopra, J. G., E. Noe, J. Matthew, C. Dheim, J. Rose, J. M. Cooperman, and A. L. Luhby. 1967. Anemia in pregnancy. Amer. J. Pub. Health. 57:857-868.

In Trinidad, detailed clinical, biochemical, and dietary data were collected simultaneously on 87 anemic pregnant women with hemoglobin levels less than 10 g/100 ml and 20 controls with hemoglobin levels greater than 12 g/100 ml. Types of anemia found: 77 percent of the cases were hypochromic microcytic, 20 percent were dimorphic, and 3 percent were macrocytic hyperchromic. On the basis of this study, it was concluded that the routine administration of iron in therapeutic quantities is justifiable as an initial measure in areas where microcytic anemia is prevalent and where routine hematological examinations are

not undertaken. Because the majority of anemias of pregnancy are the iron-deficient type, it is uneconomical to administer folic acid and vitamin B_{12} routinely to all pregnant women

Committee on Nutrition. 1969. Iron balance and requirements in infancy. Pediatrics 43(1):134-142.

Memorandum reviews current knowledge of iron needs of infants, attempts to quantitate iron requirements in relation to birth weight and age, and examines methods for assuring adequate iron intake. Factors affecting iron endowment of the newborn are discussed.

Council on Foods and Nutrition. 1968. Iron deficiency in the United States. J. Amer. Med. Ass. 203:407-412.

Includes a discussion of iron requirements and iron-deficiency anemia in pregnancy. Suggests prophylactic iron administration of 30 mg/day in the latter half of pregnancy.

Davis, L. R., and R. F. Jennison. 1954. Response of the physiological anaemia of pregnancy to iron therapy. J. Obstet. Gynaecol. Brit. Emp. 61:103-108.

A discussion of the effect of iron sulfate supplements on packed cell volume, hemoglobin levels, and mean corpuscular hemoglobin in approximately the 32nd week of pregnancy. Authors conclude that "physiological anemia" is the result of iron deficiency.

Demulder, R. 1958. Iron. Arch. Intern. Med. 102:254-301.

A review of literature on iron metabolism, biochemistry, and clinical pathological physiology.

Edgar, W., and H. M. Rice. 1956. Administration of iron in antenatal clinics. Lancet 1:599-602.

Ferrous sulfate (315 mg of elemental iron daily) maintained hemoglobin levels and other hematological indices during pregnancy in 77.6 percent of 89 women studied. A tendency toward iron deficiency was noted in pregnant women not receiving ferrous sulfate.

Elwood, P. C. 1968. Some epidemiological problems of iron deficiency anaemia. Proc. Nutr. Soc. 27:14-23.

Discusses iron-deficiency anemia in terms of prevalence in a community, the amount of morbidity or mortality caused by the condition, and prevention.

Evers, J. E. M. 1966. Premature birth and iron deficiency. Nederl. T. Geneesk. 110:2244-2247.

Mean duration of pregnancy and incidence of obstetrical complication were compared in primigravidas and multigravidas with a total iron-binding capacity of both less than and greater than 500 μg/100 ml.

Fathalla, M. F., and I. Kamal. 1964. Haemoglobin levels before and after parturition. J. Egypt. Med. Ass. 47:373-381.

The total average loss of hemoglobin during parturition and 6 days after delivery was 64.5 g, which represents an average blood loss of 588 cc and an average iron loss of 215 mg.

Finch, C. A. 1965. Iron balance in man. Nutr. Rev. 23:129-131.

A discussion of iron that includes iron losses and growth needs, absorption, and requirements in pregnancy.

Fisher, M., and R. Biggs. 1955. Iron deficiency in pregnancy. Brit. Med. J. 1:385-386.

Confirms earlier work that suggested that physiological anemia of pregnancy can be prevented by routine administration of iron.

Gatenby, P. B. 1963. Anaemias in pregnancy. Curr. Med. Drugs 4:12-18.

Many studies indicate that iron-deficiency anemia is common in pregnancy, and routine oral iron treatment is justifiable and probably necessary for most pregnant women. The use of folic acid to prevent megaloblastic anemia is more controversial. The argument that folic acid may mask

true pernicious anemia is not a strong one, because pernicious anemia is rare in women of childbearing age, and patients with pernicious anemia are usually sterile. There is general agreement that twin pregnancies greatly increase liability to megaloblastic anemia, and therefore the routine use of folic acid in this case is recommended.

Gatenby, P. B., and E. W. Lillie. 1955. Iron-deficiency anaemia in pregnancy. Lancet 268:740-743.

Case history data indicated that a deficiency of iron in the diet is the most important cause of iron-deficiency anemia.

Giles, C., and H. Burton. 1960. Observations on prevention and diagnosis of anaemia in pregnancy. Brit. Med. J. 2:636-637.

Hemoglobin levels, at term, of 758 women treated daily with both 15 mg of folic acid and 540 mg of ferrous sulfate during the last trimester of pregnancy are reported and compared with 721 controls treated with ferrous sulfate alone. The mean hemoglobins were higher in those receiving both folic acid and iron.

Girdwood, R. H. 1966. The anaemias of pregnancy. Curr. Med. Drugs 6:20-30.

Anemia is common during pregnancy and is usually due to iron deficiency. There is good evidence to support the administration of iron and folic acid during pregnancy. The divergence of opinion with regard to the value of FIGLU estimations to determine folic acid depletion during pregnancy is discussed.

Hallberg, L., A. Hogdahl, L. Nilsson and G. Rybo. 1966. Menstrual blood loss--a population study. Acta Obstet. Gynecol. Scand. 45:320-351.

In a study of nearly 500 women, 15 to 50 years of age, the mean value of menstrual blood loss was determined.

Halsted, J. A. 1968. Geophagia in man: Its nature and nutritional effects. Amer. J. Clin. Nutr. 21:1384-1393.

A review of geophagia in terms of its nature, incidence, history in medicine, and clinical effects. Iron deficiency is discussed with relation to geophagia.

Hankin, M. E. 1963. The value of iron supplementation during pregnancy. Aust. N.Z. J. Obstet. Gynaecol. 3:111-118.

In a study of 174 healthy primigravidas and secundigravidas on adequate diets, hemoglobin and hematocrit levels were found to be unrelated to dietary iron intake. The value of iron supplementation on these values could not be demonstrated, although the supplemented group (118 women) had significantly higher hemoglobin values in the third trimester and on the fifth day after delivery. Maternal supplementation had no effect on cord hemoglobin levels and those of offspring at 6 weeks, 3 months, and 6 months postpartum.

Henderson, P. A. 1964. Anemia of pregnancy. Obstet. Gynecol. 24:752-757.

Among 168 pregnant women with anemia, iron deficiency was found in only 57 percent. The usual diagnostic aids available to the average clinician may be insufficient to make definitive diagnoses in many instances.

Holly, R. G. 1959. The practical aspects of anemia therapy in pregnancy. Postgrad. Med. 26:418-424.

Discussion of iron-deficiency anemia in pregnancy, its prevention and therapy, with a review of iron metabolism.

Holly, R. G. 1960. Anemia in pregnancy. Amer. J. Obstet. Gynecol. 79:401-402.

In a discussion of hematopoiesis and pregnancy, the author suggests that 10 g of hemoglobin and a corresponding 33 percent hematocrit are too low to accept as normal and minimum standards in pregnancy.

Holly, R. G. 1960. Anemias of pregnancy. Clin. Obstet. Gynecol. 3:921-927.

A review of normal fetal and maternal hematopoiesis and folic acid, vitamin B_{12}, and iron requirements by the maternal organism and developing fetus.

Holly, R. G. 1965. Dynamics of iron metabolism in pregnancy. Amer. J. Obstet. Gynecol. 93:370-375.

A normal adult woman has 3.5 to 4.0 g of body iron, excretes 0.5 to 1.5 mg daily in feces, urine, and sweat, and loses an equivalent amount in normal menstruation. The intake of dietary iron is usually not adequate to compensate for these normal losses; consequently, the majority of women are deficient in iron when they enter pregnancy and because of increased iron demands are prone to develop iron-deficiency anemia during pregnancy.

Holly, R. G., and W. J. Grund. 1959. Ferrodynamics during pregnancy. Amer. J. Obstet. Gynecol. 77:731-742.

Storage iron estimates made on pregnant women between the 8th and 14th weeks of gestation revealed reduced storage iron deposits in the majority of women. Radioiron studies indicated a decrease in plasma iron and red-cell iron turnover rate during pregnancy. Physiological anemia, utilization of storage iron, and iron from food and supplements are discussed.

Hunter, C. A., Jr. 1960. Iron-deficiency anemia in pregnancy. Surg. Gynecol. Obstet. 110:210.

Hemoglobin level determinations were completed on 4,744 pregnant women. Hemoglobin values in 852 of the women were 9 to 11 g/100 ml, and 89 had hemoglobin values less than 9 g/100 ml.

Hytten, F. E., and D. L. Duncan. 1956. Iron deficiency anaemia in the pregnant woman and its relation to normal physiological changes. Nutr. Abstr. Rev. 26:855-868.

Paper stresses individual variation in hemoglobin concentration and questions the advisability of raising the hemoglobin level in all women during a normal pregnancy.

Ibbotson, R. N., and B. A. Crompton. 1964. Red cell mass in anaemia during pregnancy. Aust. Ann. Med. 13:139-142.

A study of 32 pregnant females with hemoglobin values less than 11 g/100 ml showed that the correlation of

hematocrit with red-cell mass calculated in terms of surface area was better than when based on either body weight or ideal weight.

Johnson, F. A., T. J. McMillan, G. E. Falconer, and E. Evans. 1952. Iron requirement of six young women. J. Amer. Diet. Ass. 28:633-635.

College women in good health, 20 to 31 years of age, were studied. The mean loss of iron for four menstrual periods ranged from 4.86 to 13.65 mg, and the amount of iron needed for replacement ranged from 0.21 to 0.49 mg. On a mean intake of 11.52 mg during 8 weeks of study, the six subjects retained sufficient iron to cover all their needs.

Johnson, J. W. C., and O. A. Ojo. 1967. Amniotic fluid oxygen tensions in severe maternal anemia. Amer. J. Obstet. Gynecol. 97:449-506.

A study of 14 anemic (hematocrit less than 30 percent) and 14 nonanemic women, during 20 to 32 weeks of pregnancy, in Nigeria. A highly significant positive correlation between maternal hematocrit values and oxygen tensions of amniotic fluid was found. There was a high fetal mortality rate in markedly anemic women.

Jones, O. P. 1950. The passage of anti-anemic substances across the placenta, and their influence on embryonic hematopoiesis. III. Effects of an iron-rich diet. Rev. Hematol. 5:618.

A maternal diet containing 1 percent ferric sulfate results in morphological changes in the fetal primitive erythroblasts.

Kessel, I., and D. J. Sills. 1968. Neonatal and maternal serum iron levels at birth. J. Obstet. Gynaecol. Brit. Commonw. 75:752-753.

The serum iron levels were measured in mothers and their newborn, full-term infants at the time of birth. No correlation was found between the maternal and neonatal levels, and high cord-blood serum iron levels bore no relation to sex or birth weight.

Kirksey, A., and M. H. Tabacchi. 1967. Pyridoxine deficiency and iron metabolism in the pregnant rat: Maternal responses. J. Nutr. 93:229-240.

The effects of pyridoxine deficiency on the normal increase in iron absorption in the pregnant rat and on the use of a large dose of iron administered orally during gestation were investigated.

Klein, L. 1962. Premature birth and maternal prenatal anemia. Amer. J. Obstet. Gynecol. 83:588-590.

The incidence of premature births associated with a maternal prenatal hemoglobin level below 10 g percent was 12.6 percent, and 7.2 percent when maternal prenatal hemoglobin was 10 g percent or above.

Land, C. J., and T. R. C. Sisson. 1958. Blood volume and anemia of mother and baby. Amer. J. Obstet. Gynecol. 76:1013-1023.

Serial determinations of 130 women studied during one or more pregnancies and the puerperium indicated a rapid increase in plasma volume to a mean of 60.2 ml/kg in early pregnancy to a mean of 68.4 ml/kg in late pregnancy. Nonpregnant controls had a mean value of 52.1 ml/kg. The mean values of circulating hemoglobin mass and total red-cell volume in eight nonanemic women in late pregnancy were 12.3 g/kg and 33.9 ml/kg, respectively.

Lanzkowsky, P. 1961. The influence of maternal iron-deficiency anaemia of the haemoglobin of the infant. Arch. Dis. Child. 36:205-209.

A survey that investigates whether maternal iron-deficiency anemia influences the level of hemoglobin in infants. Infants of women with a mean corpuscular hemoglobin concentration of 27 percent or less at term, and those with values of 31 percent or more, were examined. No significant difference was found in hemoglobin level in the two groups during the first 24 hours of life or at 3 months.

Lawrence, A. C. 1962. Iron status in pregnancy. J. Obstet. Gynecol. 69:29-37.

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Measurements of iron status, independent of hemoglobin concentrations, were made in pregnant women given iron therapy and in untreated pregnant subjects. Measurements of serum iron concentration and bone-marrow iron content were sensitive indicators of iron deficiency. Average mean corpuscular hemoglobin concentrations rose in pregnancy and were significantly higher in pregnant women than in nonpregnant women. Serum iron concentration greater than 75 μg/100 ml and an increase in bone-marrow iron were noted in women receiving iron therapy. Iron-deficient subjects showed lower hemoglobin concentration in late pregnancy and smaller average increase in red-cell volume than did iron-sufficient subjects.

Lourdenadin, S. 1964. Pattern of anaemia and its effect on pregnant women in Malaya. Med. J. Malaya 19:87-93.

A review of 1,066 cases of severe anemias of pregnancy (6.5 g and under) in the maternity hospital at Kuala Lumpur. Macrocytic anemia constituted 22 percent among the anemic mothers. The incidence of toxemia, premature labor, puerperal pyrexia, and perinatal and maternal mortality was higher than among controls.

Lowenstein, L., Y. S. Hsieh, L. Brunton, N. M. deLeeuw, and B. A. Cooper. 1962. Nutritional deficiency and anemia in pregnancy. Postgrad. Med. 31:72-78.

Administration of 78 mg of elemental iron daily by mouth prevented iron-deficiency anemia during pregnancy in 20 women studied. Iron deficiency was the primary cause of anemia in 23 of 26 pregnant patients who did not receive supplemental iron and who evidenced mild or moderate anemia and macrogranulocytic or megaloblastic changes in the bone marrow. A decrease in serum folic acid activity or the serum concentration of vitamin B_{12} or both was observed in all 26 patients.

Metz, J., T. Edelstein, M. Dovaris, and S. S. Zail. 1967. Effect of total dose infusion of iron-dextran on iron, folate, and vitamin B_{12} nutrition in postpartum anaemia. Brit. Med. J. 3:403-406.

Iron-dextran by total dose infusion was administered to 81 postpartum anemic subjects with diminished marrow iron

stores. The therapy corrected anemia, and tests of iron nutrition were normal. Authors recommend administration of folic acid to patients receiving iron-dextran.

Metz, J., L. Turchetti, B. Combrink, and S. Krawitz. 1966. Significance of tests of iron nutrition in pregnancy. J. Clin. Pathol. 19:173-176.

The authors concluded that pregnant women with normal marrow iron stores and transferrin saturation of 20 percent or more at 24 weeks of gestation are unlikely to develop iron-deficiency anemia during the remainder of pregnancy.

Monson, E. R., I. N. Kuhn, and C. A. Finch. 1967. Iron status of menstruating women. Amer. J. Clin. Nutr. 20:842-849.

The iron content of normal diets of 13 university women was calculated for 7 days from food composition tables and by chemical determination. The daily iron intake averaged 9.2 to 9.9 mg. Four of the subjects were considered normal (two of these were taking medicinal iron or iron supplemented food); three exhibited increased iron absorption; five exhibited iron deficiency. Decreases in caloric intake, increased cleanliness in commercial food handling, and declining use of iron cooking utensils were cited as causes of a gradual decrease in iron consumption in the population.

Morgan, E. H. 1961. Plasma iron and haemoglobin levels in pregnancy. Lancet 1:1-12.

Changes in blood hemoglobin level, mean corpuscular hemoglobin concentration, plasma iron, and total iron-binding capacity were studied in two groups of pregnant women. One group received iron as ferrous gluconate, and a comparable group received no iron supplement. Throughout pregnancy the iron-supplemented group showed reduced mean total iron-binding capacity and increased MCHC. No difference was noted in the two groups for plasma iron or blood hemoglobin levels until the last month of pregnancy, when both values were higher in the treated than in the untreated group.

Paintin, D. B., A. M. Thomson, and F. E. Hytten. 1966. Iron and haemoglobin level in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:181-190.

Responses to small and large doses of oral iron supplements were studied. Large doses prevented anemia in the few with iron deficiency but did not eliminate the need for hematological investigation in the last trimester. Large routine iron supplementation, while not harmful, is not necessary for most women in Great Britain. It is better to diagnose and treat iron deficiency specifically.

Patwardhan, V. N. 1963. Nutritional anemias in the tropics. J. Amer. Med. Women's Ass. 18:375-380.

In a review, the chief of the Nutrition Unit, World Health Organization, concludes that iron-deficiency anemia is the predominant type of anemia in developing countries and is widespread. From present evidence it is not possible to judge the prevalence of megaloblastic anemia, but is probably more common among pregnant women. Association of poor diets with nutritional megaloblastic anemia is discussed.

Patwardhan, V. N. 1966. Nutritional anemias--WHO research program; Early development and progress report of collaborative studies. Amer. J. Clin. Nutr. 19:63-71.

A review of the progress of collaborative investigations sponsored by WHO, with a description of studies to be completed on pregnant women in New Delhi, India; Vellore, South India; Johannesburg, South Africa; Jerusalem, Israel; and Warsaw, Poland.

Pirrie, R. 1950. Iron metabolism: A review of recent literature. Glasgow Med. J. 31:397-416.

Of 4.5 g of iron in the body, 60 percent occurs as combined iron in circulating hemoglobin, 23 percent as parenchymal iron, and the remainder as storage available for body requirements. Daily loss for the normal female is 2.1 mg, increasing to 3.8 mg during pregnancy and lactation. The daily minimal requirement for a female to maintain iron balance is 12 to 15 mg. Excretion data were reviewed.

Rachmilewitz, M., J. Nitzkin, S. Levy, A. Salomonowitz, N. Grossowicz, and G. Izak. 1966. Anemia of pregnancy in a rural community of upper Galilee. Israel J. Med. Sci. 2:472-479.

The prevalence of anemia was studied in 890 women during the second and third trimesters of pregnancy, and attention was given to county of origin, parity, and socioeconomic conditions. Hemoglobin levels below 10.0 g percent were found in 22.2 percent and were due mainly to combined deficiency, mainly iron and folic acid, and increased requirement of the fetus.

Robbe, H. 1958. Total amount of haemoglobin and physical working capacity in anaemia of pregnancy. Acta Obstet. Gynecol. Scand. 37:312-335.

In 19 healthy pregnant women with hemoglobin values below 32 percent, the absolute physical working capacity (such work as is limited by circulatory capacity) remained within relatively normal limits and approximately constant during and after pregnancy.

Rolson, J. R. 1964. Haemoglobin levels in apparently normal men, women and children in Tanganyika. J. Trop. Med. Hyg. 67:282-283.

A high incidence of low hemoglobin levels was found in pregnant women in Tanganyika.

Roszkowski, I., J. Wojcicka, and K. Zalewska. 1966. Low fetal body weight and the serum iron level in the mother and newborn. Pol. Med. J. 5:492-500.

A correlation was found between low serum iron in mother and low body weight of the newborn. The cause of the low serum iron level was found to be infection in the mother or other pathology.

Roszkowski, I., J. Wojcicka, and K. Zalewska. 1966. Serum iron deficiency during the third trimester of pregnancy: Maternal complications and fate of the neonate. Obstet. Gynecol. 28:820-825.

In a study of 486 women with serum iron values below 60 μg percent in the third trimester of pregnancy, there was a tendency for their newborn infants to be underweight and shorter than controls. Incidence of fetal heart rate alterations, severe asphyxia, stillbirths, neonatal deaths, and congenital abnormalities were compared with controls.

Scott, D. E., and J. A. Pritchard. 1967. Iron deficiency in healthy young college women. J. Amer. Med. Ass. 199:897-900.

A study was made of 114 healthy white privileged women. Iron stores were scant or absent in two thirds of the 22 on whom evaluations were made of marrow-stainable iron and quantitative phlebotomy. The mean values for hemoglobin, hematocrit, serum iron, and transferrin saturation were lower for women with no stainable iron in the bone marrow; however, values overlapped those in women with stainable iron. Serum iron and transferrin saturation were correlated with iron stores.

Sluglett, J. 1963. Hemoglobin levels in pregnancy. Coll. Gen. Pract. 6:66-70.

Hemoglobin estimations on 200 unselected pregnant women receiving iron and vitamin supplements at their first attendance in an antenatal clinic at the 32nd week and at the final postnatal examination suggest that hemodilution occurs near the 32nd week despite iron supplementation. Routine iron supplementation in pregnancy was suggested to ensure maintenance of hemoglobin levels above the physiological optimum.

Strauss, M. B. 1933. Anemia of infancy from maternal iron deficiency in pregnancy. J. Clin. Invest. 12:345-353.

The common form of anemia during infancy is due to deficient storage of iron in the fetus, a condition that results from deficient maternal supply.

Sturgeon, P. 1959. Studies of iron requirements in infants. III. Influence of supplemental iron during normal pregnancy on mother and infant. B. The infant. Brit. J. Haematol. 5:45-55.

Iron nutrition was studied in three groups of infants.

The mothers of the first group received no iron supplements during pregnancy, mothers of the second group received 0.5 g of ferrous sulfate, and mothers of the third group received 1,000 mg of saccharated iron oxide intravenously. No difference in the state of iron nutrition was found in the three groups of infants at birth or at 6, 12, and 18 months of age.

Vyas, R. B., V. R. Patel, V. G. Patel, and R. M. Bhatty. 1968. Blood volume in pregnant women with severe anaemia. J. Obstet. Gynaecol. Brit. Commonw. 75:713-717.

The plasma volume of 100 pregnant women with hemoglobin concentration of 4 g/100 ml or less was similar to that of 35 healthy pregnant women at the same stage of pregnancy. Red-cell mass was reduced and resulted in a greatly diminished circulating blood volume in the anemic women.

Whiteside, M. G. 1960. The dilution anaemia of pregnancy. Med. J. Aust. 2:284-286.

In 11 out of 23 pregnant women receiving 200 mg of ferrous sulfate three times a day, a significant fall in hemoglobin was observed and was associated with an excessive rise in plasma volume and a normal rise in red-cell volume. Serum protein changes also differed from the nonanemic group. It was concluded that an excessive rise in plasma volume may be a cause of anemia in pregnancy despite iron therapy. Dilution anemia requires further study.

Wintrobe, M. M. 1933. Blood of normal men and women. Erthrocyte counts, hemoglobin and volume of packed red cells of 229 individuals. Bull. Johns Hopkins Hosp. 53:118-130.

Several hundred blood determinations are analyzed. An average red-cell count of 4.8 million, with a range from 4.2 million to 5.4 million, is suggested for women. An average hemoglobin of 14 g, with a range from 12 to 16 g, is also suggested, as is a packed red-cell volume averaging 42 cc with a range from 37 to 47 cc.

Woodruff, A. W. 1955. The natural history of anaemia associated with protein malnutrition. Brit. Med. J. 1:4925-5307.

Special features of anemia associated with protein malnutrition and its pathogenesis are discussed.

World Health Organization 1959. Iron deficiency anaemia. WHO Tech. Rep. Ser. No. 182, Geneva, 15 p.

Report discusses the 1959 status of iron-deficiency anemia in relation to etiology, prevention, and evaluation.

The Effect of Nutritional Deficiencies on Fetal Growth and Development

Agustin, C. E., and B. F. Chow. 1968. Amino acid imbalance and prematurity in rats. Fed. Proc. 27:728.

Amino acid imbalance in pregnant rats was created to test its effects on length of gestation.

Aksu, O., B. Mackler, T. H. Shepard, and R. J. Lemire. 1968. Studies of the development of congenital anomalies in embryos of riboflavin-deficient galactoflavin fed rats. II. Role of the terminal electron transport systems. Terratology 1(1):93-102.

Studies of the tissues of embryos and fetuses of rats treated with a riboflavin-deficient, galactoflavin-augmented diet during pregnancy demonstrated that activities of the succinic and DPNH oxidase systems were reduced from control values.

Anilane, J. K. 1968. Muscle growth in congenitally malnourished offspring. Fed. Proc. 27:728.

The leg musculature, muscle DNA, protein, and RNA of progeny from underfed and normal mother rats were examined at birth and weekly to 21 days of age, after which restricted animals were given an ad libitum food intake.

Apgar, J. 1968. Comparison of the effect of copper, manganese, and zinc deficiencies on parturition in the rat. Amer. J. Physiol. 215(6):1478-1481.

A comparison of the effects of diets low in copper, manganese, or zinc on parturition in the rat has shown that a low-copper or low-manganese diet has little or no effect, whereas a low-zinc diet results in difficult parturition and high mortality of the pups.

Asling, C. W., M. M. Nelson, H. D. Dougherty, H. V. Wright, and H. M. Evans. 1960. The development of cleft palate resulting from maternal pteroylglutamic (folic) acid deficiency during the latter half of gestation in rats. Surg. Gynecol. Obstet. 111:19-28.

Development of cleft palate resulting from maternal deficiency of pteroylglutamic acid during the latter part of gestation was studied and compared with normal palatal development.

Barnes, R. H. 1967. Experimental animal approaches to the study of early malnutrition and mental development. Fed. Proc. 26:144-147.

A general background review of experimental animal studies, which indicates that restriction of total food intake, either prior to weaning or immediately after weaning, appears to affect animal behavioral patterns. It discusses the difficulty in interpreting these behavioral changes.

Barnes, R. H., S. R. Cunnold, R. R. Zimmermann, H. Simmons, R. B. MacLeod, and L. Kroot. 1966. Influence of nutritional deprivations in early life on learning behavior of rats as measured by performance in a water maze. J. Nutr. 89:399-410.

A study of learning behavior in rats subjected to different forms of nutritional deprivation before and after weaning. Visual performance in a Y water maze at 6 to 9 months of age, and testing for position reversal performance in the water maze shortly after weaning, indicated retardation in the development of learning behavior in the deprived groups.

Berdjis, C. C. 1958. Late effects of hypervitaminosis A in the rat. Amer. Med. Ass. Arch. Pathol. 66:278-281.

Rats fed excessive vitamin A (100-300 IU/g of body weight per day) during pregnancy produced litters prematurely and had reduced numbers of offspring per litter. Most of the offspring showed severe congenital anomalies, and the 40 percent that survived were underweight, weak, and growth-retarded.

Berg, B. N. 1965. Dietary restriction and reproduction in the rat. J. Nutr. 87:344-348.

Investigation of the effect of 25, 50, and 75 percent restrictions of a stock diet on reproduction in the rat revealed that severe restrictions resulted in decreased maternal and fetal weight, ability to litter, and fetal skeletal development.

Berg, B. N. 1967. Maintenance of pregnancy in protein-deprived rats by transitory protein supplements during early gestation. J. Nutr. 92:66-70.

From mating and throughout pregnancy, rats received a protein-free diet with a protein supplement on either days 0 to 4, or 5 to 9; controls were given a diet containing 20 percent protein throughout pregnancy. In the deprived rats, the protein supplements at either stage supported the pregnancy and maintained litter size, but fetal growth was retarded.

Borglin, N. E., and V. Falk. 1959. Observations on the supposed deficiency of vitamin B_6 in pregnancy. Acta Obstet. Gynceol. Scand. 38:190-196.

A study of the excretion of xanthurenic acid after tryptophan loading and serum transaminase activity with 40 mg and without supplementary pyridoxin in 20 healthy pregnant women. A significantly higher excretion of xanthurenic acid was noted in the group without supplementary pyridoxin, and no difference was noted between groups in serum transaminase activity.

Brown, M. L. 1963. Effect of a low dietary level of three types of fat on reproductive performance and tissue lipid content of the vitamin B_6 deficient female rat. J. Nutr. 79:124-130.

A study was made to determine the effect of a low level of three dietary fats fed with and without pyridoxine supplementation on reproductive performance and tissue lipid content of female rats. No significant difference was found, although rats fed corn oil showed some advantage in fetal weight. Organ lipid composition and serum glutomic oxalacetic transaminase were related to type of dietary fat or to vitamin B_6 deprivation.

Cabak, V., and R. Najdanvic. 1965. Effect of undernutrition in early life on physical and mental development. Arch. Dis. Child. 40:532-534.

Serbian children who had been seriously undernourished in infancy were found at school age to have normal physical characteristics but subnormal mental capacity when compared with children who had not suffered malnutrition.

Caldwell, D. F., and J. A. Churchill. 1966. Learning impairment in rats administered a lipid free diet during pregnancy. Psychol. Rep. 19:99-102.

Offspring (N = 31) of 10 pregnant rats fed a fat-free diet during pregnancy were compared with offspring (N = 41) of 10 pregnant rats fed a standard laboratory diet throughout pregnancy. The mean weight of the fat-deprived offspring at birth was less than for the controls but thereafter did not differ. Performance on a maze for a food reward was significantly poorer for fat-deprived animals than for controls.

Caldwell, D. F., and J. A. Churchill. 1967. Learning ability in the progeny of rats administered a protein-deficient diet during the second half of gestation. Neurology. 17:95-99.

The offspring of rats deprived of protein from midpregnancy to term were found to be significantly smaller at birth than offspring of rats maintained on regular feed throughout pregnancy. The protein-deficient rats had longer gestation and higher early mortality and were smaller at weaning than controls. When tested in a water maze at 30 and 55 days, the protein-deficient rats were able to learn tasks but were not so proficient as controls.

Chamberlain, J. G., and M. M. Nelson. 1963. Multiple congenital abnormalities in the rat resulting from acute maternal niacin deficiency during pregnancy. Soc. Exp. Biol. Med. 112:836-840.

Pregnant rats were given a purified niacin-deficient diet containing a niacinamide antimetabolite during pregnancy. A high incidence of embryonic mortality or abnormality was observed.

Chase, H. P., J. Dorsey, and G. M. McKhann. 1967. The effect of malnutrition on the synthesis of a myelin lipid. Pediatrics. 40:551-559.

Nutritional deprivation during early life of the rat is associated with a decreased synthesis of sulfatide (a lipid that is a component of the myelin sheath) as determined by both in vivo and in vitro methods. The decreased synthesis after a period of deprivation is not corrected by refeeding.

Cheek, D. B., and R. E. Cooke. 1964. Growth and growth retardation. Ann. Rev. Med. 15:357-382.

A review article that includes a survey of the literature to September 1963.

Chow, B. F. 1964. Growth of rats from normal dams restricted in diet in previous pregnancies. J. Nutr. 83:289-292.

Dietary restriction of dams from mating until weaning of progeny, or during lactation alone, resulted in growth-stunting of offspring. Offspring of subsequent pregnancies, during which the dams were fed normally, were not affected.

Chow, B. F., and C. E. Agustin. 1965. Induction of premature birth in rats by a methionine antagonist. J. Nutr. 87:293-296.

With date of conception inferred from first appearance of sperm in the vagina, pregnant rats were injected with graded doses of ethionine up to and including the amount known to cause resorption or stillbirth. The incidence of prematurity, and its degree, were directly related to the dose of ethionine. The weight deficits of the premature pups were corrected within 3 months.

Chow, B. F., and C. J. Lee. 1964. Effect of dietary restriction of pregnant rats on body weight gain of the offspring. J. Nutr. 82:10-18.

Restriction of dietary intake of rats during gestation by as little as 25 percent resulted in growth-stunting and anemia and reduced resistance to stress of pregnancy that was correctable by early administration of pituitary growth hormone. The stunting was not thought to be due to insufficient intake of a single limiting nutrient.

Chow, B. F., and R. W. Sherwin. 1965. Fetal parasitism? Arch. Environ. Health 10:395-398.

The relationship between maternal diet and the production of runts in litters of rats was investigated.

Coursin, D. B. 1965. Undernutrition and brain function. Bordens Rev. Nutr. Res. 26:1-16.

A discussion of normal brain development, range of mental retardation, biochemical causes, and clinical manifestations.

Cowley, J. J., and R. D. Griesel. 1966. The effect on growth and behaviour of rehabilitating first and second generation low protein rats. Anim. Behav. 14:506-517.

Comparisons are made between the first and third generations of rats born to mothers reared on a low-protein diet and first-generation deprived rats rehabilitated for two generations on a stock diet. The conclusion is that a low-protein diet given to one generation of rats decreases growth, development, and intelligence, and that more than one generation may have to be given a high-protein diet before the effects of protein deprivation are eliminated.

Cravioto, J. 1963. Application of newer knowledge of nutrition on physical and mental growth and development. Amer. J. Pub. Health 53:1803-1809.

Selective discussion of the effects of varying levels of undernutrition on different parameters of physical and mental maturation.

Davison, A. N., and J. Dobbing. 1966. Myelination as a vulnerable period in brain development. Brit. Med. Bull. 22:40-44.

The authors review recent work on the chemical constitution of the myelin sheath and on its metabolic stability. They suggest that the myelin content of the brain may be reduced only during the period of myelination. The hypothesis that small dietary restrictions imposed during this vulnerable period of brain development may have lasting effects on intellectual capacity and physical growth needs investigation.

Dickerson, J. W. T., G. A. Gresham, and R. A. McCance. 1964. The effect of undernutrition and rehabilitation on the development of reproductive organs: Pigs. J. Endocrinol. 29:111-118.

In female pigs subjected to prolonged undernutrition, the primordial follicles in the ovaries developed and some became large and cystic, but ovulation did not follow. During rapid rehabilitation, ovulation did not occur until the females approached the body weight at which ovulation normally occurs.

Dobbing, J. 1964. The influence of early nutrition on the development and myelination of the brain. Proc. Roy. Soc. (Biol.) 159:503-509.

By adjusting litter size at birth, the rate of growth of brain and brain cholesterol accumulation in suckling rats was investigated. Cholesterol deposition in myelin sheath was affected by a reduced plane of nutrition.

Dobbing, J. 1965/1966. The effects of undernutrition on myelination in the central nervous system. Biol. Neonat. 9:132-147.

Studies of (1) the effects of undernutrition during the maximum rate of myelination in rats and (2) the effect of severe prolonged undernutrition in rats after weaning and in pigs during the second half of the period of maximum myelination and prolonged to 1 year of age.

Dobbing, J., and E. M. Widdowson. 1965. The effect of undernutrition and subsequent rehabilitation on myelination of rat brain as measured by its composition. Brain 88:357-366.

The effects of a reduced plane of nutrition on rat's brain, its lipid, and other constituents were examined from the time of weaning to 11 weeks of age. Brain weight was reduced and there was a high concentration per unit weight of brain constituents. Recovery was achieved with unlimited food.

Everitt, G. C. 1964. Maternal undernutrition and retarded foetal development in Merino sheep. Nature 201:1341-1342.

Fetal weight and the weight of associated placental cotyledons at 90 days' gestation were significantly lighter in severely malnourished ewes than in well-nourished controls.

Fisher, C. J., and J. H. Leathem. 1965. Effect of a protein-free diet on protein metabolism in the pregnant rat. Endocrinology 76:454-462.

Changes in RNA, DNA, protein, alanine transaminase, and aspartate transaminase of uterus, placenta, and liver were investigated in nutritionally deficient pregnant rats maintained with and without estrogen and progesterone. Serum total protein and electrophoretic fractions were also studied.

Frobish, L. T., V. C. Speer, and V. W. Hays. 1966. Effect of protein and energy intake on reproductive performance in swine. J. Anim. Sci. 25:729-733.

Three trials were conducted to study effects of protein and energy intake during gestation on reproductive performance of sows that were fed an experimental diet through three successive reproductive cycles. No significant difference was found in the number of pigs farrowed alive, birth weight of live pigs, or weight gain from birth to weaning.

Fuhrmann, W. 1965. Genetics of growth and development of the fetus. Pediat. Clin. N. Amer. 12:457-475.

A review of present knowledge of the role of genetics in fetal growth and development.

Geysta, S. R., and B. Christie. 1968. Effect of protein-calorie deficiency on prenatal mortality. Indian J. Med. Res. 56:114-122.

Prenatal mortality during the first 13 days of pregnancy was determined in rats that had been maintained from weaning on diets low in protein value. The preimplantation and postimplantation mortality was higher in protein-deficient rats than in controls.

Giroud, A. 1959. The nutritional requirements of embryos and the repercussions of deficiencies. World Rev. Nutr. Diet. 1:231-263.

The author reviews the nutritional requirements of higher vertebrates throughout embryonic and fetal life. Included are sections on nutritional sources of the embryo, maternal nutritional stores, requirements of the various nutrients, effects of general and specific deficiencies, causes of deficiencies, and the role of genetic conditions.

Giroud, A. 1968. Nutrition of the embryo. Fed. Proc. 27:163-184.

A discussion of the origin and utilization of foodstuffs in birds and mammalian embryos, nutrient requirements in relation to embryonic development, the repercussion of deficiencies and excesses, and the interaction between nutrition and genetic conditions in fetal growth.

Goldstein, H. B., and D. G. McKay. 1965. Lipid peroxides and the diet-induced Shwartzman reaction. Amer. J. Obstet. Gynecol. 91:843-846.

In pregnant rats, a deficiency of the antioxidant properties of vitamin E was suspected as the basis of cellular damage leading to an eclamptic state.

Gruenwald, P. 1963. Chronic fetal distress and placental insufficiency. Biol. Neonat. 5:215-265.

The author differentiates between chronic fetal distress of prolonged duration, subacute distress extending over days, and acute perinatal distress of a few hours and discusses characteristics of infants subjected to chronic distress, evidences of malnutrition, and retarded growth.

Gruenwald, P., M. Dawkins, and R. Hepner. 1963. Chronic deprivation of the fetus. Sinai Hosp. J. 11:51-80.

A report of a panel discussion on the etiology and effects of chronic fetal deprivation. In animals (sheep), intrauterine malnutrition can be produced in the latter half of gestation by underfeeding and results in underweight fetus with low levels of liver and cardiac glycogen.

Guilbert, H. R., and H. Goss. 1932. Some effects of restricted protein intake on the estrous cycle and gestation in the rat. J. Nutr. 5:251-265.

In a diet containing less than 7 percent protein, estrous became irregular and finally ceased.

Guthrie, H. A., and M. L. Brown. 1968. Effect of severe undernutrition in early life on growth, brain size and composition in adult rats. J. Nutr. 94:419-426.

A study to determine the effect of varying periods of undernutrition in postnatal life on the size and chemical composition of the brain in rehabilitated adult rats.

Hafez, E. S. E. 1963. Symposium on growth: Physiogenetics of prenatal and postnatal growth. J. Anim. Sci. 22:779-791.

A review of knowledge concerning the factors that affect prenatal and postnatal growth in farm animals. Genetic factors, size, age, and maturation of the maternal organism, litter size, and ambient temperature are discussed with reference to prenatal growth.

Hammond, J. 1961. Effect of nutrition on the stage of development of the young at birth in farm animals. In Ciba Foundation Symposium on Somatic Stability in the Newly Born, Little Brown & Company, Boston, 393 p.

Discusses varying effects of nutrition during embryonic and fetal growth stage in animals and suggests that the nutritional level of the mother in the fetal stage affects both the size of young at birth and the level of anatomical and physiological development.

Hazelwood, R. L., and M. M. Nelson. 1965. Steroid maintenance of pregnancy in rats in the absence of dietary protein. Endocrinology 77:999-1013.

Alterations of blood, liver, skeletal muscle, uterus, placenta, and fetuses of mother rats fed a zero percent casein diet, and injected with estrone and progesterone from the 3rd to the 20th day of gestation, were investigated. The results suggest that skeletal protein is utilized and transferred to the developing fetuses in the absence of dietary protein.

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Heap, F. C., G. A. Lodge, and G. E. Lamming. 1967. The influence of plane of nutrition in early pregnancy on the survival and development of embryos in the sow. J. Reprod. Fert. 13:269-279.

Sows were given varying levels of reduced quantities of diet and were slaughtered at 28 days' gestation. The plane of feeding, after adjustment for differences in sow weight, had no significant effect on the number of normal embryos or embryo survival. Mean number of corpora lutea increased with plane of feeding, and mean weight of embryonic membranes and volume of embryonic fluids were greater at a low plane of feeding.

Howard, E., and D. M. Granoff. 1968. Effect of neonatal food restriction in mice on brain growth, DNA and cholesterol, and on adult delayed response learning. J. Nutr. 95:111-121.

A study of the long-term effects of a limited period of nutritional restriction on ultimate brain size and functional capacity.

Hsueh, A. M., C. E. Agustin, and B. F. Chow. 1967. Growth of young rats after differential manipulation of maternal diet. J. Nutr. 91:195-200.

Differential manipulation of various nutrients in restricted maternal diet in pregnant rats was used to determine which nutrient would prevent stunting. Protein was found to be critical.

Hsueh, A. M., and B. F. Chow. 1968. Gastric secretion in progeny of underfed mother rats. Fed. Proc. 27:728.

The function of the stomach of progeny of mother rats on restricted diets during gestation was investigated. The pH and titratable acidity of gastric secretion was compared at 4 and 10 months.

Huggett, A. St. G. 1946. Applications of prenatal nutrition to infant development. Brit. Med. Bull. 4:196.

This review includes the composition of the human fetus at various stages of development and the rate of sodium transfer across the placenta by various animals.

Huggett, A. St. G. 1959. Some aspects of development--prenatal and neonatal. Ann. Paediat. Fenn. 5:94-97.

The roles of genetic (or species) impulse and of food supply in determining the rate of fetal growth in several animals and the human are discussed.

Hurley, L. S., and H. Sevenerton. 1966. Congenital malformations resulting from zinc deficiency in rats. Proc. Soc. Exp. Biol. Med. 123:692-696.

A mild but specific zinc deficiency in pregnant female rats produced gross congenital malformations in full-term fetuses. The fetuses from zinc-deficient females contained less zinc than did their controls.

Hurley, L. S., N. E. Volkert, and J. T. Eichner. 1965. Pantothenic acid deficiency in pregnant and non-pregnant guinea pigs, with special reference to effects on the fetus. J. Nutr. 86:201-208.

The effect of temporary pantothenic acid deficiency was studied in pregnant guinea pigs, starting during the 9th week of gestation.

Johnson, E. M., M. M. Nelson, and I. W. Monie. 1963. Effects of transitory pteroylglutamic acid (PGA) deficiency on embryonic and placental development in the rat. Anat. Rec. 146:215-219.

Gravid rats placed on pteroylglutamic acid-deficient diets from the 8th to the 10th day of gestation, followed by a PGA-supplemented diet from the 10th day to autopsy, resulted in 18 percent embryonic death by the 11th day to 100 percent by the 13th day. The nature of the resulting morphological change and growth retardation is discussed.

Juneja, H. S., S. K. Murthy, and J. Ganguly. 1964. Effect of retinoic acid on reproductive performance of male and female rats. Indian J. Exp. Biol. 2:153-154.

In female rats on a diet deficient in vitamin A, retinoic acid maintained normal estrus cycles and fertilization but not full-term gestation.

Kendall, K. A., and R. L. Hays. 1960. Maintained pregnancy in the rat as associated with progesterone administration and multiple-nutrient deficiency. J. Nutr. 70:10-12.

Progesterone (5 mg daily) maintained pregnancy in sucrose-fed rats. Data suggest that progesterone may play a role in mobilization of nutrients from maternal stores or may contribute to the maintenance of placental function.

Kinzey, W. G., and H. H. Srebnik. 1963. Maintenance of pregnancy-deficient rats with short term injections of ovarian hormones. Soc. Exp. Biol. Med. 114:158-160.

Rats carried fetuses to term in the complete absence of dietary protein when estrone and progesterone were administered on the 5th to 9th days of pregnancy.

Langman, J., H. vanDrunen, and F. Bouman. 1959. Maternal protein metabolism and embryonic development in human beings. Amer. J. Obstet. Gynecol. 77:546-555.

The paper-electrophoretic method was used to analyze sera of 22 normal nonpregnant, 178 normal pregnant, and 265 selected pregnant women with histories of having had infants with congenital malformations. The diagrams were abnormal in 19 of the 265 selected patients, and in 15 of these the pregnancy resulted in abortion, birth of immature, premature infants with or without malformations, or abnormal full-term infants.

Langman, J., and G. W. Welch. 1967. Excess vitamin A and development of the cerebral cortex. J. Comp. Neur. 131: 15-25.

The effect of large doses of vitamin A on the mitotic behavior and cell cycle of the neuroepethelial cells lining the lateral ventricle of the cerebral hemisphere during the 14th and 15th days of gestation was investigated.

Lee, C. J., and B. F. Chow. 1965. Protein metabolism in the offspring of underfed mother rats. J. Nutr. 87:439-443.

Fifty percent dietary restriction during pregnancy and lactation in rats resulted in permanent growth stunting of the progeny. The purpose of this paper was to determine the cause of impaired growth. In the pups of restricted dams, the mean daily food consumption per unit body weight was greater. the fecal and urinary losses were higher, and the mean nitrogen retention was lower. Stunted growth appeared to be attributable to both greater fecal loss and abnormal metabolism.

Lee, C. J., and B. F. Chow. 1968. Metabolism of proteins by progency of underfed mother rats. J. Nutr. 94:20-26.

A 50 percent restrictive diet was fed to rats during gestation and lactation. The progeny of the restricted mothers were subsequently fed diets of different nutritive quality (egg albumin, alpha protein, and casein) in successive periods. Utilization by the restricted progeny varied with the quality of protein. Qualitative and quantitative analysis of urinary amino acids showed restricted progeny excreted more free basic amino acids and more total amino acids than did the unrestricted progeny.

McCance, R. A. 1960. Severe undernutrition in growing and adult animals. Brit. J. Nutr. 14:59-73.

The immediate and delayed effects of prolonged undernutrition were investigated in weaned pigs and young poultry.

McCance, R. A. 1964. The bearing of early nutrition on later development. p. 74-81. In C. F. Mills, and R. Passmore (ed.) Proc. Sixth Int. Cong. Nutr., Livingstone Ltd., London.

The permanent effects of variance in the general plane of nutrition in early life on physical development are discussed.

McCay, C. M., M. F. Crowell, and L. A. Maynard. 1935. The effect of retarded growth upon length of life span and upon ultimate body size. J. Nutr. 10:63-79.

In one group of rats, calories were restricted to maintain weight but not growth, and the animals were not allowed to attain maturity until after 766 and 911 days. In the second group, calories were restricted to cause weight loss. It was found that both groups resumed growth without evidence of permanent stunting, and the life-span was prolonged.

McKay, D. G., and T. C. Wong. 1962. Studies of the generalized Shwartzman reaction produced by diet pathology. J. Exp. Med. 115:1117-1125.

A generalized Shwartzman reaction was produced in near-term pregnant rats by exposure to a diet low in tocopheral followed by a diet containing oxidized lipids during the gestation period without injection of exogenous bacterial endotoxin.

Metcoff, J. 1967. Biochemical effects of protein-calorie malnutrition. Man. Ann. Rev. Med. 18:377-422.

A review that discusses, at length, the biochemical effects of protein-calorie malnutrition during the fetal period.

Millen, J. W., and D. H. M. Woollam. 1959. Maternal nutrition in relation to abnormal foetal development. Proc. Nutr. Soc. 19:1-5.

The relationship between maternal nutrition and abnormal fetal development is reviewed. The need for further clinical investigation of the effect on fetal growth of minor vitamin deficiencies in the first 3 months of pregnancy is stressed.

Mulinos, M. G., and L. Pomerantz. 1940. Pseudohypophysectomy. A condition resembling hypophysectomy produced by malnutrition. J. Nutr. 19:493.

Both growth and thyroid and adrenotropic activity may be disturbed by undernutrition of long duration through changes in the pituitary gland.

Naeye, R. L. 1964. The fetal and neonatal development of twins. Pediatrics 33:546-553.

Body, organ, and placental development were studied in 272 infants who were stillborn or died within the first week of life. Most twin measurements matched mean values for single gestations through the 24th gestational week, but then growth was not so rapid in the twins. More than half the twins weighed less than 1,000 g at birth. Most measurements for dizygotic twins were greater than for monozygotic twins at the equivalent stage of gestation.

Naeye, R. L. 1965. Cardiovascular abnormalities in infants malnourished before birth. Biol. Neonat. 8:104-113.

The neonatal condition of heart and blood vessels of 16 infants malnourished before birth was investigated. Hearts and arterial muscle mass were found to be small for gestational age but relatively large for body weight. Structural variations were noted.

Naeye, R. L. 1965. Malnutrition, probable cause of fetal growth retardation. Arch. Pathol. 79:284-291.

A quantitative, histological study of organ and cellular development in 11 infants with antenatal growth retardation and of 7 infants with postnatal alimentary malnutrition showed that brain, pancreas, heart, lungs, and kidneys were nearest to normal weight, with liver and spleen being disproportionately small. Cell number and quantity of cytoplasm were subnormal in some organs for both groups. The similarities of the abnormalities for both groups suggest that prenatal malnutrition is the cause of fetal growth retardation.

Naeye, R. L. 1965. Organ abnormalities in a human parabiotic syndrome. Amer. J. Pathol. 46:829-842.

Using the cross-placental transfusion syndrome, the author compared organ size and structure of the donor and recipient twin and then compared them with the organ size and structure of the single-born malnourished infant. Organ abnormalities observed in the donor twin closely resemble those abnormalities observed in the malnourished single-born infant.

Naeye, R. L., K. Benirschke, J. W. C. Hagstrom, and C. C. Marcus. 1966. Intrauterine growth of twins as estimated from liveborn birth-weight data. Pediatrics 37:409-416.

Percentile curves representing intrauterine growth of monochorionic and dichorionic twins were constructed from birth weights of liveborn infants at gestational age from 24 to 42 weeks. Monochorionic twins were smaller and had greater interpair variation in birth weight than did dichorionic pairs. The role of subnutrition in the abnormal growth of twins in late fetal life is discussed.

Nelson, M. M., and H. Evans. 1953. Relation of dietary protein levels to reproduction in the rat. J. Nutr. 51: 71-84.

The relation of dietary protein to reproduction was studied in adult rats that were placed the day of breeding on purified diets supplemented with all known dietary essentials, with casein as the protein source. A 5 percent protein (6 percent casein) level was critical. Above this level, reproductive performance was normal, and below, it was severely disturbed.

Newberne, P. M. 1963. Effect of vitamin B_{12} deficiency and excess on embryonic development of the rat. Amer. J. Vet. Res. 24:1304-1312.

The influence of dietary variations of vitamin B_{12} on the structure and tissue composition of 21-day-old rat fetuses was assessed by histological and biochemical means. Absence of vitamin B_{12} resulted in immaturity and damage of offspring.

Newberne, P. M., and B. L. O'Dell. 1959. Hematology in vitamin B_{12} deficient newborn rat. Proc. Soc. Exp. Biol. Med. 100:335-337.

Vitamin B_{12} deficiency in the dam resulted in offspring with anemia, leukopenia, and granulocytopenia. The liver showed reduction in hemopoietic elements, vascular blocks, and an accumulation of lipids in the hepatic cells. Analysis of bone composition was completed.

Ounsted, M., and C. Ounsted. 1968. Rate of intra-uterine growth. Nature 220:599-600.

From a study of growth-retarded and of growth-accelerated newborns, the authors conclude that slow intrauterine growth is determined by a single constraining influence imposed on the conceptus related to a familial maternal factor. Accelerated fetal growth appears to be determined by several additive biological factors.

Payne, P. R., and E. F. Wheeler. 1967. Comparative nutrition in pregnancy. Nature 215:1134-1136.

Maternal starvation reduces the viability of the offspring less in man than in other mammals. It is suggested that this is associated with the less exacting demands made on the mother by slower growing primate fetuses.

Payne, P. R., and E. F. Wheeler. 1968. Comparative nutrition in pregnancy and lactation. Proc. Nutr. Soc. 27:129-137.

From a comparison of rates of prenatal and postnatal growth of various species, the authors offer a quantitative description of mammalian reproduction. The growth of the fetus proceeds as a parabolic function of time to a birth weight that is proportional to metabolic body size of the mother. Milk output during lactation is directly proportional to maternal metabolic body size, and concentration of the milk is proportional to birth weight. Postnatal growth is a function of total protein intake in the milk.

Pearson, P. B. 1937. Effect of a lysine-deficient diet on the estrous cycle. Amer. J. Physiol. 118:786.

It is suggested that lysine may be the limiting amino acid in maintaining ovarian function.

Pfaltz, H., and E. L. Severinghaus. 1956. Effect of vitamin deficiencies on fertility, course of pregnancy, and embryonic development in rats. Amer. J. Obstet. Gynecol. 72:265-276.

Female rats were fed purified diets from which thiamin, riboflavin, panothenic acid, or pyridoxine was withheld during varying periods of gestation and lactation to determine the effect on course and outcome of pregnancy.

Pike, R. L., and M. L. Brown. 1959. Changes in hemoglobin, hematocrit and plasma proteins in vitamin B_6-deficient rats during pregnancy. J. Nutr. 68:551-559.

Vitamin B_6-depleted rats in the 3rd week of pregnancy exhibited a marked fall in concentration of total protein, hemoglobin, and hematocrit, an effect that appeared to be independent of fetal development. Weekly changes of total protein of the plasma, in hemoglobin, and hematocrit were also observed in nonpregnant vitamin B_6-depleted rats.

Pike, R. L., A. Kirksey, and J. Callahan. 1959. Effects of 6 aminonicotinamide administration during pregnancy upon the maternal rat and her offspring. Fed. Proc. 18:541.

An abstract from an annual meeting presents evidence that 6-aminonicotinamide administration to a rat at a level of 1 mg/kg/day throughout pregnancy led to resorption of all fetuses. As the length of time that the antagonist was administered was decreased, the number of resorptions decreased, and the number of live young and mean fetal weight increased. In addition to the effects on the fetuses, the antagonist was found to lower systolic blood pressure in the rat.

Pitt, D. B., and P. E. Samson. 1961. Congenital malformations and maternal diet. Aust. Ann. Med. 10:268-273.

When estimated diets of 99 mothers who bore malformed children were matched with control diets, small differences were noted in several groups of malformations. The differences in diets were usually found in the content of iron and the B vitamins thiamin and niacin.

Platt, B. S. 1962. Proteins in nutrition. Proc. Roy. Soc. (Biol.) 156:337-344.

Pups born of malnourished mothers exhibited pathological changes. These preliminary findings suggest that the nutritional state of the mother before and during pregnancy and lactation may be of importance in the development of neurological disorders in the offspring.

Pond, W. G., W. C. Wagner, J. A. Dunn, and E. F. Walkner. 1968. Reproduction and early postnatal growth of progeny fed a protein-free diet during gestation. J. Nutr. 94:309-316.

Studies were made to ascertain the effect of a diet containing 0.5 percent protein, supplemented with vitamins and minerals, on swine reproduction and subsequent growth and serum protein of progeny when dietary treatment was introduced before breeding for 24 to 28 days after breeding. The protein-free regimen introduced before breeding had a greater adverse effect than introduction at the 24th to 28th day of pregnancy.

Pratt, C. W. M., and R. A. McCance. 1960. Severe undernutrition in growing and adult animals. 2. Changes in the long bones of growing cockerels held at fixed weights by undernutrition. Brit. J. Nutr. 14:75-84.

General observations and histological changes in the long bones of cockerels whose growth was arrested by undernutrition are included.

Radhakrishnan, M. R. 1966. Effect of protein depletion and subsequent repletion in rats at different age period. Part I: Effect of body weight, body measurements and reproductive performance. Indian J. Med. Res. 54(5):486-493.

Groups of rats were maintained on a protein-free diet for a period of 4 weeks when they were 4, 12, 16, and 20 weeks old, respectively. They were then rehabilitated with a diet providing 20 percent protein. Reproductive performance was studied when the rats were 20 weeks old. No difference was noted in the size of litters born to mother depleted at any stage. However, male and female offspring born to mother rats depleted between 12 and 16 weeks weighed significantly less than offspring of controls.

Richardson, L. R., J. Godwin, S. Wilkes, and M. Cannon. 1964. Reproductive performance of rats receiving various levels of dietary protein and fat. J. Nutr. 82:257-262.

Quantitative determinations were made of the amount of protein or fat required for female rats to produce and wean maximal numbers of young when the same diets are fed for several generations.

Ritchie, J. H., M. B. Fish, V. McMasters, and M. Grossman. 1968. Edema and hemolytic anemia in premature infants. New Engl. J. Med. 279:1185-1190.

Widespread edema, anemia, reticulocytosis, and thrombocytosis in premature infants in the second month of life were found to be associated with vitamin E deficiency. The infants were fed commercial formulas with low ratios of vitamin E to polyunsaturated fatty acids with added iron. The low body tocopherol store of prematures (possibly caused by poor maternal nutrition), their rapid rate of growth, and the inadequacies of the artificial feeding were cited as causative factors.

Schlicker, S. A., and D. H. Cox. 1968. Maternal dietary zinc, and development and zinc, iron, and copper content of the rat fetus. J. Nutr. 95:287-294.

Excess zinc (0.4 percent) in the diet of the maternal rat caused a reduction in fetal growth, varying degrees of fetal resorption, increase in total zinc and the concentration of zinc in the maternal rat and fetus, and a reduction of total iron, concentration of iron, and copper in the fetus. No external anatomical malformations were observed in the fetus.

Scott, K. E., and R. Usher. 1966. Fetal malnutrition: Its incidence, causes, and effects. Amer. J. Obstet. Gynecol. 94:951-963.

An epidemiological study was made of infants who were small for gestational age. It is suggested that this group is nutritionally, not genetically, produced, and that intrauterine malnutrition has drastic effects on the fetus.

Scrimshaw, N. S. 1967. Malnutrition, learning and behavior. Amer. J. Clin. Nutr. 20:493-502.

Evidence is reviewed that suggests that malnutrition during the first few years of life may have an adverse effect on subsequent learning and behavior.

Scrimshaw, N. S., and M. Behar. 1961. Protein malnutrition in young children. Science 133:2039-2047.

Status of knowledge as of 1961 in all aspects of protein malnutrition is reviewed.

Serrano, C. V., L. M. Talbert, and L. G. Welt. 1964. Potassium deficiency in the pregnant dog. J. Clin. Invest. 43:27-31.

Bitches were depleted of potassium from mating, and the deficiency did not interfere with pregnancy. Total carcass analysis of the litter and blood samples from the mother and umbilical vessels at 3 or 4 days before term indicated that serum and muscle potassium and carbon dioxide were depleted, and that muscle sodium increased at term in deficient dogs. In the fetus, electrolytes were similar in experimental and control groups, an indication of active potassium transfer to the fetus against a gradient.

Shapiro, S., L. J. Ross, and H. S. Levine. 1965. Relationship of selected prenatal factors to pregnancy outcome and congenital anomalies. Amer. J. Pub. Health 55:268-282.

Maternal morbidity and prior pregnancy experience are examined in relation to fetal loss, prematurity, and congenital anomalies.

Shaw, J. H., and D. Griffiths. 1963. Dental abornormalities in rats attributable to protein deficiency during reproduction. J. Nutr. 80:123-141.

Experiments were conducted on two strains of caries-susceptible rats and a caries-resistant strain to determine the influence of low-protein diets during reproduction on the development and maintenance of dental structures. The offspring of all three strains showed increased frequency of dental abnormalities. A supplement of 1.0 percent DL-methionine to the low-protein diet during gestation led to striking reductions in dental abnormalities.

Shepard, T. H., R. J. Lemire, O. Aksee, and B. Mackler. 1968. Studies of the development of congenital anomalies in embryos of riboflavin-deficient, galactoflavin fed rats. I. Growth and embryologic pathology. Teratology 1:75-92.

Embryos from maternal rats that were maintained on a riboflavin-deficient, galactoflavin-augmented diet were compared by gross and histological examinations with normal controls. The embryos from deficient maternal rats suffered delayed tissue differentiation, mortality, and severe histological anomalies.

Simonson, M., R. W. Sherwin, H. H. Hanson, and B. F. Chow. 1968. Maze performance in offspring of underfed mother rats. Fed. Proc. 27:727.

The progeny of mother rats restricted by 50 percent of an ad libitum dietary intake during gestation and lactation were fed ad libitum after weaning, and performance on an elevated T maze was compared with that of normal controls.

Sinclair, J. C., and W. A. Silverman. 1966. Intrauterine growth in active tissue mass of the human fetus, with particular reference to the undergrown baby. Pediatrics 38:48-62.

Oxygen consumption was measured between 2 and 10 days of postnatal life under resting, thermoneutral conditions. Babies with normal in utero growth exhibited an increase in oxygen consumption with increasing gestation age and birth weight. Babies who were undergrown in utero consumed more oxygen per kilogram of body weight than did normally grown babies of similar weight. The degree of hypermetabolism was correlated with the degree of undergrowth.

Stephan, J. K., and B. F. Chow. 1968. Growth of progeny from rats underfed during gestation only. Fed. Proc. 27:728.

The effect on progeny of dietary restriction during gestation only was investigated.

Stewart, R. J. C. 1968. The influence of protein-calorie deficiency on the central nervous system. Proc. Nutr. Soc. 27:95-101.

Growth, survival, behavior abnormalities, and brain changes in congenitally malnourished pups were studied.

Stoch, M. B., and P. M. Smythe. 1963. Does undernutrition during infancy inhibit brain growth? Arch. Dis. Child. 38:546-552.

A group of Cape colored children who were grossly undernourished during the first year of life were compared with a control group who were adequately nourished. Brain growth, as reflected by head circumference and IQ of the undernourished group, was found to be significantly lower than in controls.

Stuart, H. C. 1945. Effects of protein deficiency on the pregnant woman and fetus and on the infant and child. N. Engl. J. Med. 236:507-513.

A critical review is made of studies of the effects of low-protein diets on pregnancy and on the health of the fetus and offspring.

Thompkins, W. I. 1948. The clinical significance of nutritional deficiencies in pregnancy. Bull. N.Y. Acad. Med. 24:376-388.

An increased incidence of 26 percent in infant deaths, 38 percent in stillbirths, 15 percent in neonatal deaths, and 70 percent in prematures was found in a control group (222) when compared with the research group (593) who received dietary instruction.

Thomson, A. M. 1951. Human foetal growth. Brit. J. Nutr. 5:158-166.

The gross variations in fetal growth under ordinary conditions are discussed. It is suggested that maternal stature, physical health, and diet during pregnancy may have some small but not significant effect on birth weight.

Thomson, A. M., and D. L. Duncan. 1954. The diagnosis of malnutrition in man. Nutr. Abstr. Rev. 24:1-18.

Mortality rate during the first week of life is an indicator of nutritional status, and the importance of growth as a measurement of general health and nutritional status is stressed.

Tompkins, W. T., and D. G. Wiehl. 1954. Epiphyseal maturation in the newborn as related to maternal nutritional status. Amer. J. Obstet. Gynecol. 68:1366-1377.

In over 300 term babies, weighing 5.5 pounds or more at birth, the tibial epiphyseal center in the knee was considered the most sensitive index of infant maturity at birth. On this basis, it was found that, in female babies, the probability that the tibial epiphyseal center of the knee would be present at birth was greater if the mother had received vitamin and protein supplements during pregnancy than if she had received no supplements.

Venkatachalam, P. S., and K. S. Ramanathan. 1964. Effect of protein deficiency during gestation and lactation on body weight and composition of offspring. J. Nutr. 84:38-42.

A study of the effects of feeding a 7 percent wheat protein diet to rats, either during gestation or lactation or both, on the body weight and composition at birth of offspring and of weaning and lactation performance of the dams.

Venkatachalam, P. S., and K. S. Ramanathan. 1966. Severe protein deficiency during gestation in rats on birth weight and growth of offspring. Indian J. Med. Res. 54:402-409.

Pregnant rats were deprived of protein during the first, second, or final week of pregnancy. Litter size, birth weight and gain in weight, mortality, time of eye opening, appearance of body fur, and incidence of congenital defects in the offspring were compared with those of well-fed controls.

Warkany, J. 1958. Production of congenital malformations by dietary measures. J. Amer. Med. Ass. 168:2020-2023.

Congenital defects of the central nervous system, eyes, skeleton, and cardiovascular, urogenital, and other systems have been produced in laboratory animals by deficiencies of various vitamins during pregnancy. The application of these experiments to man is difficult.

Warkany, J. 1960. Nutrition experiments as an instrument of teratologic research in mammals. Borden Rev. Nutr. Res. 21:1-12.

A historical and interpretive discussion of nutritional experiments in animals and production of congenital malformation.

Warkany, J., B. B. Monroe, and B. S. Sutherland. 1961. Intrauterine growth retardation. Amer. J. Dis. Child. 102:249-279.

Literature is reviewed, and a series of cases are diagnosed retrospectively.

Widdowson, E. M. 1968. How the fetus is fed. Proc. Nutr. Soc. 28(1):17-24.

The rate of growth before birth in mammals and the ways through which nutrients reach and are utilized by the fetus are discussed.

Widdowson, E. M., J. W. T. Dickerson, and R. A. McCance. 1960. Severe undernutrition in growing and adult animals; 4. The impact of severe undernutrition on the chemical composition of the soft tissues of the pig. Brit. J. Nutr. 14:457-471.

The chemical composition of skeletal muscles, skin, heart, liver, kidneys, and brain of eight pigs was examined after prolonged periods of undernutrition. The undernutrition caused reversal of normal chemical development, delay in normal maturation, or both.

Widdowson, E. M., and R. A. McCance. 1960. Some effects of accelerating growth. I. General somatic development. Proc. Roy. Soc. (Biol.) 152:188-206.

Accelerating growth in rats by varying the number of rats suckled by a mother from the first day of life resulted in variation in the time needed for the rats to reach maturity.

Widdowson, E. M., W. O. Mavor, and R. A. McCance. 1964. The effect of undernutrition and rehabilitation on the development of reproductive organs: Rats. J. Endocrinol. 29:119-126.

387-735 O - 70 - 9

In female rats, undernourished so that they gained only 20 g of body weight between the 3rd and 11th week of life, the vagina of more than half of the females opened while the animals were still undernourished and had small body weights. The ovarian follicles appeared active, but no corpora lutea were found. Return to unlimited food caused formation of corpora lutea.

Wigglesworth, J. S. 1966. Foetal growth retardation. Brit. Med. Bull. 22(1):13-15.

Mechanism of fetal growth control, clinical features, and pathological findings in "small-for-date" babies, causation of fetal growth retardation, and long-term effects of fetal growth retardation are discussed.

Wigglesworth, J. S. 1968. Disorders of fetal growth. J. Obstet. Gynecol. Brit. Commonw. 75:1234-1236.

The factors that influence growth of the fetus are reviewed, and possible sites of block in fetal nutrition, maternal nutrition, and maternal-fetal transfer especially as related to small-for-date infants are discussed.

Winick, M. 1968. Cellular response with increased feeding in pituitary dwarf mice. J. Nutr. 94:121-124.

In rats, absence of the anterior pituitary hormone reduced cell division even when adequate calories were available.

Winick, M. 1968. Nutrition and cell growth. Nutr. Rev. 26(7):195-197.

A review of techniques for measurement of growth as determined by the number of cells within an organ or the size or density of individual cells and the specific effects of nutrition on cell growth.

Winick, M., and A. Noble. 1966. Cellular response in rats during malnutrition at various ages. J. Nutr. 89:300-306.

Total organ weight, protein, RNA, and DNA were measured during periods of caloric restriction and subsequent refeeding. The results suggest that cellular effects of

malnutrition depend on the phase of growth at the time of malnutrition. Early restriction impeded cell division, and the animal did not recover. Malnutrition at a later stage of growth resulted in reduction of cell size from which recovery could be made. Thus caloric restriction results in some curtailment of normal growth that is reversible by refeeding as long as cell division has not been affected.

Winick, M., and A. Noble. 1967. Cellular response with increased feeding in neonatal rats. J. Nutr. 91:179-182.

The cellular events associated with increased growth rate in rats nursed in groups of three to six animals per mother were examined. Total organ DNA indicated that the organs of the animals contained more cells than the controls; individual cell size was normal.

Woodruff, C. 1966. Nutritional aspects of metabolism of growth and development. J. Amer. Med. Ass. 196:214-218.

Growth patterns in Lebanon, Venezuela, and Ethiopia were compared with more intensive observations carried out in Michigan. Growth failure in underdeveloped countries occurs in the first one-half year of life with some partial recovery among survivors. Malnutrition appears to be most destructive during rapid periods of change during fetal life and early infancy, affecting both the physical and the intellectual function. More research is needed.

Younoszai, M. K., and J. C. Haworth. 1968. Chemical composition of the placenta in normal pre-term, term and intrauterine growth-retarded infants. Amer. J. Obstet. Gynecol. 103:262-264.

Protein concentration of placentas of growth-retarded infants was significantly higher than that of term and preterm infants.

Zamenhof, S., E. van Marthens, and F. L. Margolis. 1968. DNA (cell number) and protein in neonatal brain: Alteration by maternal dietary protein restriction. Science 160:322-323.

Female rats were maintained on an 8 or 27 percent protein diet by pair-feeding for one month before mating and throughout gestation. The brains of the newborn rats from females on the 8 percent protein diet contained significantly less DNA and protein than progeny of the females on the 27 percent diet. Data on DNA indicated that there were fewer cells, and the protein content per cell was also lower.

Zeman, F. J. 1967. Effect on the young rat of maternal protein restriction. J. Nutr. 93:167-173.

Pregnant rats fed a diet that contained 6 percent casein as the only source of protein produced litters of pups with decreased total body weight and length, decreased liver and kidney weights, and increased heart, brain, and thymus weights in relation to total body weight. Half of each litter was given at birth to protein-restricted and control dams to suckle. Lactation failure in the restricted dams resulted in death of the pups. Control-fed pups of restricted dams showed increased mortality and decreased growth rate because of lack of vigor to suckle. The postnatal effects of prenatal protein restruction are the sum of effects observed in the newborn plus inability to suckle adequately.

Zeman, F. J. 1968. Effects of maternal protein restriction on the kidney of newborn young rats. J. Nutr. 94:111-116.

Morphological and histochemical differences were studied in kidneys of newborn young of rats fed a semipurified diet containing either 24 or 6 percent unsupplemented casein as the sole source of protein throughout pregnancy.

Aboul-Khair, S. A., J. Crooks, A. C. Turnbull, and F. E. Hytten. 1964. The physiological changes in thyroid function during pregnancy. Clin. Sci. 27:195-207.

In a study of 15 pregnant and 13 nonpregnant women using labeled iodine at various stages of pregnancy and during 3 months postpartum, it was found that renal clearance of iodine increased early in pregnancy and was associated with a fall in plasma inorganic iodine concentration. All indices of thyroid function returned to the nonpregnant level by the sixth week postpartum. It was concluded that, in pregnancy, goiter reflects and compensates for the lower concentration of iodine available in plasma for the synthesis of thyroxin.

Assali, N. S., R. A. Douglas, Jr., W. W. Baird, D. B. Nicholson, and R. Suyemoto. 1953. Measurement of uterine blood flow and uterine metabolism. Amer. J. Obstet. Gynecol. 66:248.

The uterine blood flow averages 15 ml/100 g of tissue per minute and the oxygen consumption averages 1.9 ml/100 g per minute during the last month of pregnancy.

Assali, N. S., and J. A. Morris. 1964. Maternal and fetal circulations and their interrelationships. Obstet. Gynecol. Surv. 19:923-948.

A review of the circulatory interrelationship of the mother and her fetus with emphasis on hemodynamics.

Aurell, M., and K. Cramér. 1966. Serum lipids and lipoproteins in human pregnancy. Clin. Chim. Acta 13:278-284.

A study of 18 healthy women during 34th to 36th week of pregnancy and 9 months postpartum, with 8 women examined during lactation, showed that hyperlipemia of pregnancy is increased and circulating triglycerides are transported by increased low-density lipoproteins. A change in composition of high-density lipoproteins was noted. Shift to "normal" values was rapid after term and completed within 2 to 3 months with no difference noted during lactation.

Baker, H., H. Ziffer, I. Pasher, and H. Sobotka. 1958. A comparison of maternal and foetal folic acid and vitamin B_{12} at parturition. Brit. Med. J. I:978-979.

Vitamin B_{12} and folic acid serum levels were lower in mothers than in their infants at delivery.

Bakwin, H., and R. M. Bakwin. 1932. Factors influencing the calcium concentration in serum of new-borns. Amer. J. Hyg. 15:766-772.

The concentration of calcium in the serum of both premature and full-term infants was found to be about 1 mg/100 ml above that in the mothers' blood.

Beaton, G. H. 1966. Some physiological adjustments relating to nutrition in pregnancy. Can. Med. Ass. J. 95:622-629.

A review of information from human and animal studies that indicates that a number of adjustments in protein, fat, iron, and calcium metabolism occur during pregnancy.

Behrman, R. E., A. E. Seeds, F. C. Battaglia, A. E. Hellegers, and P. D. Bruns. 1964. The normal changes in mass and water content in fetal rhesus monkey and placenta throughout gestation. J. Pediat. 65:38-44.

During intrauterine development, the rhesus monkey fetus increased proportionately more in total mass than in total body water content. The percentage of fetal total body water, water in fetal plasma, whole blood, and liver decreased linearly to term. The placenta increased in weight and decreased in water.

Biezenski, J. J., and H. C. Moore. 1958. Fibrinolysis in normal pregnancy. J. Clin. Pathol. 11:306-310.

Assessment of fibrinolytic activity in normal pregnancy, labor, and the puerperium indicated a marked decrease in activity in later months of pregnancy and labor and a return to normal after delivery.

Boger, W. P., G. M. Bayne, L. D. Wright, and G. D. Beck. 1957. Differential serum vitamin B_{12} concentrations in mothers and infants. New Engl. J. Med. 256:1085-1087.

A study of 96 paired serum samples from others and infants (umbilical-cord blood) showed that the average serum vitamin B_{12} concentration of the infant is about twice that of the mother. Nonpregnant controls had higher average vitamin B_{12} concentrations than did pregnant women at term.

Boyle, J. A., S. Campbell, A. M. Duncan, W. R. Greig, and W. W. Buchanan. 1966. Serum uric acid levels in normal pregnancy with observations on renal excretion of urate in pregnancy. J. Clin. Pathol. 19:501-503.

The mean serum uric acid levels of 106 healthy pregnant women (mean age 25.8 years) in late pregnancy was 3.61 ± 0.75 mg/100 ml as compared with 3.86 ± 0.72 mg/100 ml in 64 healthy nonpregnant controls. Normal urinary urate excretion in early pregnancy and enhanced renal loss in midpregnancy and late pregnancy were found.

Burt, R. L. 1960. Plasma nonesterified fatty acids in normal pregnancy and the puerperium. Obstet. Gynecol. 15:460-463.

In pregnant women, the nonesterified fatty acid concentration of plasma is increased during 20 to 40 weeks' gestation.

Cheney, M. C., and Z. I. Sabry. 1968. Response of the pregnant rat to variation in protein quality. Fed. Proc. 27:725.

Difference in efficiency of protein metabolism during pregnancy was investigated in rats.

Coons, C. M., and K. Blunt. 1930. The retention of nitrogen, calcium, phosphorus, and magnesium by pregnant women. J. Biol. Chem. 86:1-16.

A series of 23 balance experiments on 9 women between the 11th and 39th weeks of pregnancy showed nitrogen retention that varied with intake, increased calcium retention

toward the end of pregnancy, and a rise in phosphorus retention by the 5th month of pregnancy.

Coryell, M. N., E. F. Beach, A. R. Robinson, and I. G. Macy. 1950. Metabolish of women during the reproductive cycle. XVII. Changes in electrophoretic patterns of plasma proteins throughout the cycle and following delivery. J. Clin. Invest. 29:1559-1567.

Total plasma protein was shown to be 13 percent lower in the third trimester than mean values for nonpregnant women. Significant variations from nonpregnant values for proteins in blood samples continued to be obtained 5 to 6 days postpartum.

Dannenburg, W. N., R. L. Burt, and H. N. Leake. 1962. Plasma lipids in early puerperium. Amer. J. Obstet. Gynecol. 84:1091-1095.

Silicic acid columns were used in separating lipids into triglyceride, phospholipid, and cholesterol ester fractions. Daily estimations were made of these plasma esterified fatty acids during the puerperium, 24 hours after delivery and 1 to 5 days postpartum, and changes were noted. Within 24 hours after delivery, all except esterified cholesterol had increased, and then there was a steady fall in all fractions from 1 to 5 days postpartum.

de Alvarez, R. R., J. F. Afonso, and D. J. Sherrard. 1961. Serum protein fractionation in normal pregnancy. Amer. J. Obstet. Gynecol. 82:1096-1110.

Presents changes in total serum protein and protein fractions determined by filter paper electrophoresis, as well as changes in fibrinogen in and following normal pregnancy.

DeJorge, F. B., D. Delascio, and M. L. Antunes. 1965. Copper and copper oxidase concentrations in the blood serum of normal pregnant women. Obstet. Gynecol. 26:225-227.

In the serum of 15 nonpregnant women, the mean value for copper was 107.7 µg, and in 10 of them copper oxidase activity was 32 mg/100 ml. In 210 pregnant women, copper rose steadily to 410 µg/100 ml from 240 days, and in 143,

the copper oxidase rose from 34 to 64 mg/100 ml. Age, race, and parity seemed to have no effect.

Do-Kumov, S. I. 1968. Serum copper and pregnancy. Amer. J. Obstet. Gynecol. 101:217-222.

Serum copper levels were investigated in 300 healthy pregnant women divided according to lunar month of pregnancy. Serum copper increased continuously during pregnancy, and the onset of labor caused a sharp decline.

Finkle, M. F. 1947. The transmission of radio-strontium and plutonium from mother to offspring in laboratory animals. Physiol. Zool. 20:405.

Before conception or during pregnancy, female rats and mice were injected with radiostrontium or plutonium, which resulted in a low incidence of pregnancy and an increased stillbirth rate. Both radiostrontium and plutonium passed through the placenta and were retained by the fetal tissues.

Gedalia, I., H. Zukerman, and H. Leventhal. 1965. Fluoride content of teeth and bones of human fetuses: In areas with about 1 ppm fluoride in drinking water. J. Amer. Dent. Ass. 71:1121-1123.

In the fetus, flouride content of bone and teeth increases with age and is greater in bones than in teeth. The uptake of fluoride by fetuses was not significantly greater in an area with 1 ppm fluoride than in an area with 0.5 to 0.6 ppm fluoride.

Gerlóczy, F., B. Bencze, J. Szénásy, and D. Kuncz. 1951. Examination of the vitamin E barrier of the placenta. Experientia 7:427.

Umbilical cord blood contains about half as much vitamin E as maternal blood.

Gillman, T., S. S. Naidoo, and M. Hathorn. 1959. Plasma fibrinogen activity in pregnancy. Lancet ii:70-71.

Plasma fibrinogen levels rose during early months of pregnancy and remained elevated into the puerperium. Plasma fibrinolytic activity decreased significantly and remained low until the second stage of labor.

Gray, M. J., A. B. Munro, E. A. H. Sims, C. I. Meeker, S. Solomon, and M. Watanabe. 1964. Regulation of sodium and total body water metabolism in pregnancy. Amer. J. Obstet. Gynecol. 89:760-765.

The amount of sodium retained by 28 normal pregnant women was not influenced by differences in the average quantity of sodium ingested during pregnancy. Both sodium space and total body water increased during pregnancy, with a relatively greater increase in water than in sodium space.

Green, J. G. 1966. Serum cholesterol changes in pregnancy. Amer. J. Obstet. Gynecol. 95:387-393.

Total serum cholesterol in nonpregnant women on hypocholesterolemic diet fell 13 percent. Pregnant women on the same diet showed a fall only in early pregnancy, with an increase in cholesterol after the first trimester to a peak at term. Total cholesterol of pregnant women on normal diets followed the same pattern.

Greenwood, F. C., W. M. Hunter, and A. Klopper. 1964. Assay of human growth hormone in pregnancy at parturition, and in lactation. Detection of a growth hormone-like substance from the placenta. Brit. Med. J. i:22-24.

Serial estimations were made of growth hormone in the plasma of women during pregnancy and the puerperium by an immunological technique. During pregnancy, a second substance immunologically similar to, but not identical with, growth hormone appeared in the blood. The substance prevented estimation of growth hormone and appeared to be derived from the placenta.

Gruenwald, P. 1966. Growth of the human fetus. I. Normal growth and its variation. Amer. J. Obstet. Gynecol. 94:1112-1132.

New values for birth weight in relation to gestational age are presented. It is suggested that the fetal growth curve during much of the third trimester follows a straight-line course. Late in this trimester, differences appear in different population groups. Departure from the straight line occurs at various points in the curve, depending on limitation of the fetal supply line (mother and placenta).

Gupta, A. N., A. K. Sarkar, and R. N. Charavarti. 1967. Lipid-protein interrelationship in pregnancy. Amer. J. Med. Sci. 253:469-472.

In 38 normal pregnant women, all serum lipid fractions were increased in the second and third trimester, and a gradual decrease in total protein level was observed as pregnancy advanced. Albumin decreased in the third trimester, and globulin fractions increased.

Hahn, P. F., E. L. Carothers, W. J. Darby, M. Martin, C. W. Sheppard, R. O. Cannon, A. S. Beam, P. M. Densen, J. C. Petersen, and G. S. McClellan. 1951. Iron metabolism in human pregnancy as studied with the radioactive isotope Fe^{59}. Amer. J. Obstet. Gynecol. 61:477-486.

Tagged iron was fed in single doses, ranging from 1.8 to 120 mg, to 466 women at various stages of pregnancy, and the uptake was determined. As the dosage increased from 9 to 18 mg and beyond, the percentage of the total dose of iron found in the maternal red cells decreased, while the amount of iron taken up increased from the lowest to the highest dose. Uptake of iron increased as gestation progressed.

Hancock, K. W., P. A. Walker, and T. A. Harper. 1968. Mobilisation of iron in pregnancy. Lancet 2:(7577)1055-1058.

Investigation of body iron stores assessed by histochemical examination of sternal bone-marrow aspirates in pregnant women suggested that storage iron in the marrow is mobilized early in pregnancy to provide a source of immediately available iron, and thus it is difficult to predict the hematological state throughout pregnancy by examination in early pregnancy.

Harrison, K. A. 1966. Blood volume changes in normal pregnant Nigerian women. J. Obstet. Gynaecol. Brit. Commonw. 73:717-723.

Serial estimations of red cell and plasma volumes were made in 20 healthy pregnant women during the second half of pregnancy by means of the chromium labeling technique and Evans blue dye dilution method.

Hellegers, A., K. Okuda, R. E. Nesbitt, D. W. Smith, and B. F. Chow. 1957. Vitamin B_{12} absorption in pregnancy and in the newborn. Amer. J. Clin. Nutr. 5:327-331.

Absorption of orally administered vitamin B_{12} was significantly increased in pregnancy in humans and in rats. In spite of increased absorption, the serum vitamin B_{12} levels and content of the liver and kidney showed a decrease, presumably due to the demands of the fetus.

Hendricks, C. H. 1964. Patterns of fetal and placental growth: The second half of normal pregnancy. Obstet. Gynecol. 24:357-365.

The normal growth patterns of fetus and placenta during the last 20 weeks of pregnancy were determined on the basis of observations of a large series of deliveries in which the fetus was living at onset of labor. Placental-fetal weight interrelationships are presented, and the influences of sex of fetus, parity, ethnic origin, and socioeconomic status on fetal growth and weight are discussed.

Hurley, L. S., and N. E. Volkert. 1965. Pantothenic acid and coenzyme A in the developing guinea-pig liver. Biochim. Biophys. Acta 104:372-376.

Pantothenate and coenzyme A levels in livers of fetal guinea pigs increased at 58 days' gestation after remaining constant from the 33rd to 58th days. These results suggest that the period before birth may be critical with respect to pantothenic acid.

Hytten, F. E., and A. I. Klopper. 1963. Response to a water load in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 70:811-816.

The urinary output after drinking 1 liter of water was measured in 22 normal pregnant women and 10 healthy, nonpregnant subjects. In the second trimester, the mean water load excretion rate and amount exceeded the nonpregnant mean. From 30 weeks of pregnancy to the end of pregnancy, the mean excretion rate and amount declined to below the nonpregnant mean.

Hytten, F. E., and D. B. Paintin. 1963. Increase in plasma volume during normal pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 70:402-407.

Plasma volume was measured during pregnancy and 6 to 8 weeks postpartum in 39 healthy, normal primigravidas to obtain the range of plasma volume increase that is normal. The maximum increase in volume ranged from 630 to 1,940 ml (mean 1,230). The rise was not proportional to the nonpregnant plasma levels. The maximum increase in plasma volume was related to birth weight of the baby, not to size of the mother.

Hytten, F. E., A. M. Thomson, and N. Taggart. 1966. Total body water in normal pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:553-561.

Measurements were taken in 93 women at 10 to 38 weeks of pregnancy, and it was found that those with no clinical edema of legs, or generalized edema gained an average of 6.84, 7.19, and 9.80 kg of body water, respectively. The women gained an average of 11 kg of body weight from 10 to 38 weeks of pregnancy and about 7.7 kg of body water or about 70 percent of the weight gain. The multigravidas gained slightly less body weight than did the primigravidas but a similar amount of body water.

Jain, R. C., O. P. Gupta, and H. S. Andleigh. 1968. Serum proteins in third trimester of pregnancy. Indian J. Med. Sci. 22:237-240.

Total serum proteins and various fractions of the serum protein pattern are reported in 50 women in the third trimester of pregnancy. Hypoproteinemia, hypoalbuminemia, and hyperalpha-2 and hyperbeta globulinemia were found. Alpha-1 globulins and gammaglobulins were constant.

Kasius, R. V., A. Randall, W. T. Tompkins, and D. G. Wiehl. 1958. Maternal and newborn nutrition studies at Philadelphia Lying-In Hospital; newborn studies. VI. Infant size at birth and parity, length of gestation, maternal age, height, and weight status. Milbank Mem. Fund Quart. 36:335-362.

Birth weight, chest circumference, crown-sole length, crown-rump length, rump-sole length, hip breadth, and head circumference measurements were completed on 1,466 infants. The mother's height was positively associated with these measurements, and the average of each measurement generally increased with increasing length of gestation. The babies of underweight mothers tend to be smaller than those of overweight mothers. Parity and maternal age were related to infant size.

Keele, D. K., J. L. Kay, J. Brown, and B. Nordquist. 1966. Plasma free fatty acid and blood sugar levels in newborn infants and their mothers. Pediatrics. 37:597-604.

In control infants, the mean free fatty acid level rose to 3 times the cord level after birth and was accompanied by a 25 percent drop in blood sugar level. Thereafter, the blood sugar level remained relatively constant, but the free fatty acid level varied 2.5 to 3 times the cord level. A high free fatty acid level was found in infants of obese mothers, and a low level was found in infants with delayed respirations or of preeclamptic or diabetic mothers.

Kerr, C., H. F. Loken, M. B. Glendening, G. S. Gordon, and E. W. Page. 1962. Calcium and phosphorus dynamics in pregnancy. Amer. J. Obstet. Gynecol. 83:2-8.

The filtered calcium load, net tubular resorption, and urinary excretion were not significantly altered in 24 women studied in the sixth, seventh, or eighth month of pregnancy.

Kidman, B. P., M. L. Tutt, and J. M. Vaughan. 1951. The retention of radioactive strontium and yttrium (Sr^{89}, Sr^{90}, and Y^{90}) in pregnant and lactating rabbits and their offspring. J. Pathol. Bact. 63:253.

Radioactive strontium was administered to normal, pregnant, and lactating rabbits. It was found that the uptake of strontium by the fetal bones was greatest toward the end of pregnancy. Strontium retention by the bones of the adult and the suckling young is inversely related to calcium intake.

Larralde, J., P. Fernandez-Otero, and M. Gonzalez. 1966. Increased active transport of glucose through the intestine during pregnancy. Nature. 209:1356-1357.

In pregnant Wistar rats, absorption of glucose was studied by the Sols and Ponz method in vivo. There was a significant increase in absorption in pregnant rats when compared with controls.

Leitch, I. 1957. Changing concepts in the nutritional physiology of human pregnancy. Proc. Nutr. Soc. 16:38-45.

In pregnancy, there is need to develop criteria of normality.

Lowenstein, L., M. Lalonde, E. B. Deschenes, and L. Shapiro. 1960. Vitamin B12 in pregnancy and the puerperium. Amer. J. Clin. Nutr. 8:265-275.

The serum B_{12} level of normal pregnant women was significantly lower than that of nonpregnant women; it decreased during pregnancy, then progressively increased during the early postpartum period.

Lund, C. J., and J. C. Donovan. 1967. Blood volume during pregnancy. Amer. J. Obstet. Gynecol. 98:393-403.

Plasma and red-cell volumes were studied in 85 normal healthy women in the reproductive age and in 375 normal healthy pregnant women. Plasma volumes first began to change at 6 weeks' gestation and rose rapidly to midpregnancy. From midpregnancy to term, the rise was slower than early in pregnancy.

MacGillivray, I. 1967. The significance of blood pressure and body water changes in pregnancy. Scot. Med. J. 12:237-245.

Blood pressure and fluid retention during pregnancy are discussed in relation to normal changes and those that may have pathological implications.

Macy, I. G., E.Z. Moyer, H. J. Kelly, H. C. Mack, P. C. DiLoreto, and J. P. Pratt. 1954. Physiological adaptation and nutritional status during and after pregnancy. J. Nutr. 52(1):1-83.

An investigation of 1,064 pregnant women (378 white and 686 Negro) from low and middle socioeconomic status is reported. The purpose was to study physiological and chemical variations occurring in normal pregnancies. Normal standards served as a basis for investigating deviations in nutritional status, dietary intakes, clinical symptoms, and racial, socioeconomic, and other influences. The lower nutritional status of the Negro mothers was verified by blood concentration levels of several components at each trimester of pregnancy and after delivery. The biochemical assessments in the Negro mothers were supported by a higher percentage of marginal dietary ratings, less prenatal care, and lower socioeconomic status.

Manahan, C. P., and N. J. Eastman. 1938. Cevitamic acid content of fetal blood. Bull. Johns Hopkins Hosp. 62:478-481.

A mean value for plasma ascorbic acid in newborn infants, premature and full-term, was found to be 1.15 mg/100 ml, and that in mothers was 0.38 mg/100 ml.

McLarly, D. C., and S. A. Fish. 1966. Fetal erythrocytes in the maternal circulation. Amer. J. Obstet. Gynecol. 95:824-830.

In a study of the peripheral blood of 223 antepartum patients, fetal cells were demonstrated in the circulation of 54.3 percent, and a significant relation was found between estimated average volume of fetal blood and length

of gestation. No significant relation was found between the incidence of transplacental hemorrhage and maternal age, parity, previous abortions, ABO-Rh blood types, or period of gestation.

Menon, K., P. D. Radha, and N. Ramaswamy. 1958. The behavior of serum protein factors in pregnancy and puerperium. Indian J. Med. Sci. 12:930-934.

No significant variation was found in the total serum proteins, albumin, and globulin fractions 12 to 24 hours before the onset of labor, at full term, and in the early days of the puerperium.

Merkatz, I., K. A. Martin, and J. M. Beal. 1964. Gastric secretion during pregnancy and lactation: Clinical and experimental studies. Obstet. Gynecol. 24:587-593.

In pregnant women, a transient increase in gastric acid secretion was detected in the postpartum period but could not be correlated with lactation.

Miller, Z. B., E. Naor, L. Milkovich, and W. M. Schmidt. 1964. Serum levels of cystine aminopeptidase, leucine aminopeptidase, and alkaline phosphatase in single and twin pregnancies. Obstet. Gynecol. 24:707-715.

In a study of 50 gravidas whose pregnancy terminated in twins and 50 whose pregnancy terminated in singletons, samples were taken as near the 28th week of pregnancy as possible. It was found that the mean levels of enzymes increased as the length of gestation increased, that mean values of CAPase and LAPase in twin pregnancies exceeded corresponding values in singleton pregnancies, and that the use of CAPase level alone appears satisfactory to distinguish between twin and singleton pregnancies.

Mor, A., W. Yang, A. Schwartz, and W. C. Jones. 1960. Platelet counts in pregnancy and labor. Obstet Gynecol. 16:338-343.

Study indicated a significant and continuous increase in platelet count during pregnancy. Thrombocyte levels are maintained in labor but drop with the onset of placental detachment. The greatest decline in platelet count is in severe hemorrhage and intrauterine death.

387-735 O - 70 - 10

Mulleck, S., O. P. Bagga, and V. DuMullick. 1964. Serum lipid studies in pregnancy. Amer. J. Obstet. Gynecol. 89:766-770.

The total lipids, total and free cholesterol, phospholipid, and B:A lipoprotein ratio in serum were studied in control and pregnant Indian women from high and low socioeconomic levels. All lipid fractions showed a gradual rise throughout pregnancy.

Naismith, D. J. 1968. The foetus as a parasite. Proc. Nutr. Soc. 28(1):25-31.

A description of work with rats in which an attempt is made to establish the physiological basis for the assessment of protein requirements and in which proposals are advanced concerning the mechanisms that act to protect rat and human fetuses against the adverse effects of poor nutrition.

Nelson, G. H. 1965. Serum nonesterified fatty acid levels in human pregnancy as determined by various titration procedures. Amer. J. Obstet. Gynecol. 92:202-206.

The level of unesterified fatty acids in serum in women late in pregnancy and after delivery was compared with levels for nonpregnant women.

Nylander, G. 1953. Placental transfer of iron, an experimental study in the rat. Acta Physiol. Scand. (suppl. 107) 29:1.

This extensive study shows that the major fraction of the fetal iron is transferred in the last 2 days of pregnancy, and that a component of the placenta, probably ferritin, is involved in the transfer.

Okuda, K., A. E. Hellinger, and B. F. Chow. 1956. Vitamin B_{12} serum level and pregnancy. Amer. J. Clin. Nutr. 4:440-443.

In a study of 25 women, 15 to 41 years of age, in their first to ninth pregnancy, the mean serum level of the fetus was considerably higher than that of the mother,

358 ± 36 and 120 ± 14 μμg/ml, respectively. The average level of a group of nonpregnant women of comparable age was 185 ± 12 μμg/ml.

Paaby, P. 1959. Changes in the water content of serum and plasma during pregnancy. Acta Obstet. Gynecol. Scand. 38:297-314.

A longitudinal study of the changes of serum and plasma water during pregnancy in nine women. Some individual variability was noted, but general cyclic changes in serum and plasma water were thought to be related to changes in estrogen production. A sharp drop in water content was found before partuition.

Paaby, P. 1960. Changes in serum protein during pregnancy. J. Obstet. Gynaecol. Brit. Emp. 67:43-55.

Nine healthy, normal pregnant women were investigated under "basal" conditions in pregnancy. During the first 6 months, concentration of total serum and plasma protein and serum albumin decreased. Concentration of globulins increased, except the V-globulin.

Paaby, P. 1960. Influence of age and parity upon the content of water, protein, and haemoglobin in certain tissue and body fluids during pregnancy. Acta Obstet. Gynecol. Scand. 38:135-142.

In 98 pregnant women, no relationship was demonstrated between the parameters in the paper title and age and parity.

Page, E. W., ed. 1960. Symposium on physiology of pregnancy. Clin. Obstet. Gynecol. P. B. Hoeber, New York. 408 p.

The physiology of the human placenta, hormones in pregnancy, carbohydrate metabolism, the plasma lipids, proteins, and other topics relating to normal physiology of pregnancy are discussed.

Paintin, D. B. 1962. The size of total red cell volume in pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 69:719-723.

In 25 normal primigravidas between the 8th and 16th weeks of gestation, total red-cell volume rose by about 250 ml, with a wide degree of individual variation.

Phillips, G. D., and S. K. Sundaram. 1966. Sodium depletion of pregnant ewes and its effects on foetuses and foetal fluids. J. Physiol. 184:889-897.

Sodium depletion of the ewe at different stages of pregnancy resulted in a fall in saliva Na^+ level, a rise in saliva K^+ level, a fall in plasma Na^+ and K^+ levels, a 35 percent reduction in plasma volume, and a 16 percent reduction in body weight. The sodium levels of fetal plasma and amniotic fluid were lower and the volume of allantoic fluid was greater than in controls. The authors concluded that sodium depletion of the ewe is related to sodium deficiency in the fetus.

Pommerenke, W. T., P. F. Hahn, W. F. Bole, and W. M. Balfour. 1942. Transmission of radioactive iron to the human fetus. Amer. J. Physiol. 137:164-170.

By studying the effect of radioactive iron given to women just before delivery, it was found that the iron reached the fetal circulation within 40 minutes after ingestation, which indicated that iron was distributed through the plasma rather than through the blood cells.

Pritchard, J. A. 1965. Changes in blood volume during pregnancy and delivery. Anesthesiology. 26:393.

In a review of previous studies, the author suggests that there is a moderate increase in blood volume during the first trimester, a more marked rise in the second, and a slight increase in the third. No late decrease in blood volume was demonstrated.

Pritchard, J. A., and R. H. Adams. 1960. Erythrocyte production and destruction during pregnancy. Amer. J. Obstet. Gynecol. 79:750-757.

Measurements during the latter half of pregnancy of total red-cell volume, red-cell utilization of isotopic iron,

reticulocyte levels, erythrocyte glycolytic and cholinesterase activities and the life span of red cells in 48 women indicate an accelerated rather than retarded erythropoiesis.

Prystowsky, H. 1957. Fetal blood studies. VII. The oxygen pressure gradient between the maternal and fetal bloods of the human in normal and abnormal pregnancy. Bull. Johns Hopkins Hosp. 101:48.

The average oxygen pressure gradient was 20 mm Hg in normal pregnant women, and in a variety of pathological states it was markedly lowered.

Prystowsky, H., A. Hellegers, E. Ranke, B. Ranke, and B. Chow. 1959. Further observations on the metabolism of vitamin B_{12} in human pregnancy. Amer. J. Obstet. Gynecol. 77:1-5.

Maternal levels of vitamin B_{12}, transaminase content in the serum, and the glutathione content of the erythrocytes of fetal and maternal blood were lower than fetal levels and those found in nonpregnant women.

Pullman, R. P., W. N. Dannenburg, R. L. Burt, and N. H. Leake. 1962. Carotene and vitamin A in pregnancy and the early puerperium. Soc. Exp. Biol. Med. 109:913-916.

Using daily serial determinations for each patient and then averaging values, the investigators found that the average values for carotene were higher, and those for vitamin A lower, in the pregnant than in the nonpregnant group, and the changes were presumably not dependent on dietary intake. Postpartum values showed a drop in carotene and an increase in vitamin A on the first day postpartum; they remained unchanged through the fifth day postpartum.

Quinto, P., F. Bottiglioni, and C. Flamigni. 1967. Metabolic studies of healthy pregnant women. J. Obstet. Gynaecol. Brit. Commonw. 74:544-555.

The results of a study of 155 nonpregnant and 785 pregnant women indicate that in normal pregnancy lipolysis, the amount of circulating fats, and peripheral utilization of fatty acids are increased to meet the energy requirements

of the tissues and to counterbalance the reduced capacity to metabolize glucose.

Reboud, P., J. Groulade, P. Groslambert, and M. Colomb. 1963. The influence of normal pregnancy and the post-partum state on plasma proteins and lipids. Amer. J. Obstet. Gynecol. 86:820-828.

In normal pregnancy, albumin levels fall gradually; fibrinogen and A_2- and B-globulin increase markedly. There is little noticeable variation in glycoprotein fractions. Lipids and lipoproteins fall slightly at onset of pregnancy, increase gradually, and are well above normal at the end of gestation.

Sadovsky, A., B. Bercovici, M. Rachmilewitz, N. Grossowicz, and J. Aronovitch. 1959. Vitamin B_{12} concentration in maternal and fetal blood. Obstet. Gynecol. 13:346-349.

Concentrations of vitamin B_{12} were higher in the newborn than in maternal blood.

Seitchik, J. 1967. Total body water and total body density of pregnant women. Obstet. Gynecol. 29:155-166.

Estimates of total body water and total body density were made on pregnant women to determine whether protoplasmic solids are retained in pregnancy.

Seitchik, J., C. Alper, and A. Szutka. 1963. Changes in body composition during pregnancy. Ann. N.Y. Acad. Sci. 110:821-829.

Measurement of total body water and body density throughout pregnancy in 21 middle-class Caucasian women, all apparently healthy, indicated a slight increase in total body water, with a redistribution between the water compartments of the mother. Body density decreased slightly in pregnancy, which indicated weight gain due to fat tissue rather than to fat-free body mass.

Singh, H., L. Ramakumar, and I. D. Singh. 1967. Serum proteins in pregnancy at term. J. Obstet. Gynaecol. Brit. Commonw. 74:254-257.

The average serum protein at term of 75 pregnant Indian women was 6.17 g/100 ml below normal adult range. Serum proteins were not affected qualitatively or quantitatively by energy intake, protein intake, or socioeconomic status.

Slater, E. C., and J. Rial. 1942. Thiamine (vitamin B_1) content of human milk. Med. J. Aust. 1:3-12.

Stored thiamin is used during the first few months after birth, until the diet supplies sufficient amounts of the vitamin.

Slobody, L. B., M. M. Willner, and J. Mestern. 1949. Comparison of vitamin B_1 levels in mothers and their newborn infants. Amer. J. Dis. Child. 77:736.

The maternal venous:umbilical cord blood ratio of thiamin averaged 7:11.6.

Smith, E. K., R. R. de Alvarez, J. Forsander. 1959. Serum protein, lipid, and lipoprotein fractions in normal human pregnancy. Amer. J. Obstet. Gynecol. 77:326-334.

In a normal primigravida, serial determinations completed during gestation indicated that serum albumin decreased, whereas alpha globulins and beta globulins increased to delivery. Serum cholesterol, lipid, phosphorus, beta lipoprotein, and total lipid increased progressively. All changes were reversed following delivery.

Snelling, C. E., and S. H. Jackson. 1939. Blood studies of vitamin C during pregnancy, birth and early infancy. J. Pediat. 14:447-451.

A slight fall in plasma ascorbic acid was found toward the end of pregnancy. A higher ascorbic acid level was found in fetal blood than in natural blood.

Speelacy, W. N., F. C. Goetz, B. Z. Greenberg, and J. Ells. 1965. Plasma insulin in normal "early" pregnancy. Obstet. Gynecol. 25:862-865.

Blood glucose and plasma insulin were estimated in 20 women between the 13th and 15th week of pregnancy and 6 weeks postpartum.

Spray, C. M. 1950. A study of some aspects of reproduction by means of chemical analysis. Brit. J. Nutr. 4:354.

The maternal tissues of rats and mice during pregnancy stored more protein, fat, potassium, zinc, and copper than was transmitted to the fetuses.

Svanborg, A., and O. Vikrot. 1965. Plasma lipid fractions, including individual phospholipids, at various stages of pregnancy. Acta Med. Scand. 178:615-630.

Twenty-one healthy, pregnant women, 18 to 33 years of age (mean 25 years of age), were studied once each during different stages of pregnancy, and two other normal pregnant women, 23 and 36 years of age, were studied serially during pregnancy. All lipid concentrations, except that of lysolecithin, increased with time and at the end of pregnancy were well above nonpregnant levels. Cholesterol increased more than phospholipid. It was assumed by the investigators that nutritional factors are not the main cause of pregnancy hyperlipemia but may be a contributing factor. They concluded that alteration in hepatic function is a general phenomenon in pregnancy and can be responsible for some part of the hyperlipemia.

Villee, C. A. 1953. Regulation of blood glucose in human fetus. J. Appl. Physiol. 5:437.

As gestation proceeds, the glycogen content and glucose-producing ability of the placenta decrease. Until the fetal liver develops the enzymes required for this process, the placenta regulates the fetal blood-glucose concentration.

Vosburgh, G. J., and L. B. Flexner. 1950. Maternal Plasma as a source of iron for the fetal guinea pig. Amer. J. Physiol. 161:202.

These studies, in which ^{55}Fe- and ^{59}Fe-labeled iron were used, show that the guinea pig fetus during the last half of gestation receives ample iron for growth from the maternal plasma.

Wachstein, M., and A. Gudaitis. 1953. Disturbance of vitamin B_6 metabolism in pregnancy. II. The influence of various amounts of pyridoxine hydrochloride upon the abnormal tryptophane load test in pregnant women. J. Lab. Clin. Med. 42:98-107.

The abnormal excretion of xanthurenic acid in pregnant women following a test dose of 10 g of DL-tryptophan was suppressed by the administration of 25 mg of pyridoxine hydrochloride given 24 hours before or 1 hour after the test dose of tryptophan.

Wachstein, M. and A. Gudaitis. 1952. Disturbance of vitamin B_6 metabolism in pregnancy. J. Lab. Clin. Med. 40:550-557.

After a test dose of 10 g of tryptophan, large amounts of xanthurenic acid were excreted in the urine of pregnant women. Administration of pyridoxine reduced excretion to normal levels.

Wertz, A. W., M. E. Lojkin, B. S. Bouchard, and M. B. Derby. 1958. Tryptophan-niacin relationships in pregnancy. J. Nutr. 64:339-353.

Twelve women between 21 and 36 years of age were studied for four 14-day periods; two women were in the third trimester of pregnancy and two were in the fourth month. The results, obtained by using divided doses of 500 mg of DL-tryptophan, suggest that conversion of tryptophan to nicotinic acid is more efficient in the pregnant than in the nonpregnant state.

Young, J. E., C. Barrows, K. Okuda, and B. F. Chow. 1959. Vitamin B_{12} serum level in pregnancy. Obstet. Gynecol. 14:149-153.

A progressive decrease in the vitamin B_{12} plasma level in rats and in the venous serum level in humans was observed during gestation. Administration of 100 μg of oral vitamin B_{12} daily did not maintain the serum level at nonpregnant values. Folic acid along with vitamin B_{12} did elevate vitamin B_{12} levels above those found in the nonpregnant state. Cumulative depletion of vitamin B_{12} was noted.

Zelenik, J. S. 1965. Endocrine physiology of pregnancy. Clin. Obstet. Gynecol. 8:528-529.

Endocrine activity and changes occurring in pregnancy are reviewed.

Zuspan, F. P., and S. Goodrich. 1968. Metabolic studies in normal pregnancy. Amer. J. Obstet. Gynecol. 100:7-14.

The daily nitrogen intake of six normal pregnant women studied in a metabolic unit was 12 to 14 g in five women and 6 g in one. All women were in positive nitrogen balance during the antepartum period. The postpartum course showed wide individual variation, with all patients in negative nitrogen balance at least once. At 24 days postpartum, all patients were again in positive balance. The authors estimate that 10 g of protein/kg of body weight should be an adequate intake.

The Role of the Placenta in Pregnancy

Amoroso, E. C. 1955. Endocrinology of pregnancy. Brit. Med. Bull. 11:117.

This brief review considers placental-ovarian relationships and the placenta as an endocrine organ. A comprehensive bibliography is included.

Athanassiu, G. 1947. Vitamin E exchange between placenta and fetus. Klin. Wochnschr. 24-25:362.

More vitamin E was found in the venous than in the arterial umbilical cord blood.

Bagley, D. H., E. J. Zapolski, M. Rubin, and J. V. Princiotto. 1968. Placental transfer of chelated iron in the guinea pig. Amer. J. Obstet. Gynecol. 102:291-296.

Using in situ perfusion technique, the authors studied the passage of chelated iron from the maternal to the fetal compartments in the guinea pig.

Barnes, A. C. 1947. Placental metabolism of vitamin C. I. Normal placental content. Amer. J. Obstet. Gynecol. 53:645.

In comparison with the content of either the fetal or maternal blood, placental tissue has exceedingly high levels of vitamin C. In vitro experiments indicate that vitamin C cannot be synthesized by the human placenta slice.

Barnes, A. C. 1951. Placental metabolism of vitamin A. Amer. J. Obstet. Gynecol. 61:368.

Carotene is the main form of vitamin A transferred by the placenta to the fetus, which converts the carotene to vitamin A and stores it in the liver.

Barrett, M., and G. Everson. 1951. Deposition of B-vitamins in normally developing fetuses as evidence for increased vitamin needs of the rat for reproduction. I. Thiamine (aneurin) and riboflavin. J. Nutr. 45:493.

Small amounts of thiamin and riboflavin are contained in placental tissue.

Bawden, J. W., A. S. Wolkoff, and C. E. Flowers. 1964. Placental transfer of F^{18} in sheep. J. Dent. Res. 43:678-683.

F^{18} was injected intravenously into pregnant sheep. Over an 80-minute period, highest fetal-blood concentration was 15 percent of corresponding maternal-blood concentration. Injection into the fetus showed transfer in the maternal direction.

Bernstine, R. L. 1960. Placental capacity and its relationship to fetal health. Clin. Obstet. Gynecol. 3:852-859.

A review of the normal physiology and anatomy of the placenta and methods of evaluating placental dysfunction.

Bothwell, T. H., W. F. Pribella, W. Mebust, and C. A. Finch. 1958. Iron metabolism in the pregnant rabbit; iron transport across the placenta. Amer. J. Physiol. 193:615.

Placental iron transport occurs against a concentration gradient. The uptake of iron by the placenta is an active process.

Brody, S. 1952. Interrelations between nucleic acids and growth of the human placenta. Exper. Cell Res. 3:702.

As gestation proceeds, the ratio of ribonucleic acid to deoxyribonucleic acid in the human placenta decreases.

Browne, J. C. McC. 1954. Utero-placental circulation. Cold Spring Harbor Symposia Quant. Biol. 19:60.

The author found that the placental blood flow is about 600 ml/minute in the last month of human pregnancy.

Byrn, J. N., and N. J. Eastman. 1943. Vitamin A levels in maternal and fetal blood plasma. Bull. Johns Hopkins Hosp. 73:132-137.

The authors studied placental transfer of vitamin A and fetal storage.

Campbell, R. M., I. R. Innes, and H. W. Kosterlitz. 1953. Some dietary and hormonal effects on maternal, fetal, and placental weights in the rat. J. Endocrinol. 9:68.

A negative correlation between placental weight and litter size was found. Reduction in food intake during late pregnancy did not affect placental weight, but removal of dietary protein did.

Chow, B. F., and K. Okuda. 1960. Transfer of vitamins from mother to fetus. J. Amer. Med. Ass. 172:422-426.

The concentration of vitamin B_{12} in umbilical cord serum in 25 fetuses was compared with maternal serum at delivery. The concentration in fetal serum was higher than that in maternal serum.

Comar, C. L., and G. K. Davis. 1947. Cobalt metabolism studies. III. Excretion and tissue distribution of radioactive cobalt as administered to cattle. Arch. Biochem. 12:257.

Significant quantities of cobalt are transmitted to the fetus from the dam.

Cox, L. W., and T. A. Chalmers. 1953. The transfer of sodium across the human placenta determined by Na^{24} tracer methods. J. Obstet. Gynaecol. Brit. Emp. 60:203.

At the period of gestation studied, the quantities of sodium transferred to the fetus were 450 times greater than were needed for growth.

D'Agostino-Barbaro, A. 1952. The role of placenta in goats. I. Calcium content of the mother's blood, the placenta, and the fetal blood. Riv. Med. Vet. Zootec. 4:19.

The fetal blood calcium content was higher than that of placental blood. The placental blood calcium content was higher than that of maternal blood.

Dancis, J. 1962. The placenta in fetal nutrition and excretion. Amer. J. Obstet. Gynecol. 84:1749-1755.

A discussion of the type of protein, carbohydrate, lipids, nucleic acids, and minerals presented to the fetus, the method of transfer, and qualitative estimates of rate of transfer.

Dancis, J. 1967. The placenta. J. Pediat. 55:85-101.

A review of current concepts of the placenta. Includes an extensive bibliography.

Dancis, J., N. Braverman, and J. Lind. 1957. Plasma protein synthesis in the human fetus and placenta. J. Clin. Invest. 36:398.

The role of the placenta in the synthesis of plasma proteins in the fetus is negligible.

Dancis, J., M. A. Brenner, and W. L. Money. 1962. Some factors affecting the permeability of guinea pig placenta. Amer. J. Obstet. Gynecol. 84:570-576.

Increased pressure in the umbilical vein produced an increase in the permeability of the placenta to water, protein, and red blood cells. Isomolar concentrations of KCL also increased permeability. Lithium and choline did not affect permeability.

Dancis, J., J. Lind, M. Oratz, J. Smolens, and P. Vara. 1961. Placental transfer of proteins in human gestation. Amer. J. Obstet. Gynecol. 82:167-171.

From experiments in early and late human pregnancy with radioactive plasma, proteins, and homologous tetanus antiserum, it was concluded that the allontoic placenta is the route of transfer from mother to fetus rather than transfer through the amniotic fluid and absorption by the fetal gastrointestinal tract or lungs.

Dancis, J., and M. Shafran. 1958. The origin of plasma proteins in the guinea pig fetus. J. Clin Invest. 37: 1093.

The placenta in vitro does not degrade maternal plasma proteins for the use of the fetus. However, it does transfer gammaglobulins and other proteins to the fetus.

Eaton, H. D., A. A. Spielman, J. K. Loosli, J. W. Thomas, C. L. Norton, and K. L. Turk. 1947. The placental transfer and colostral storage of vitamin D in the bovine. J. Dairy Sci. 30:787.

An increase in the maternal blood vitamin D level was caused by supplementary dietary vitamin D, but it did not affect the blood or liver vitamin D levels of the fetuses at birth.

Feaster, J. P., S. L. Hansard, J. T. McCall, and G. K. Davis. 1955. Absorption, deposition and placental transfer of zinc in the rat. Amer. J. Physiol. 181:287-290.

The data presented indicate that zinc moves freely across the placenta to the fetus at all stages of gestation in rats.

Feaster, J. P., S. L. Hansard, J. C. Outler, and G. K. Davis. 1956. Placental transfer of calcium-45 in the rat. J. Nutr. 58:399.

Calcium-45 readily crosses the placenta.

Feltman, R., and G. Kosel. 1955. Prenatal ingestation of fluorides and their transfer to the fetus. Science 122:560.

Fluoride ion is concentrated by the placenta and is readily passed from the mother to the fetus.

Flexner, L. B., D. B. Cowie, L. M. Hellman, W. S. Wilde, and G. J. Vosburgh. 1948. The permeability of the human placenta to sodium in normal and abnormal pregnancies and the supply of sodium to the human fetus as determined with radioactive sodium. Amer. J. Obstet. Gynecol. 55:469.

From the 9th week of gestation to term, the permeability of the placenta to sodium increases about 70 times. Marked reduction of permeability to sodium was shown by a few cases of preeclampsia.

Gardner, D. E., F. A. Smith, H. C. Hodge, D. E. Overton, and R. Feltman. 1952. The fluoride content of placental tissue as related to the fluoride content of drinking water. Science 115:208.

Increased fluoride in drinking water resulted in elevated blood fluorine and placental tissue fluorine content. The placenta contained a higher concentration of fluorine than did the corresponding maternal blood sample.

Gedalia, I., A. Brzezinski, H. Zuckerman, and A. Mayersdorf. 1964. Placental transfer of fluoride in the human fetus at low and high F intake. J. Dent. Res. 43:669-671.

The authors conclude that the placenta is freely permeable to fluoride at low maternal intakes but assumes a regulatory rate at higher intakes.

Gersten, J. 1954. The transfer of radioiodine across placenta and breast. Ohio J. Sci. 54:161.

Iodine was shown to pass the placenta in both directions by injection of radioiodine into pregnant rats or their fetuses *in situ* and by subsequent examination of the animals' thyroid glands.

Hoskins, F. H., and S. L. Hansard. 1964. Placental transfer and fetal tissue iron utilization in sheep. J. Nutr. 83:10-14.

Radioiron and total iron data from blood balance and tissue distribution studies from 24 ewes indicated that fetal sheep organs and tissues possess greater turnover rates during the second trimester of pregnancy than at later stages of gestation. The liver and spleen were important in hematopoiesis early in development, and bone marrow was important later. It appeared that iron crossed the placenta at all stages of gestation and was probably of plasma origin.

Itoh, H., S. L. Hansard, J. C. Gleen, F. H. Hoskins, and D. M. Thrasher. 1967. Placental transfer of calcium in pregnant sows on normal and limited-calcium rations. J. Anim. Sci. 26:335-340.

Twenty-four yearling sows were maintained on normal (0.7 percent) and low calcium (0.3 percent) diets throughout the final trimester of pregnancy. Sows on the normal rations absorbed less labeled calcium, transferred more total and radiocalcium to their fetuses, and deposited more absorbed calcium in maternal tissues. Results indicate that maternal needs influence calcium movement and transfer to the fetus, and the maternal-calcium ratios were lower when dietary calcium was restricted.

Jeacock, M. K. 1963. Calcium content of the human placenta. Amer. J. Obstet. Gynecol. 87:34-40.

In normal placentas, mean calcium concentration at term was double that for placentas delivered before 37 weeks' gestation. At the same gestational age, placentas from patients with severe toxemia had a higher mean calcium concentration than did normal placentas.

Kinzey, W. G., and H. H. Srebnik. 1963. Maintenance of pregnancy in protein-deficient rats with short-term injections of ovarian hormones. Soc. Exp. Biol. Med. 114:158-160.

In the absence of dietary protein, the placenta was able to carry rat fetuses to term, provided estrone and progesterone were administered on the 5th to 9th days of pregnancy.

Koren, Z., and E. Shafrir. 1964. Placental transfer of free fatty acids in the pregnant rat. Soc. Exp. Biol. Med. 116:411-414.

There was little direct placental transfer of albumin-bound, labeled palmitate, stearate, or linoleate administered to albino rats.

Lane, R. S. 1968. Regulating factors in the transfer of iron across the rat placenta. Brit. J. Haemat. 15:365-369.

The effect of maternal blood loss on the iron uptake of the fetectomized placenta was studied in rats. The results suggest that maternal blood flow and plasma iron concentration control placental iron uptake. During the last third of gestation, the fetal liver starts to synthesize transferrin; this permits the establishment of a fetal plasma iron pool.

Lanman, J. T. 1953. Adrenal function in premature infants. II. Adrenocorticotropic hormone-treated infants born of toxemic mothers. Pediatrics 12:62.

Adrenocorticotropin readily crosses the placenta, but maternal cortisone does not.

Laurell, C. 1947. Studies on the transportation and metabolism of iron in the body, with special reference to the iron-binding component in human plasma. Acta Physiol. Scand. 14:129.

Blood serum contains a specific component, perhaps the B_1 globulin fraction, which binds iron and is about one half saturated with it. From the sixth month of gestation, the

387-735 O - 70 - 11

saturation limit rises and then falls immediately after parturition. It was demonstrated that the iron-binding component does not freely pass through the placenta.

Lewis, H., and G. Everson. 1952. Deposition of B vitamins in normally developing fetuses as evidence for increased vitamin needs of the rat for reproduction. II. Pantothenic acid and biotin. J. Nutr. 46:27.

Placental tissue contains very low concentrations of pantothenic acid and biotin. More than 600 μg of pantothenic acid and 3 μg of biotin per 24 hours were transferred through the placenta and deposited in fetal tissues during the period of maximum fetal growth.

Light, A. E. 1953. Fluoride intake with relation to milk and water consumption. Arch. Biochem. Biophys. 47:477.

The fluoride content of the placenta varies widely and parallels the mother's milk intake.

Logothetopoulos, J., and R. F. Scott. 1956. Active iodide transport across the placenta of the guinea pig, rabbit, and rat. J. Physiol. 132:365.

In the animals studies, the placenta actively transports iodide ion into the fetal blood stream.

Lust, J. E., D. D. Hagerman, and C. A. Villee. 1954. The transport of riboflavin by human placenta. J. Clin. Invest. 33:38.

It was found that the placenta may transport riboflavin by taking up flavinadenine dinucleotide from the maternal blood, splitting it enzymatically, and secreting the free riboflavin into the fetal blood.

MacRae, D. J., and D. Palavradji. 1964. Some aspects of the transfer of glucose to the foetus. J. Obstet. Gynaecol. Brit. Commonw. 71:954-959.

Glucose was estimated with glucose oxidase in blood from maternal vein and from umbilical vein and artery at delivery and in the capillary blood from the baby at

6 hours of age. Umbilical-vein blood had less glucose than did that of the mother, but the difference was less in normal conditions than when the mother had pre-eclamptic toxemia and hypertension or when the delivery was premature. At 6 hours of age, the infants had similar values except for the premature group.

Maplesdon, D. C., I. Motzok, W. T. Oliver, and H. D. Branion. 1960. Placental transfer of fluorine to the fetus in rats and rabbits. J. Nutr. 71:70-76.

After dietary supplements of fluoride, the fluoride content of fetal rats and rabbits was slightly increased.

McLaurin, L. P., and J. R. Cotter. 1967. Placental transfer of iron. Amer. J. Obstet. Gynecol. 98:931-937.

In rabbits, the maternal plasma iron turnover was greatly increased at the end of gestation because of the transfer of iron to the fetus. Transfer to the fetus was independent of the maternal plasma level and was probably influenced by affinity of fetal tissues for iron.

Moore, W. M. O., A. E. Hellegers, and F. C. Battaglia. 1966. In vitro permeability of different layers of the human placenta to carbohydrates and urea. Amer. J. Obstet. Gynecol. 96:951-954.

The in vitro permeabilities of different layers of human placenta to D-arabinose, sucrose, urea, and antipyrine were investigated. The chorioamnion and chorion are less permeable per unit area than the amnion. In all three tissues resistance increases in the following order: urea < D-arabinose < sucrose.

Nataf, B., M. Sfez, R. Michel, and J. Roche. 1957. The role of the placenta in regulating iodide and thyroid hormone exchanges between the mother and the fetus in rats. Bull. Soc. Chim. Biol. 39:233.

The placenta may promote the transfer of iodide from the mother to the fetus during the latter part of gestation. The mechanism of placental concentration of iodides appears to be similar to that of iodide concentration in the thyroid gland.

Niswander, K. R., E. A. Friedman, D. B. Hoover, H. Pietrowski, and M. C. Westphal. 1966. Fetal morbidity following potentially anonigenic obstetric conditions. I. Abruptio placentae. Amer. J. Obstet. Gynecol. 95:838-845.

Incidence of abruptio placentae in 17,265 parturients and its relationship to race, parity, maternal age, duration of pregnancy, and toxemia were investigated. Perinatal mortality and the chances of the surviving infant to be neurologically intact are evaluated.

Page, E. W. 1957. Transfer of materials across the human placenta. Amer. J. Obstet. Gynecol. 74:705.

A review of placental transfer.

Piccioni, V., and M. Piccioni. 1947. Vitamin A during pregnancy and lactation. I. Influence of loading doses of vitamin A on the mother and passage of the vitamin through the placenta and through suckling. Acta Vitaminol. Milano 1:27.

Vitamin A can be transmitted from the mother to the young both through the milk and through the placenta.

Plumlee, M. P., S. L. Hansard, C. L. Comar, and W. M. Beeson. 1952. Placental transfer and deposition of labeled calcium in the developing bovine fetus. Amer. J. Physiol. 171:678.

It was found that there is relatively free movement of calcium across the placenta, and the rate of fetal bone mineralization was maximal between the 4th and 7th months.

Wasserman, R. H., C. L. Comar, M. M. Nold, and F. W. Lengemann. 1957. Placental transfer of calcium and strontium in the rat and rabbit. Amer. J. Physiol. 189:91.

Aided by radioactive tracers, the authors showed that the placental transfer from mother to fetus of strontium was about one half that of calcium, and that the site of discrimination was the placenta. About 92 percent of the fetal calcium originated from maternal diet in the rat. About 24 mg of calcium/fetus/day crossed the placenta in the rabbit in late pregnancy.

Winick, M. 1967. Cellular growth of human placenta. III. Intrauterine growth failure. J. Pediat. 71:390-395.

In the placentas from 17 infants with low birth weight but no malformations, protein/DNA was normal and the RNA/DNA ratio was elevated.

Winick, M., A. Coscia, and A. Noble. 1967. Cellular growth in human placenta. I. Normal placental growth. Pediatrics 39:248-251.

The pattern of cellular growth of 50 normal placentas was investigated. Interpretation of the data suggests that cell division stops in the human placenta about 1 month prior to term; the remaining growth is enlargement of existing cells.

Winick, M., and A. Noble. 1966. Quantitative changes in ribonucleic acids and protein during normal growth of rat placenta. Nature 212:34-35.

Total organ weight, DNA, RNA, and protein in the placenta of the rat were measured from 10 days after conception to term. It demonstrated that the placenta increased in weight until 1 day before birth, but DNA synthesis (cell division) ceased on the 17th day.

Younoszai, M. K., and J. C. Haworth. 1968. Placental dimensions and relations in pre-term, term and growth-retarded infants. Amer. J. Obstet. Gynecol. 103:265-271.

A study of placentas of three groups of newborns suggests that the placental decidual area may be an important determinant of infant birth weight.

Beardsley, G. S. 1941. Implications of weight-gain during pregnancy. West. J. Surg. 49:350-353.

Historical discussion of the use of weight control as a method for preventing toxemia. Recommended that the patient's desirable weight at the end of pregnancy be correlated with her normal weight rather than prepregnancy weight. A gain of 25 lb above this normal is considered desirable.

Dennis, K. J., and W. R. Bytheway. 1965. Changes in body weight after delivery. J. Obstet. Gynaecol. Brit. Commonw. 72:94-102.

In 107 normal primigravidas, those with no edema showed slight increases in weight for 3 days after delivery, and no weight was lost until the fourth day. Those with edema showed no weight increase, and the greater the edema the more rapid the weight loss. In 71 normal multigravidas, all showed a rise in weight on the first and second day after delivery, and loss was apparent soonest in those with generalized edema.

Eastman, N. J., and E. Jackson. 1968. Weight relationships in pregnancy. I. The bearing of maternal weight gain and pre-pregnancy weight on birth weight in full term pregnancies. Obstet. Gynecol. Surv. 23:1003-1025.

Except for cases of weight loss and gains under 11 lb in pregnancy, progressive increase in weight gain was paralleled by progressive increase in mean birth weight and decrease in incidence of low-birth-weight infants. Increase in prepregnancy weight showed the same progression. These two factors act independently of each other, but when acting jointly, they become additive in their effects. It was recommended that women under 120 lb be allowed to eat to appetite to the 20th week and thereafter be considered high risk if total average gain for that period is less than 10 lb.

Emerson, R. G. 1962. Obesity and its association with the complications of pregnancy. Brit. Med. J. 5303:516-518.

The results of an investigation of 1,145 pregnant women (one out of five was regarded as overweight) suggested that obesity in pregnancy is associated with essential hypertension and preeclampsia, breech presentations that are less frequently diagnosed, antepartum hemorrhage, increased fetal loss, prolonged labor, and failure of breast feeding.

Flanagan, B. 1964. The relation of total body water to lean body mass during pregnancy in the rat. Clin. Sci. 27:335-340.

Using direct carcass analysis, the author found that rats gained fat during pregnancy proportional to the gain in lean body weight. The percentage of water rose in the second half of pregnancy, remaining constant in controls. It was concluded that the formula used to calculate lean body mass from total body water may result in an over-estimate in pregnancy.

Jacobson, H. N., B. S. Burke, C. A. Smith, and D. E. Reid. 1962. Effect of weight reduction in obese pregnant women on pregnancy, labor and delivery and on condition of the infant at birth. Amer. J. Obstet. Gynecol. 83:1609-1616.

Intensive dietary instruction given to 89 obese women who had been pregnant between 16 and 30 weeks was associated with reduced complications during pregnancy, labor, and delivery for the 47 women who lost weight. Closely supervised weight reduction during pregnancy had no demonstrable effect on the infants.

Kerr, M. G. 1962. The problem of the overweight patient in pregnancy. J. Obstet. Gynecol. Brit. Commonw. 69:988-995.

A retrospective study in which the obstetric histories of 503 obese women are compared with the histories of standard-weight women. Obese women had a higher incidence of hypertension and mild preeclampsia. They also appeared to give birth to infants over 10 lb and have prolonged labor.

Mullins, A. 1960. Overweight in pregnancy. Lancet 1:146-147.

Discusses the management and counseling of the obese pregnant woman.

Paul, G. W., and S. S. Martin. 1964. Obesity in pregnancy. J. Amer. Med. Women's Ass. 19:761-763.

Records of 1,000 patients (1961-1962) were reviewed. Incidence of prolonged labor, Cesarean section, prematurity, large babies, and toxemia were studied in 378 patients (10.9 percent, 10 to 15 years of age; 45.6 percent, 16 to 19 years of age) who were 10 lb overweight at conception or who gained more than 20 lb during pregnancy. Findings during prenatal, delivery, and postpartum periods are reported.

Pedlow, P. R. B. 1964. The significance of weight gains and losses during selected periods of pregnancy in Jamaican women. J. Obstet. Gynaecol. Brit. Commonw. 71:908-913.

The weight gained between the 20th and 30th weeks and the 30th and 36th weeks of pregnancy in a series of 500 patients is presented. The author found wide weight fluctuations among all patients, and such fluctuations made average weight-gain figures misleading.

Petry, J. A. 1956. Obesity with pregnancy. Obstet. Gynecol. 7:299-303.

In a series of 6,512 pregnant women, the obese patients showed no evidence of excessive obstetric morbidity but had larger babies than did the nonobese women.

Poidevin, L. O. S. 1960. Weight gain in pregnancy. Med. J. Austr. 47(2):324-328.

In 205 cases of toxemia and 935 controls, 24 percent of the toxemic subjects and 10 percent of the controls gained in excess of 30 lbs. No specific relationship was found between severity of toxemia and weight gained.

Sands, R. X. 1964. Intermittent modified total-fasting in the treatment of obstetric obesity. Amer. J. Obstet. Gynecol. 90:885-890.

In a study of 132 pregnant women weighing 180 to 322 lb, food intake was reduced to 900 calories per day, 6 days of the week, but on the 7th day the patient was permitted to eat whatever she liked. Of the patients who started the program, 94.7 percent continued to term, and 91.8 percent were successful in that they achieved a negative corrected weight benefit.

Sauramo, H. 1950. Overweight, underweight and mostly normal weight obstetric patients with special reference to gestosis. Acta Obstet. Gynecol. Scand. 29:360-376.

The course and outcomes of 300 parturients who weighed 90 kg or more at delivery are compared with a group of underweight and normal-weight women. The order of succession in terms of complications was (1) the overweight patients, 64 percent (especially those who were also short); (2) the underweight, 50 percent; and (3) those of normal weight, 32 percent.

Schram, M., and M. Raji. 1966. The problem of underweight pregnant patients. Amer. J. Obstet. Gynecol. 94:595-596.

Among 100 underweight maternity patients, the incidence of low-birth-weight infants was more than twice that of all deliveries; 85 percent of these births were considered full term. Incidence of toxemia was much lower.

Seitchik, J., and C. Alper. 1954. The relationship of body composition to changes in weight during pregnancy. Surg. Clin. N. Amer. 34:1535-1544.

In an effort to define limits of "normal" weight gain in pregnancy, qualitative and quantitative elements of this weight are discussed.

Singer, J. E., M. Westphal, and K. Niswander. 1968. Relationship of weight gain during pregnancy to birth weight and infant growth and development in the first year of life. Obstet. Gynecol. 31:417-423.

From an analysis of data on 10,000 children born in the Collaborative Study of Cerebral Palsy, it was found that a high gain in weight during pregnancy is related to higher birth weights and a decreased prematurity rate. The high maternal weight gain was also associated with better growth and performance of the infants during their first year of life. The evidence indicates that abandonment of the traditional obstetric practice of restricting weight gain during pregnancy might significantly reduce the incidence of prematurity and its attendant mortality and morbidity.

Smibert, J. 1967. The significance of weight gain in pregnancy as assessed by various authors. Med. J. Aust. 1:878-880.

A historical tour is made of the changing philosophy of desirable gain in weight in pregnancy. The discussion begins with a statement made in 1862 and ends with one made in 1966 by Dr. D. F. Lawson: "The greatest problem of modern medicine is to distinguish between gimmickry and real progress."

Taggart, N. R., R. M. Holliday, W. Z. Billewicz, F. E. Hytten, and A. M. Thomson. 1967. Changes in skinfolds during pregnancy. Brit. J. Nutr. 21:439-451.

Skinfold thicknesses at seven sites were measured during and after pregnancy in 84 women, and in 48, total body water was measured concurrently. At 10 weeks' gestation, skinfold measurements were highly correlated with each other and with maternal weight, ratio of observed weight for height, "dry" weight, and calculated estimates of body fat. At nearly all sites, skinfold thickness increased to 30 weeks and was greater at central sites. From 30 to 38 weeks the patterns were variable and decreased after 38 weeks to the first postpartum week. The changes were not related to the presence or absence of edema. Increase in skinfold thickness during pregnancy was greater in overweight women and in primigravidas than in multigravidas.

Thomson, A. M., and W. Z. Billewicz. 1957. Clinical significance of weight trends during pregnancy. Brit. Med. J. 5013:243-247.

The most favorable pregnancy outcome is associated with a moderate prenatal weight gain. Rigorous restriction of weight gain during pregnancy may be harmful.

Thomson, A. M., W. Z. Billewicz, B. Thompson, and I. A. McGregor. 1966. Body weight changes during pregnancy and lactation in rural African women. J. Obstet. Gynaecol. Brit. Commonw. 73:724-733.

Total weight gain in pregnant Gambian women was higher during November to August than from August to November. This difference may have been due to scarcity of food and increased hard physical work during July to October.

Thomson, A. M., and F. E. Hytten. 1960. Body stores in pregnancy and lactation. Proc. Nutr. Soc. 19:5-8.

Distribution of weight gain during pregnancy is reviewed.

Thomson, A. M., and F. E. Hytten. 1961. Calorie requirements in human pregnancy. Proc. Nutr. Soc. 20:76-83.

Normal weight gain during pregnancy, the components of the weight gained, the caloric cost of this gain, and practical caloric allowances in pregnancy are discussed.

Tompkins, W. T., D. G. Wiehl, and R. Mitchell. 1955. The underweight patient as an increased obstetric hazard. Amer. J. Obstet. Gynecol. 69:114-127.

Premature labor was inversely associated with immediate pregravid weight of the patient. It was lowest among overweight and increased sharply for underweight patients. For underweight patients, the occurrence of toxemia or symptoms increased for those who failed to gain an average amount as well as among those who gained excessively. The risk of premature labor was higher for underweight patients if gain in weight in the first two trimesters was less than average. Babies of mothers who are underweight at the onset of pregnancy are significantly lighter and shorter than babies of mothers of standard weight.

Tracy, T. A., and G. Miller. 1969. Obstetric problems in massively obese. Obstet. Gynecol. 33:204-208.

In a select group of pregnant women weighing at least 250 lb during any part of pregnancy, there was a 35 percent incidence of operative obstetrics, a 62.5 percent incidence of complications, and increased incidence of toxemia.

Widdowson, E. M. 1955. Reproduction and obesity. Amer. J. Clin. Nutr. 3:391-396.

Obesity in animals tends to reduce fertility, but in humans this relationship is not established. Obesity in human pregnancy does, however, increase the risk of complications. Obese women tend to have larger babies, and these babies tend to maintain their larger size into adult life.

Wiehl, D. G., and W. T. Tompkins. 1954. Size of babies of obese mothers receiving nutrient supplements. Milbank Mem. Fund Quart. 32:125-140.

A comparison of 235 obese women (20 percent or more overweight) with 467 women less than 5 percent above or below standard weight at the beginning of pregnancy indicated that a much larger percentage of obese women delivered late and a smaller percentage delivered early than did normal-weight women. Babies of obese women were about 7 oz heavier and 1 cm longer than those of normal-weight women. When the obese women supplemented their diets with protein and vitamin supplements, the babies weight at birth relative to skeletal size was increased, which suggests better general development.

Social, Economic, and Other Variables That Affect Pregnancy

Abramowicz, M., and E. H. Kass. 1966. Pathogenesis and prognosis of prematurity. New Engl. J. Med. 275:938-943.

The relations between (1) complications of pregnancy, nutrition, anemia, smoking, and attitude and (2) birth weight are discussed.

Ackar, S. T., and A. Yankauer. 1962. Studies on the birth weight of South Indian infants Indian J. Child. Health 11:157-167.

A comparison of birth weights of infants born to primigravidas from the lowest, middle, and highest economic groups in Madras, India, was made. The percentage of birth weights below 2,500 g was 3 times greater in the lowest economic group than in the highest.

Baird, D. 1945. The influence of social and economic factors on stillbirths and neonatal deaths. J. Obstet. Gynaecol. Brit. Emp. 52:217-234.

Stillbirth rates were highest (30.4) in infants born to mothers in the lowest social class in Aberdeen and lowest (10.0) in the highest social class. Neonatal mortality followed the same breakdown, with the highest rate for the lowest social class (34.5) and the lowest for the highest social class.

Baird, D. 1949. Social factors in obstetrics. Lancet 1:1079-1083.

A study of the social variables that affect reproductive performance.

Baird, D. 1952. The cause and prevention of difficult labor. Amer. J. Obstet. Gynecol. 63:1200-1212.

The frequency and types of difficult labor are influenced by social class. The environment in which a woman is brought up has an important influence on her reproductive performance. It affects her physical development and health, and it influences her attitude toward childbearing and the age at which she has her first child.

Baird, D. 1964. The epidemiology of prematurity. J. Pediat. 65:909-924.

A discussion of biological, environmental, obstetric, and emotional factors associated with low birth weight. Includes comments on selected diet studies.

Baird, D. 1965. Variations in fertility associated with changes in health status. J. Chron. Dis. 18:1109-1124.

A review of evidence of relationships between maternal health status and reproductive efficiency.

Baird, D., F. E. Hytten, and A. M. Thomson. 1958. Age and human reproduction. J. Obstet. Gynaecol. Brit. Emp. 65:865-876.

The ideal biological age for childbearing is 18 to 20 years. However, women who have their first pregnancy between 25 and 30 years of age usually have social and educational advantages that offset the biological advantages of earlier pregnancy.

Baird, D., and R. Illsley. 1953. Environment and childbearing. Proc. Roy. Soc. Med. 46:53-59.

The fetal mortality and prematurity rates are lowest when the mother is tall and healthy, and highest when she is small and unhealthy. The methods used for social classification of patients are described.

Chassy, J. P., A. G. van Veen, and F. W. Young. 1967. The application of social science research methods to the study of food habits and food consumption in an industrializing area. Amer. J. Clin. Nutr. 20:56-63.

Using a Guttman scale of the seven basic food groups, the authors found that with increased urbanization, as measured by educational and occupational history and other parameters, food habits or patterns changed progressively and became increasingly more complex and varied.

Christakis, G., A. Miridjanian, I. Nath, H. S. Khurana, C. Cowell, M. Archer, O. Frank, H. Ziffer, H. Baker, and G. James. 1968. A nutritional epidemiologic investigation of 642 New York City children. Amer. J. Clin. Nutr. 21:107-126.

Diet histories (24-hour recall) were obtained from 642 Puerto Rican, Chinese, Negro, and Caucasian boys and girls

10 to 13 years of age. Adequacy of diets was determined and discussed in relation to children's race, welfare status of parents, and biochemical determinations.

Collins, G. E. 1955. Do we really advise the patient? J. Fla. Med. Ass. 42:111-115.

An informal survey of 100 patients attending public health clinics indicated that many of the words commonly used in dietary advice to antepartum patients are not understood by the patients.

Council on Foods and Nutrition. 1963. Nutrition teaching in medical schools. J. Amer. Med. Ass. 183:955-957.

Report of a conference. The need to define the responsibilities and challenges of medical schools with respect to the teaching of nutrition was discussed, and a series of recommendations was made.

Day, R. L. 1967. Factors influencing of offspring, number of children, interval between pregnancies, and age of parents. Amer. J. Dis. Child. 113:179-185.

A review of the literature on survival of the fetus and child in relation to pregnancy spacing and age of parents. The importance of environmental factors is discussed.

Donabedian, A., and L. S. Rosenfeld. 1961. Some factors influencing prenatal care. New Engl. J. Med. 265:3-6.

The adequacy of prenatal care was found to be related to socioeconomic status, and the greatest neglect was found to be among groups of women of low income and limited education. Lack of participation and understanding by the recipient of the care, and lack of facilities, were cited as causative factors.

Donnelly, J. F., C. E. Flowers, Jr., R. N. Creadick, B. G. Greenberg, and H. B. Wells. 1957. Parental, fetal and environmental factors in perinatal mortality. Amer. J. Obstet. Gynecol. 74(6):1245-1256.

An examination of 279 perinatal deaths indicated that socioeconomic factors are of primary importance in perinatal mortality.

Drillien, C. M. 1957. Social and economic factors affecting incidence of premature birth. J. Obstet. Gynaecol. Brit. Emp. 64:161-185.

The social class into which the mother was born and grew up is a better predictor of prematurity than is that of her husband. The higher the class, the lower the incidence of low birth weight. Inadequate diet in the mother confers higher risk of premature delivery but not of a small full-term baby. The mothers of small full-term babies were significantly shorter than were the mothers of preterm babies.

Edwards, C. H., S. McDonald, J. R. Mitchell, L. Jones, L. Mason, A. M. Kemp, D. Laing, and L. Trigg. 1959. Clay and cornstarch eating women. J. Amer. Diet. Ass. 35:810-815.

The diets of pregnant clay and cornstarch eaters were found to be low in calories, calcium, iron, thiamin, and niacin.

Glenning, P. P. 1965. The short primigravida. Aust. N. Z. J. Obstet. Gynaecol. 5:174-177.

A higher incidence of contracted pelvis was found among women of South European origin less than 152 cm (5 feet) tall than among taller Australian women.

Gruenwald, P., H. Funakawa, S. Mitani, T. Nishimura, and S. Takeuchi. 1967. Influence of environmental factors on foetal growth in man. Lancet I:1026-1028.

Information on birth weight and gestational age was obtained from three large hospitals in Japan in the period 1945-1965. Duration of pregnancy was not increased during this period, but fetal growth curves showed a striking increase in weight for gestational age. The authors conclude that socioeconomic factors have an influence on fetal growth.

Hendricks, C. H. 1967. Delivery patterns and reproductive efficiency among groups of differing socioeconomic status and ethnic origins. Amer. J. Obstet. Gynecol. 97:608-624.

Because of the relationship consistently found between poor socioeconomic status and poor obstetric performance, the author concludes that the greatest reproductive difficulties are basically socioeconomic rather than ethnic and that the greatest challenge to successful human reproduction resides more in the patient and her community than in the physician's office or the hospital.

Jansen, A. A. J. 1962. Birthweight, birthlength, prematurity and neonatal mortality in New Guineans. Trop. Geogr. Med. 14:341-349.

A comparison of birth weights and lengths and of neonatal fatality rates of babies of different ethnic and socioeconomic backgrounds born in New Guinean hospitals revealed that those born of mothers living in more favorable circumstances, and having better nutrition and fewer diseases, were heavier at birth and had lower fatality rates. The author recommends that the prematurity standard for native newborns be lowered to 2,250 g.

Jones, N. W. 1954. The production of change in a pre-natal clinic--some influential factors. Hum. Organ. 12:21-26.

Factors that influenced success in modifying the diets of a group of low-income patients seen in a prenatal clinic in a southern industrial city were economic means, level of intelligence and schooling, living arrangements, and the existing dietary pattern. Improvements in the quality of diets resulted when changes were introduced gradually and took account of these factors.

Kane, S. H. 1964. Significance of prenatal care. Obstet. Gynecol. 24:66-72.

A study to determine whether there is a relationship between prenatal care and outcome of pregnancy. Previous pregnancy experience was found to be one of the major factors in infant survival, and prenatal care did not materially influence infant survival except in first pregnancies.

387-735 O - 70 - 12

Kark, E. 1956. Puberty in South African girls: II. Social class in relation to the menarche. S. Afr. J. Clin. Med. 2:84-88.

Social class and nutritional status were found to affect the age of menarche.

Llewellyn-Jones, D. 1965. The effect of age and social status on obstetric efficiency. J. Obstet. Gynaecol. Brit. Commonw. 72:196-202.

The factors of age and social status in relation to obstetric performance were compared in primigravidas of high and low social classes attending a Malayan hospital. The effect of increasing age on obstetric performance is considerable. However, the advantages of better nutrition and more skillful obstetric care, found in higher socio-economic groups, offset the obstetric risk incurred with increasing age.

Mayer, J. 1965. The nutritional status of American Negroes. Nutr. Rev. 23:161-164.

A discussion (based on interviews and correspondence) of the food habits of Negroes of low socioeconomic status living in rural and urban areas, and of factors that affect these habits.

Pakter, J. H., H. J. Rosner, H. Jacobziner, and F. Greenstein. 1961. Out-of-wedlock births in New York City. I. Socio-logic aspects. II. Medical aspects. Amer. J. Public Health 51:683-696, 846-865.

A comprehensive analysis of the medical and sociological aspects of all out-of-wedlock births that occurred in New York City from 1955 to 1959.

Perkin, G. W. 1968. Assessment of reproductive risk in nonpregnant women. Amer. J. Obstet. Gynecol. 101:709-713.

Includes a discussion of evidence of a relationship between (1) age, parity, medical and obstetric history, birth interval, and nutritional and socioeconomic status and (2) maternal and infant mortality and morbidity.

Proceedings of the Seventh International Conference of the International Society of Geographical Pathology, London, June 28-30, 1960. Pathol. Microbiol. 24:428-556.

Epidemiological reports from various countries on the incidence of toxemias and factors thought to affect the rate are presented.

Reeder, S. J., and L. G. Reeder. 1964. Some correlates of prenatal care among low income wed and unwed women. Amer. J. Obstet. Gynecol. 90:1304-1314.

This is part of a large study in which prenatal health habits and characteristics of wed and unwed mothers are explored.

Schneider, J. 1967. Fetal wastage: A survey of unsuccessful pregnancy. Univ. Mich. Med. Cent. J. 33:110-113.

Fetal death, infant and neonatal death, and low-birth-weight infants are discussed. The importance of better education, improved nutrition, and general well-being is emphasized.

Theobald, G. W. 1965. Reproductive ability in woman. Amer. J. Obstet. Gynecol. 92:332-340.

Superior reproductive ability of Chinese women in Hong Kong was compared with the reproductive ability of low-income maternity patients in England and Madras, India. Roles of maternity care, socioeconomic status, and reproductive efficiency are discussed.

Thompson, J. F. 1968. Some observations on the geographic distribution of premature births and perinatal deaths in Indiana. Amer. J. Obstet. Gynecol. 101:43-52.

The relation between reproductive performance and economic and demographic characteristics was studied. In an urbanized, industrialized, metropolitan county, prematurity and perinatal mortality were associated with poverty, poor housing, and relative population crowding. Thinly populated rural areas had the lowest prematurity and perinatal mortality rates. The factor that was consistently associated with poor reproductive outcome was population crowding.

Thomson, A. M., and W. Z. Billewicz. 1963. Nutritional status, maternal physique and reproductive efficiency. Proc. Nutr. Soc. 22:55-61.

Tall women generally have superior nutritional status and reproductive efficiency when compared with short women. The authors stress that nutritional preparation for pregnancy begins in the mother's infancy.

Thomson, A. M., W. Z. Billewicz, and R. M. Holliday. 1967. Secular changes in the physique of Aberdeen mothers, 1950-1954. Brit. J. Prev. Soc. Med. 21:137-140.

Height and weight records of Aberdeen primigravidas for the years 1959 to 1964 were examined for the presence of secular trends. Primigravidas under 25 years of age showed a trend toward increased height. Body weight at 20 weeks' gestation showed an upward trend in all age groups.

Thomson, A. M., D. Chun, and D. Baird. 1963. Perinatal mortality in Hong Kong and in Aberdeen, Scotland. J. Obstet. Gynaecol. Brit. Commonw. 70:871-877.

The incidence of stillbirths, perinatal mortality, and pre-eclampsia was found to be lower in Chinese mothers in Hong Kong than in a comparable European population.

Timonen, S., U. Uotila, P. Kuusisto, P. Vara, and O. Lokki. 1966. Effect of certain maternal, foetal and geographical factors on the weight and length of the newborn and on the duration of pregnancy. Ann. Chir. Gynaecol. Fenn. 55:196-213.

Such factors as maternal age, parity, weight and sex, and marital status, which influence measurements of the newborn and the duration of pregnancy, are analyzed.

Udani, P. M. 1963. Physical growth of children in different socio-economic groups in Bombay. Indian J. Child Health 12:594-611.

Birth weights, heights, and weights of 5,540 children in low, middle, and high socioeconomic groups in Bombay were

compared. A definite inverse relationship of the measures with socioeconomic status was found.

Woolf, B., and J. Waterhouse. 1945. Studies on infant mortality; influence of social conditions in country boroughs of England and Wales. J. Hyg. 44:67-98.

The influence of social conditions in England and Wales on infant deaths was studied.

Yankauer, A., K. G. Goss, and S. M. Romeo. 1953. An evaluation of prenatal care and its relationship to social class and social disorganization. Amer. J. Public Health 43:1001-1010.

The characteristics of women who neglected to seek care until late pregnancy, and the outcomes of their pregnancies, are compared with those of women of similar socioeconomic status who received adequate prenatal care. Attention is drawn to the adverse effect on the fetus of maternal nutrition and pattern of living in the former.

The Toxemias

Aboul-Khair, S. A., E. Turnbull, A. C. Turnbull, and J. Crooks. 1968. Effects of pre-eclampsia on the change in iodine metabolism during pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 75:1040-1044.

In 35 pregnant women with pre-eclampsia, renal clearance of iodine was reduced in women with moderate or severe pre-eclampsia.

Adams, E. M., and A. Finlayson. 1961. Familial aspects of pre-eclampsia and hypertension in pregnancy. Lancet 7210(7218):1375-1378.

The findings suggest a strong familial tendency to pre-eclampsia and hypertension in pregnancy.

American Committee on Maternal Welfare. 1935. The management of preeclamptic toxemia and eclampsia. J. Amer. Med. Ass. 104:1703-1705.

An adequate diet with reduced amounts of carbohydrates, fats, and salt to prevent toxemia and severe reduction of animal protein, salt, and fluid as a treatment for toxemia is recommended.

Bonar, J., J. J. Brown, D. L. Davies, H. G. Langford, A. F. Lever, and J. I. S. Robertson. 1966. Plasma renin concentration in American Negro women with hypertensive disease of pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:418-420.

Plasma renin concentration in nine American Negro women with severe hypertensive disease of pregnancy varied but was significantly lower than that found in normal pregnancy.

Brewer, T. H. 1966. Good prenatal nutrition prevents toxemia of late pregnancy. Postgrad. Med. 39A:119-124.

No toxemia of late pregnancy developed among 235 clinic patients of low socioeconomic status who received a lecture at their first prenatal visit that stressed the values of a diet high in protein. No restriction of gain in weight or of salt was advocated.

Brown, J. J., D. L. Davies, P. B. Doak, A. F. Lever, and J. I. S. Robertson. 1966. Plasma renin concentration in the hypertensive diseases of pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 73:410-417.

Includes a review of theories on etiology of toxemias of pregnancy.

Bryans, C. I., Jr. 1966. The remote prognosis in toxemia of pregnancy. Clin. Obstet. Gynecol. 9:973-990.

The relationship of pre-eclampsia and eclampsia to chronic vascular (essential) hypertension was studied in 168 Caucasian and 167 Negro women with a history of eclampsia. The incidence of hypertension was not significantly different from that of controls.

Chakravorty, A. P. 1967. Foetal and placental weight changes in normal pregnancy and pre-eclampsia. J. Obstet. Gynaecol. Brit. Commonw. 74:247-253.

In 200 cases of pre-eclamptic toxemia, there was a significant reduction in mean fetal weight when compared with fetal weight of normal pregnancies. No difference in placental weights was found. In the majority of pre-eclamptics, the placental-fetal weight ratio was higher than in the normal pregnancy group.

Chesley, L. C. 1958. The renal excretion of sodium in women with pre-eclampsia. Clin. Obstet. Gynecol. 1:317-323.

Discusses sodium excretion in relation to toxemia of pregnancy, and suggests that pre-eclamptics excrete less sodium than do normal pregnant women, and that this excessive sodium reabsorption occurs in the distal portion of the nephron. Increased tubual reabsorption also appears to be present in pre-eclampsia.

Chesley, L. C. 1961. Renal function in relation to salt and water regulation in normal and toxemic pregnancy. Pathol. Microbiol. 24:631-638.

A review of pertinent work on hydration and sodium retention in normal compared with pre-eclamptic pregnancies. Concludes that normal pregnancy is associated with slight hydration of maternal tissues and little sodium retention compared with pre-eclampsia and that the pre-eclamptic state, because of altered renal function, may represent new aberrations rather than an extension of normal changes in pregnancy.

Chesley, L. C. 1966. Sodium retention and pre-eclampsia. Amer. J. Obstet. Gynecol. 95:127-132.

Reviews evidence on sodium retention in preeclampsia and discusses the divergent views held by various investigators. Concludes that sodium retention is a characteristic of pre-eclampsia and eclampsia.

Chesley, L. C., J. E. Annitto, and R.A. Cosgrove. 1968. The familial factor in toxemia of pregnancy. Obstet. Gynecol. 32:303-311.

A follow-up study of daughters and granddaughters of 268 women known to have had eclampsia revealed that about one fourth developed toxemia during their first pregnancies. The authors conclude that preeclampsia and eclampsia "run in families" and suggest that some factor other than a familial predisposition to essential hypertension may be involved. They suggest that some common environmental factor, such as a poor diet, may be operant.

Chesley, L. C., J. E. Annitto, and R. A. Cosgrove. 1968. Long-term follow-up study of eclamptic women. Amer. J. Obstet. Gynecol. 101:886-898.

In a study of the relation between preeclampsia and eclampsia and subsequent chronic hypertension, 268 women surviving eclampsia between 1931 and 1951 were traced to 1966 and examined. The authors concluded that eclampsia does not cause chronic hypertension, and posteclamptic women with hypertension either had it before pregnancy or would have developed it even if never pregnant.

Chesley, L. C., and E. R. Chesley. 1943. An analysis of some factors associated with the development of preeclampsia. Amer. J. Obstet. Gynecol. 45:748-761.

Thiocyanate available water (extra cellular) was measured in 1,388 women in the last 10 weeks of pregnancy. The incidence of preeclampsia was 6 times higher in women with excessive available water than in those with normal amounts. When excessive available water and lowered serum proteins were found together, an increased predisposition to preeclampsia was noted. Body weight, pattern of weight gain, blood pressure, and rate of gain of available water were discussed.

Chesley, L. C.,R. A. Cosgrove, and J. E. Annitto. 1961. Pregnancy in the sisters and daughters of eclamptic women. Pathol. Microbiol. 61:662-666.

The incidence of toxemia of pregnancy approached 40 percent in both the sisters and daughters of 98 women who

had eclampsia. This familial tendency to toxemia was not related to hypertensive diathesis.

Clemetson, C. A. B., and L. Andersen. 1964. Ascorbic acid metabolism in preeclampsia. Obstet. Gynecol. 24:774-782.

Analysis of fasting blood samples from 19 normal pregnant women and 11 women with mild to moderate preeclampsia showed a significantly lower ratio of reduced to oxidized ascorbic acid in the plasma of women with preeclampsia.

Craig, J. M. 1969. Models for obstetrical and gynecological disease. Fed. Amer. Soc. Exp. Biol. 28:206-210.

Because of the unique characteristics of human reproduction, few satisfactory research models exist outside the primate. The author includes a discussion and references to animal models that have been used for research in various aspects of toxemia of pregnancy.

Dalderup, L. M. 1959. Atherosclerosis and toxemia of pregnancy in relation to nutrition and other physiological factors. Vitamin Horm. 17:223-306.

Comparison was made of a number of factors that seem to influence the incidence of atherosclerosis and toxemia. From available data, the author concluded that conditions of life and food constituents induce shifts in the frequency of both diseases.

Davey, D. A., and W. J. O'Sullivan. 1961. Sodium metabolism in normal pregnancy and pre-eclampsia. Pathol. Microbiol. 24:642-651.

In five preeclamptic patients, with a rise in blood pressure together with edema or albuminuria (or both), there was an increase in absolute amounts of exchangeable sodium and total exchangeable sodium between the 16th and 26th weeks of pregnancy, followed by a decrease to the time the signs of preeclampsia occurred. These changes were the direct opposite of those found in normal pregnant women.

de Bacalao, E. B., H. Kaunitz, J. Joseph, and D. G. McKay. 1964. Lipid metabolism in toxemia and normal pregnancy. Obstet. Gynecol. 24:909-913.

The rise in total serum lipid was less pronounced in women with preeclampsia or hypertension than in normal pregnant women. Total lipid and arachidonic acid were elevated in the placentas of preeclamptic women.

Diding, N. A., and S. E. Melander. 1961. Serum vitamin B_6 levels in normal and toxaemic pregnancy. Acta Obstet. Gynecol. Scand. 40:252-261.

Describes a microbiological assay in which a pyridoxine-auxotroph strain of E. coli was used as the test organism. No significant difference between the serum levels was found for healthy men, nonpregnant women, women in the last trimester of uncomplicated pregnancy, and women with toxemia of pregnancy. The toxemic women had lower, but not significantly different, serum levels. The alimentary supply of pyridoxine was not controlled. The diet high in B complex vitamins and multivitamin tablets seemed sufficient to maintain serum levels in pregnant women.

Dodge, E. F., and T. T. Frost. 1938. Relation between blood plasma proteins and toxemias of pregnancy. J. Amer. Med. Ass. 111:1898-1902.

In patients with mild toxemia, an increased intake of protein was well tolerated and diminished objective and subjective symptoms.

Douglas, B. H., and H. G. Langford. 1966. Toxemia of pregnancy, production of lesions in the absence of signs. Amer. J. Obstet. Gynecol. 95:534-537.

A vitamin E-deficient diet containing substantial amounts of partially peroxidized polyunsaturated fatty acids was fed to pregnant rats. Tissue changes similar to those seen in toxemia of pregnancy developed in the rats. Hypertension, proteinuria, and edema were not present.

Eastman, N. J. 1930. Serum proteins in toxemias of pregnancy. Amer. J. Obstet. Gynecol. 19:343-351.

The average albumin-globulin ratios in the blood of normal nonpregnant women, normal gravidas, and women with eclampsia or preeclampsia were calculated to be 1.7, 1.6, and 1.3, respectively. With persistent albuminuria, total serum protein fell between 4.0 and 5.0 g/100 ml of blood. Such alteration was believed to play an important role in the production of edema associated with toxemias of pregnancy.

Editorial. 1917. Eclampsia rare on war diet in Germany. J. Amer. Med. Ass. 68:732.

The restriction of fat and meat in Germany during the war was postulated as the cause of the decreased rate of toxemia.

Finnerty, F. A. 1966. Treatment of mild toxemia. Clin. Obstet. Gynecol. 9(4):944-953.

Recommends a restricted sodium diet and thiazide for pregnant women who retain sodium. The treatment should be prompt and continuous.

Fish, J. S., R. A. Bartholomew, E. D. Colvin, W. H. Grimes, W. M. Lester, and W. H. Galloway. 1959. The relationship of pregnancy weight gain to toxemia. Amer. J. Obstet. Gynecol. 78:743-754.

In a group of 1,000 pregnant women, an analysis was made of the association between the pattern of gain in weight and the appearance of toxemia. Data appear to indicate that type or amount of excessive weight gain does not predispose patients to the development of toxemia, except in some instances of gains of 4 pounds or more in a week.

Fisher, J. J., and I. Frey. 1958. Pregnancy and parturition in the obese patient. Obstet. Gynecol. 11:92-94.

In 100 obese pregnant women of different parities, the incidence of hypertension, toxemia, and prolonged labor was higher than in normal-weight controls.

Gibson, G. B., and R. Platt. 1959. Incidence of hypertension after pregnancy toxemia. Brit. Med. J. 2:159-163.

The mean blood pressures of women about 4 years after toxemic pregnancy were higher than the mean expected pressures of women of similar age. Severe hypertension, however, was a rare sequal of toxemic pregnancy in women who were previously normotensive.

Gordon, R. D., S. Parsons, and E. M. Symonds. 1969. A prospective study of plasma-renin activity in normal and toxaemic pregnancy. Lancet I(7590):347-348.

From a study of apparently healthy women between the 13th and 27th weeks of pregnancy, the authors conclude that an elevated renin activity is an etiological factor in development of toxemia of pregnancy. The more striking the elevation is in early pregnancy, the greater the likelihood is of developing toxemia later on.

Holmes, O. M. 1941. Protein diet in pregnancy. West J. Surg. 49:56-60.

The incidence of toxemia in 350 primigravidas and 350 multigravidas ingesting low-protein diets was twice as great as the incidence in similar groups receiving high-protein diets.

Hytten, F. E., and A. M. Thomson. 1966. Body water in pre-eclampsia. J. Obstet. Gynaecol. Brit. Commonw. 73:714-716.

Total body water was measured in six women who developed preeclampsia, as indicated by a rise in blood pressure, proteinuria, and some edema. The amount of water gained was within the limits found in normal women, but in the last 10 weeks of pregnancy the rate of gain of water was faster than in normal women with generalized edema.

Interim Report of the People's Health League. 1942. Nutrition of expectant and nursing mothers. Lancet 2:10-12.

The incidence of toxemia and prematurity was reduced in the approximately 2,500 women receiving supplementary minerals and vitamins when compared with the incidence in an equal number of control women.

Jeffcoate, T. N. A. 1966. Pre-eclampsia and eclampsia: The disease of theories. Proc. Roy. Soc. Med. 59:397-404.

The author believes that the difference between a normal pregnancy and one complicated by toxemia lies in the response of the maternal organism to the normal process of pregnancy. The influence of nutritional status in this response remains obscure.

Jeffcoate, T. N. A., and J. S. Scott. 1959. Some observations on the placental factor in pregnancy toxemia. Amer. J. Obstet. Gynecol. 77:475-489.

A review of several aspects of the relationship of the placenta to toxemia. The authors suggest that the quest for a single cause of toxemia is likely to meet with little success, and that the syndromes of preeclampsia and eclampsia will be found to be more complex than thought in 1959.

Johnson, T., and C. G. Clayton. 1957. Diffusion of radioactive sodium in normotensive and pre-eclamptic pregnancies. Brit. Med. J. I:312.

In preeclamptic pregnant women, the rate of disappearance of ^{24}Na from an injection site in the myometrium was significantly lower than in normally pregnant women. The absorption rate in eclamptics was increased by the injection of hydrallazine hydrochloride.

Johnson, W. L., H. S. McGaughey, and W. N. Thornton. 1961. Serum sodium and total solute concentrations in normal and toxic pregnancy. Surg. Forum. 12:422-424.

From an investigation of 1,249 pregnant women in the latter half of pregnancy, divided into normal, toxic, and borderline categories on the basis of blood pressure and clinical criteria, it was concluded that the depression of serum sodium and total solute concentration in pregnancy were physiological and not major factors in the pathophysiology of toxemia.

Jones, E. M. 1968. Capillary permeability to plasma proteins during pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 75:295-299.

A study to determine whether changes in capillary permeability to plasma proteins occur in normal pregnancy and in pregnant patients with hypertensive disorders and whether such changes relate to the occurrence of edema. The observations indicate that in normal pregnancy and in preeclampsia, protein loss from forearm capillaries is not increased above that seen in normal nullipara; this suggests that there is no increase in capillary permeability to plasma proteins.

Klieger, J. A., J. R. Evrard, and R. Pierce. 1966. Abnormal pyridoxine metabolism in toxema of pregnancy. Amer. J. Obstet. Gynecol. 94:316-321.

Placental tissue from 43 women, normal or toxemic was extracted, and pyridoxine derivatives were estimated microbiologically in the extracts. The placentas of toxemic women were characterized by marked pyridoxine deficiency. The continued investigation of pyridoxal kinase activity in patients with toxemia is indicated.

Konttinen, A., T. Pyorala, and E. Carpen. 1964. Serum lipid pattern in normal pregnancy and pre-eclampsia. J. Obstet. Gynecol. Brit. Commonw. 71:453-458.

Serum lipids were studied late in pregnancy, at delivery, and during early puerperium in 28 normal pregnant women and 19 women with preeclampsia. Serum total cholesterol, phospholipids, and serum triglycerides were higher in pregnant women than in nonpregnant women. No significant difference was noted in the serum lipids of preeclamptic women when compared with those of normal pregnant women.

Krokfors, E., and L. Tervilä. 1966. Correlation of the nature of toxemia of late pregnancy to the birthweight of the child and to maternal weight increase during pregnancy. Ann. Chir. Gynaecol. Fenn. 55:289-295.

In a series of 3,000 consecutive parturients, the authors found that maternal constitution influences weight increase in pregnancy and the birth weight of the child. Maternal edema of the mother tends to increase birth weight, and hypertension tends to decrease birth weight.

Krokfors, E., and L. Tervilä. 1966. Correlation of the standard of living to the nature of toxaemia of late pregnancy. Ann. Chir. Gynaecol. Fenn. 55:219-226.

Comparison of 1,638 parturients with toxemia of late pregnancy with an equal number of healthy parturients was made to determine whether correlations exist between incidence of proteinuria, edema, hypertension, and the parturients' standard of living. Hypertension was more prevalent among those living in less favorable conditions.

Krokfors, E., and L. Tervilä. 1966. Influence of the maternal constitution on the nature of toxaemia of late pregnancy. Ann. Chir. Gynaec. Fenn. 55:227-232.

Maternal constitution was classified by height and pre-pregnancy weight. Analysis of data on 11,000 patients showed decrease in incidence of toxemia with increasing height. The highest incidence was among those of short stature and heavy body weight. It is believed that obese women have a tendency to impaired circulation, especially weakness of the capillary system, and that these women tend to have more renal insufficiency.

Kuhlbäck, B., and O. Widholm. 1964. Serum uric acid in toxemia of pregnancy with special reference to the prognosis of the fetus. Acta Obstet. Gynecol. Scand. 43:330-337.

The uric acid concentration in the serum of 72 normal pregnant women and 92 pregnant women with varying degrees of toxemia revealed a marked elevation of serum uric acid levels in toxemic women.

Kulkarni, B. S., R. G.Chitre, and M. N. Parikh. 1960. Electrophoretic study of the serum proteins during normal pregnancy, labour, puerperium and toxemia of pregnancy. Indian J. Med. Sci. 14:689-694.

In 166 pregnant Indian women of low socioeconomic status, total globulins rose and the gamma globulin level remained unchanged. In 58 cases of preeclampsia and eclampsia, there was a significant reduction in the albumin and total protein values. A marked drop in total protein content of anemic women was attributed to a drop in albumin level.

Little, B. 1964. Treatment of pre-eclampsia. New Engl. J. Med. 270:94-96.

The characteristics, prevention, and treatment of pre-eclampsia are discussed.

Lowe, C. R. 1961. Toxaemia and pre-pregnancy weight. J. Obstet. Gynaecol. Brit. Commonw. 68:622-627.

Hospital records of 2,042 women investigated shortly after delivery indicated no significant relationship between recorded toxemia or preeclamptic toxemia and body build. However, diastolic pressure of 90 mm or higher was related to obesity.

McCartney, C. P. 1966. Toxemia of pregnancy--classification. Clin. Obstet. Gynecol. 9:(4):864-869.

Among subjects assessed by renal biopsy, the incidence of chronic renal disease was 25 percent in primigravidas with preeclampsia and 21 percent in multigravidas with chronic hypertensive renal vascular disease and superimposed acute toxemia.

McCartney, C. P., R. E. Pottinger, and J. P. Harrod, Jr. 1959. Alterations in body composition during pregnancy. Amer. J. Obstet. Gynecol. 77:1038-1053.

In a study of gross body composition, sodium-22 space, and exchangeable sodium determined in normal and abnormal pregnancies, women with preeclampsia and eclampsia evidenced marked antepartum and postpartum increases in the proportion of exchangeable sodium contained in the fat-free bodies.

MacGillivary, I. 1961. Salt and water balance in normal and toxaemic pregnancy. Pathol. Microbiol. 24:639-641.

By measuring total exchangeable sodium, total body water, and total exchangeable chloride, it was found that the retention of water in preeclamptic women is not accompanied by a comparable retention of sodium or chloride.

MacGillivary, I., and T. J. Buchanan. 1958. Total exchangeable sodium and potassium in non-pregnant women and in normal and pre-eclamptic pregnancy. Lancet 2(7056): 1090-1093.

The results of a study of sodium and potassium by the isotope-dilution technique indicate that the amount of sodium retained in preeclampsia is the same as in normal pregnancy, but in preeclampsia there is greater water retention. Potassium is also discussed.

MacGillivary, I., and D. A. Davey. 1963. Hypertension in late pregnancy. Biochem. Clin. 2:299-308.

The probable multifactorial causes of hypertension, the difficulties of determining the normal response of blood pressure to pregnancy, and the probable mechanisms involved in the production of hypertension in late pregnancy are discussed.

McIlroy, L., and H. E. Rodway. 1937. Weight changes during and after pregnancy with special reference to the early diagnosis of toxaemia. J. Obstet. Gynaecol. Brit. Emp. 44:221-244.

Women with toxemia of pregnancy gained 50 percent more weight between the 24th and 38th weeks of pregnancy, and 3 times more during the last 2 weeks, than did normal pregnant controls. Exercise and a low carbohydrate diet were recommended for these women.

Mack, H. C., H. J. Kelly, and I. G. Macy. 1956. Complications of pregnancy and nutritional status. I. Toxemias of pregnancy. Amer. J. Obstet. Gynecol. 71:577-595.

Data are presented on 54 pregnancies complicated by preeclampsia and eclampsia. The data are based on clinical observations, medical histories, food intakes, and microchemical determinations.

Mahran, M. 1961. Water and electrolyte metabolism in pregnant women. Pathol. Microbiol. 24:647-651.

In 26 preeclamptic women, the water turnover was lower than in normal pregnant and nonpregnant women, and there

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was an abnormal retention of exchangeable sodium. Measuring water turnover was discussed as a method of detecting preclinical preeclamptic toxemia.

Maqueo, M., J. C. Azuela, M. Dosal de la Vega. 1964. Placental pathology in eclampsia and preeclampsia. Obstet. Gynecol. 24:350-356.

A morphological study of 162 placentas of women with toxemia of pregnancy, from mild to eclamptic, and of 55 women with normal gestation, revealed a much higher frequency of pathological alterations in the toxemic placentas, but the authors believe that these lesions play no role in the etiology of toxemia.

Mengert, W. F., and D. A. Tacchi. 1961. Pregnancy toxemia and sodium chloride. Amer. J. Obstet. Gynecol. 81:601-605.

Forty-eight consecutively admitted patients with acute vasospastic toxemia of pregnancy were alternately given diets with 10.0 to 11.5 g of sodium chloride and 0.9 to 1.7 g of sodium chloride daily. There was no discernible effect on the clinical course of toxemia.

Naeye, R. L. 1966. Abnormalities in infants of mothers with toxemia of pregnancy. Amer. J. Obstet. Gynecol. 95:276-283.

Study of organ and cellular development of 11 infants with fetal growth retardation associated with maternal toxemia of pregnancy revealed disproportionately small size of adrenals, liver, spleen, and thymus. The small size was due to a subnormal amount of cytoplasm rather than a deficient number of cells.

Nelson, G. H. 1966. Lipid metabolism in toxemia of pregnancy. Clin. Obstet. Gynecol. 9(4):882-894.

The alterations in maternal serum lipids found in toxemia of pregnancy are reviewed. Two studies that show statistically significant elevations in placental triglycerides in toxemia are reported.

Nelson, G. H., F. P. Zuspan, and L. T. Mulligan. 1966. Defects of lipid metabolism in toxemia of pregnancy. Amer. J. Obstet. Gynecol. 94:310-315.

Phospholipid, total and free cholesterol, triglyceride, and unesterified fatty acids were estimated in maternal and fetal serum and placental tissue immediately after birth in 12 normal pregnant women and compared with those of 10 women with toxemia.

Ojanen, R., E. Keskitalo, and E. Hirsjarvi. 1965. Acid-base status in late toxemia of pregnancy. Ann. Chir. Gynaecol. Fenn. 54:347-350.

A study of the acid-base status of 22 healthy pregnant women and of 62 patients suffering from toxemia.

O'Leary, J. A., G. S. Novalis, and G. J. Vosburgh. 1966. Maternal serum copper concentrations in normal and abnormal gestations. Obstet. Gynecol. 28:112-117.

Average serum copper concentration was higher in a small sample of pregnant women with preeclampsia than in 175 normal pregnant women. Normal pregnant women had higher values than did nonpregnant women.

O'Rourke, D. E., J. G. Quinn, J. O. Nicholson, and H. H. Gibson. 1967. Geophagia during pregnancy. Obstet. Gynecol. 29:581-584.

Among 200 randomly selected obstetrical patients, the incidence of geophagia was 55 percent. The occurrence of toxemia was noted more than twice as often in the pica group, although no apparent adverse effects resulted in the newborn. The mean consumption of clay was 50 g/day and laundry starch 120 g/day. Some patients ate dry milk of magnesia, coffee grounds, and paraffin. There is little information about this practice in the literature, and it may be more widespread than realized.

Pike, R. L., J. E. Miles, and J. M. Wardlaw. 1966. Juxtaglomerular degranulation and zona glomerulosa exhaustion in pregnant rats induced by low sodium intakes and reversed by sodium load. Amer. J. Obstet. Gynecol. 95:604-614.

Data are presented that suggest that juxtaglomerular degranulation occurs when the stress of pregnancy is combined with low sodium intake in rats. ·Administration of a sodium load 4 days prior to term reverses the histological and biochemical signs of sodium depletion. Relevance to sodium intake in human pregnancy is discussed.

Plentl, A. A., and M. J. Gray. 1959. Total body water, sodium space, and total exchangeable sodium in normal and toxemic women. Amer. J. Obstet. Gynecol. 78:472-478.

Distribution of water and total exchangeable sodium during the third trimester of pregnancy was studied in an attempt to establish a normal range. Values obtained were compared with those of toxemic women of comparable gestational age.

Pritchard, J. A. 1961. Toxemia of pregnancy. Postgrad. Med. 30:407-411.

Definition of terms, description of potential victims of toxemia, and discussion of the treatment of edema, hypertension, and eclamptic convulsions are included.

Pystynen, P., and P. Pankamaa. 1965. Diurnal variations in urine excretion, and sodium and potassium excretion in healthy and toxaemic women in late pregnancy. Acta Obstet. Gynecol. Scand. 44:408-415.

The diurnal cycle of urine excretion and the excretion of potassium and sodium in normal pregnant women and in those with hypertension, mild preeclampsia, or severe preeclampsia in the last month of pregnancy was investigated. The rhythm of excretion was similar in all groups.

Quigley, H., L. L. Phillips, and D. G. McKay. 1965. Transport of radioactive sodium across the rat placenta in experimental toxemia of pregnancy. Amer. J. Obstet. Gynecol. 91:377-383.

Placental permeability of rats in the third trimester of pregnancy was evaluated by determination of radioactivity of placenta and fetus following intravenous injection of

labeled sodium chloride. Rats that were fed a low vitamin E diet containing oxidized cod liver oil during pregnancy had a lower placental permeability to sodium ion in the 15th through 17th days of gestation than did control animals. Near term this trend was reversed. A toxic effect of the oxidized cod liver oil diet on the trophoblast is postulated as the etiology of diet-induced experimental toxemia of pregnancy.

Quinto, P., F. Bottiglioni, and C. Flamigni. 1966. Metabolic studies in toxaemic, obese, and diabetic pregnant women. J. Obstet. Gynaecol. Brit. Commonw. 74:556-562.

A study was made of the modifications of carbohydrate and lipid metabolism complicated by toxemia in four women, diabetes mellitus in four women, and obesity in ten women. Differences in the metabolic patterns were compared with changes in normal pregnant women.

Ross, R. A., W. A. Perlzweig, H. M. Taylor, A. McBryde, A. Yates, and A. A. Kondritzer. 1938. A study of certain dietary factors of possible etiologic significance in toxemias of pregnancy. Amer. J. Obstet. Gynecol. 35:426-440.

In a study of 54 primiparas, the addition of dried milk, vitamins, calcium, and iron to a "marginal" diet did not affect the incidence of toxemic symptoms.

Scrimshaw, N. S. 1947. Toxic complications of pregnancy in Gorgas Hospital, Panama Canal Zone, 1931-1945; analysis of 10,000 pregnancies. Amer. J. Obstet. Gynecol. 54:428-444.

An analysis of 10,000 pregnancies revealed differences in the incidence of preeclampsia and eclampsia among Panamanian, West Indian Negro, and American residents. The higher incidence in Negroes could not be accounted for by racial, dietary, therapeutic, or climatic factors. Adverse social and psychological factors that affect the Negroes to a greater degree than the other population groups were suggested as an important influence.

Siddall, R. S., and H. C. Mack. 1933. Weight changes in the last four months of pregnancy. Amer. J. Obstet. Gynecol. 26:244-249.

Excessive weight gain was noted in the majority of patients with late toxemia of pregnancy, but it occurred with the same frequency in normal pregnancy. The clinical value of weight changes for detection of impending toxemia is questionable.

Sprince, H., R. S. Lowy, C. E. Folsome, and J. S. Behrman. 1951. Studies on the urinary excretion of xanthurenic acid during normal and abnormal pregnancy: A survey of the excretion of xanthurenic acid in normal nonpregnant, normal pregnant, preeclamptic and eclamptic women. Amer. J. Obstet. Gynecol. 62:84-92.

Data are presented to show that preeclamptic and eclamptic patients after ingestion of a 10-g dose of DL-tryptophan excreted much larger amounts of xanthurenic acid in the urine than did normal nonpregnant and normal pregnant women under the same conditions.

Stamler, F. 1959. Fatal eclamptic disease of pregnant rats fed anti-vitamin E stress diet. Amer. J. Pathol. 35:1207-1231.

A fatal condition of pregnant rats receiving an anti-vitamin E stress diet is reported. Regimen produces a normal gestation pattern until the abrupt onset of symptoms that lead to maternal fatality in the terminal phase of pregnancy. This disorder is often culminated in eclamptic symptoms with clinical and pathological aspects comparable with eclampsia in human pregnancy. α-tocopheral, anti-oxidants, and polyvitamin supplements offered partial or complete protection.

Stevenson, R. B. C. 1958. The prevention of eclampsia and severe pre-eclampsia. J. Obstet. Gynaecol. Brit. Emp. 65:982-987.

The incidence of eclampsia was greatly reduced by a regime of antenatal care that included a high-protein diet, careful regulation of gain in weight, and close observation.

It was concluded that preconceptual obesity and greater rate of gain in weight in pregnancy predispose to toxemia.

Stewart, A., and D. Hewitt. 1960. Toxaemia of pregnancy and obesity. J. Obstet. Gynaecol. Brit. Emp. 67:812-818.

Body build indices based on prepregnancy and postpregnancy weight records for 2,522 mothers indicated that the incidence of toxemia was higher than average among mothers overweight for their height and lower than average among mothers who were underweight for height.

Strass, M. B. 1935. Observations on the etiology of the toxemias of pregnancy. Amer. J. Med. Sci. 190:811-824.

Expresses the belief that restricted dietary intake in pregnancy is harmful and that high protein diets, along with vitamin B complex administration, have beneficial results for toxemic women.

Tenney, B., and R. V. Dandrow. 1961. Clinical study of hypertensive disease in pregnancy. Amer. J. Obstet. Gynecol. 81:8-15.

From clinical experience, the authors suggest that essential hypertension in pregnancy is a benign complication. When toxemia is superimposed on hypertension, however, the disease is more acute than pre-eclampsia alone. Sodium restriction and careful control of weight are recommended as prophylaxis.

Tervilä, L., and E. Krokfors. 1966. Correlation of nature of toxaemia of pregnancy to perinatal mortality and underweight of newborn. Ann. Chir. Gynaecol. Fenn. 55:109-111.

Underweight and perinatal mortality of the child were studied in relation to maternal toxemia of late pregnancy in a series of 3,336 deliveries. Elevation in blood pressure (145/90 mm Hg) was related to a higher than average perinatal mortality and low birth weight. Edema had no effect, but proteinuria was found to affect the incidence of perinatal mortality.

Theobald, G. W. 1935. The dietetic deficiency hypothesis of the toxaemias of pregnancy. Proc. Roy. Soc. Med. 28:1388.

The author suggests that the toxemias associated with pregnancy are caused by an absolute or relative insufficiency of some substance or substances in the diet. The most important nutrient is calcium, and a deficiency leads to an inharmonious working of the various physiological processes in the body and ultimately to hepatic dysfunction.

Thompson, B., and D. Baird. 1967. Some impressions of childbearing in tropical areas. II. Pre-eclampsia and low birthweight. J. Obstet. Gynaecol. Brit. Commonw. 74:499-509.

In West Africa and Borneo, where women are apt to be thin and gain a small amount of weight, there is a low rate of pre-eclampsia but a high rate of prematurity; in China, where the average age of primigravidas is 20 to 24, and pregravid weight and weight gain in pregnancy are moderate, there is a low incidence of both pre-eclampsia and prematurity.

Thomson, A. M., F. E. Hytten, and W. Z. Billewicz. 1967. The epidemiology of oedema during pregnancy. J. Obstet. Gynaecol. Brit. Commonw. 74:1-10.

The incidence of edema in normal and abnormal pregnancies was investigated.

Tobian, L. 1964. Similarities between clinical toxemia of pregnancy and experimental hypertension. Circulation. 30(2):80-95.

A toxemia hypothesis that involves the rate of synthesis of placental steroids, their rate of degradation, and the degree of antihypertensive capacity was investigated.

Tompkins, W. T., and D. G. Wiehl. 1951. Nutritional deficiencies as a causal factor in toxemia and premature labor. Amer. J. Obstet. Gynecol. 62:898-919.

Pregravid weight, gain in weight in pregnancy in relation to toxemia, and premature labor are discussed. Women who were 20 percent overweight at the outset of pregnancy showed a 6 percent incidence of toxemia compared with 1.7 percent for women of normal pregravid weight. In this group, there was no increase over normal in premature labor. In women who were 20 percent or more underweight at the outset of pregnancy, both the incidence of toxemia and premature labor were increased.

Tompkins, W. T., and D. G. Wiehl. 1954. Maternal and newborn studies at Philadelphia Lying-In Hospital. Maternal studies: III. Toxemia and maternal nutrition. Proc. Ann. Conf. Milbank Mem. Fund. 1954:62-90.

The incidence of toxemia in 1,570 patients was highest for patients 20 percent or more overweight at the outset of pregnancy. However, the incidence of moderate or severe toxemia was highest in women 15 percent or more underweight at the outset of pregnancy. Patients whose diets were supplemented with polyvitamin and protein concentrates had a lower overall incidence of toxemia than women receiving protein or vitamin supplements or no supplements. Rate of gain in weight is also discussed.

Wachstein, M. 1954. Vitamin B_6 in pregnancy. Proc. Ann. Conf. Milbank Mem. Fund. 1954:91-108.

Xanthurenic excretion varied among individuals but was lowest for nonpregnant controls, higher in pregnancy, and highest in patients with toxemia. The increase in xanthurenic excretion occurred between the 12th and 14th weeks of pregnancy. Ingestion of 25 mg of pyriodoxine caused the disappearance of xanthurenic acid excretion.

Wachstein, M., and L. W. Graffeo. 1956. Influence of vitamin B_6 on the incidence of preeclampsia. Obstet. Gynecol. 8:177-180.

Compared with pregnant controls who did not receive pyridoxine, incidence of mild and severe pre-eclampsia was reduced in white pregnant women when 10 mg of pyridoxine was given from the second or third month of pregnancy.

Wachstein, M., and A. Gudaitis. 1953. Disturbance of vitamin B_6 metabolism in pregnancy. III. Abnormal vitamin B_6 load test. Amer. J. Obstet. Gynecol. 66:1207-1213.

Urinary excretion of 4-pyridoxic acid, the main metabolic end product of vitamin B_6, was measured for an 8-hour period following the ingestion of 25 mg of pyridoxal hydrochloride. Full-term and toxemic pregnant women excreted smaller amounts of 4-pyridoxic acid than did normal controls or patients with various diseases.

Walters, W. A. W. 1966. Effects of sustained maternal hypertension on foetal growth and survival. Lancet 2:1214-1217.

In Aberdeen, 50 primigravidas with sustained hypertension experienced a perinatal mortality of 7.3 percent compared with a perinatal mortality of 2.6 percent for normotensive primigravidas.

Wiehl, D. 1952. Prevention of eclampsia and pre-eclampsia. Milbank Mem. Fund Quart. 30:382-384.

The incidence of eclampsia was reduced in Australia by close perinatal supervision, including supervision of diet.

Wiehl, D. G. 1950. Changes in blood values during pregnancy and the relation of protein levels to toxemia symptoms. Milbank Mem. Fund Quart. 28:238-262.

In 161 white women, blood values for the volume of red blood cells, hemoglobin, total protein, and albumin decreased during the first half of pregnancy; the average decrease was about equal to the average expected as a result of the average increase in plasma volume. In the second half of pregnancy, hematological values and total protein and albumin showed a slight additional decrease.

Globulin increased during the second and third trimesters. The incidence of toxemic symptoms was higher among women having both total serum protein levels and serum albumin levels below average values at any period during pregnancy.

Williams, C. 1957. Weight in relation to pregnancy toxaemia. Brit. Med. J. 1:1338-1340.

When 50 cases of normal pregnancies were compared with 50 cases of pre-eclamptic toxemia, the toxemic women had higher initial weights, larger average weekly weight gain, and larger total weight gain.

Young, B. K., and F. K. Beller. 1968. Plasma acid phosphatase in normal and preeclamptic pregnancy. Amer. J. Obstet. Gynecol. 101:1068-1072.

Plasma acid phosphatase activity was measured in 12 normal nonpregnant women, 55 normal pregnant women, and 10 severely pre-eclamptic women. The third trimester was associated with a rise in plasma acid phosphatase, and severe pre-eclampsia caused a marked rise in this enzyme.

Zunker, H. O., L. L. Phillips, and D. G. McKay. 1965. Potassium transport in kidney and placenta in normal pregnancy and in experimental toxemia. Amer. J. Obstet. Gynecol. 91:369-376.

Pregnant rats fed a vitamin E-deficient diet containing toxic fractions of oxidized cod liver oil developed a toxemic disease in which an imbalance of electrolyte metabolism occurred.

Zunker, H. O., L. L. Phillips, and D. G. McKay. 1965. Sodium transport in the kidney and placenta in normal pregnancy and in experimental toxemia. Amer. J. Obstet. Gynecol. 92:325-331.

A report on the rate of exchange of sodium in the cells of the kidney and placenta of the rat under the influence of eclamptogenic diet as well as during the course of a normal pregnancy.

U. S. GOVERNMENT PRINTING OFFICE : 1970 O - 387-735