Adolescent NUTRITION

Edited by
Myron Winick

Volume 11 in the Wiley Series on Current Concepts in Nutrition, edited by Myron Winick

ADOLESCENT NUTRITION

Edited by

MYRON WINICK
Institute of Human Nutrition
Columbia University College of Physicians and Surgeons

A Wiley-Interscience Publication

JOHN WILEY & SONS
New York • Chichester • Brisbane • Toronto • Singa

Library of Congress Cataloging in Publication Data:

Main entry under title:

Adolescent nutrition.

(Current concepts in nutrition, ISSN 0090-0443; v. 11)
"A Wiley-Interscience publication."
Includes index.
1. Youth—Nutrition. I. Winick, Myron.
II. Series.
TX361.Y6A36 613.2'088055 81-19748
ISBN 0-471-86543-5 AACR2

Printed in the United States of America

10 9 8 7 6 5 4 3 2 1

Adolescent Nutrition

CURRENT CONCEPTS IN NUTRITION

Myron Winick, Editor

Institute of Human Nutrition
Columbia University College of Physicians and Surgeons

Volume 1: Nutrition and Development
Volume 2: Nutrition and Fetal Development
Volume 3: Childhood Obesity
Volume 4: Nutrition and Aging
Volume 5: Nutritional Disorders of American Women
Volume 6: Nutrition and Cancer
Volume 7: Hunger Disease: Studies by the Jewish Physicians in the Warsaw Ghetto
Volume 8: Nutritional Management of Genetic Disorders
Volume 9: Nutrition and Gastroenterology
Volume 10: Nutrition and the Killer Diseases
Volume 11: Adolescent Nutrition

Preface

Adolescence is a period of marked change, a period during which the individual rapidly undergoes a series of sequential physical and mental changes that transform a small child into a young adult. This volume has been oganized to document these changes, explore the mechanisms by which they occur, and examine the effects of altered nutritional states on the sequence of adolescence and its ultimate outcome.

Part 1 examines the normal adolescent. Chapter 1 describes the endocrine changes that occur in both boys and girls which initiate the entrance into adolescence and which govern the changes that occur. Chapter 2 focuses on the changes in body composition accompanying adolescence in both boys and girls. These changes are very different in the two sexes, boys depositing mostly muscle whereas girls deposit mostly fat, and therefore the nutritional requirements will differ in boys and girls. These requirements are considered in Chapter 3. The concept of sex maturity index is introduced—a method of assessing the stage of adolescence, independent of age, in both boys and girls. Although the sequence of events in both sexes follows a relatively fixed schedule once initiated, the time of initiation of adolescence may vary widely. Thus the *average* girl begins her growth spurt around age twelve but this can vary from as early as nine to as late as sixteen. In boys the average age is two years later but again the spread may be very wide. In considering nutritional requirements it is the stage rather than the chronological age that is important. For example, iron deficiency is most likely to occur after the growth spurt begins in boys and can be exaggerated after menstruation begins in girls.

The adolescent in our society is subjected to various stresses. Part 2 discusses two of the most common: pregnancy and lactation, and competitive sports. In both of these situations requirements for all nutrients increase. The increase, however, is specific for the stress that is being imposed, for it must take care of the particular condition while at the same time allowing the adolescent to grow.

Part 3 describes the two most prevalent specific deficiencies encountered by adolescents: iron deficiency and zinc deficiency. Both of these

deficiencies occur because of the increased demands imposed by rapid growth and both can cause troublesome and sometimes extremely serious symptoms.

Part 4 discusses the nutritional management of certain diseases that are common in adolescents. The discussion of Keshan Disease by Dr. Keyou Ge of the Institute of Health of the Chinese Academy of Medicine is a classic description of the discovery of a specific nutrient deficiency in a large population which was resulting in widespread mortality and morbidity and the public health measures taken to eradicate the disease. The chapter on anorexia nervosa covers both the nutritional and psychiatric management of this disease. The importance of team management is emphasized. The same is true in the obese adolescent.

Chronic bowel disease can be effectively treated medically by dietary means but care must be taken to avoid undernutrition and subsequent growth failure. Steroids should be used with great caution because of their potential growth stunting effects. Finally, some new data are presented which suggest that saturated fat may not be the only dietary component to increase the risk for coronary artery disease.

Taken in its entirety, this volume covers the nutritional needs of adolescents under a variety of circumstances during health and disease. I hope the new information it includes will lead to new discoveries for the scientist and fresh treatment approaches for the practitioner.

MYRON WINICK

New York, New York
January 1982

Adolescent Nutrition

Contents

PART 4 NUTRITION IN DISEASES COMMON IN ADOLESCENCE

PART 1

Normal Nutrition

1

Nutrition and the Neuroendocrinology of Puberty

JEREMY S. D. WINTER

University of Manitoba, and Children's Hospital of Winnipeg, Winnipeg, Manitoba, Canada

Puberty is one phase in a process of neuroendocrine maturation which begins in early fetal life and continues through to adulthood (Figure 1-1). Not surprisingly this process can be influenced by external factors, one of the most important of which is the level of nutrition.

One sign of this influence has been the secular trend in Western industrialized nations to earlier puberty as well as to more rapid physical growth. Between 1850 and 1950 the mean age of menarche decreased by 3 to 4 months each decade, a trend that has only levelled off in the last 20 years. In North America today most girls begin breast and sex hair development between 8 and 13 years of age, and experience menarche about 2½ years later (with considerable individual variation). In boys, testicular enlargement begins between 9 and 14 years of age, and other secondary sexual characteristics appear over the next 2 to 2½ years.

THE DEVELOPMENT OF THE REPRODUCTIVE ENDOCRINE SYSTEM

The Fetus

The first sign of sexually dimorphic development is the appearance in the 6-week male fetus of testes. Under the initial stimulus of chorionic

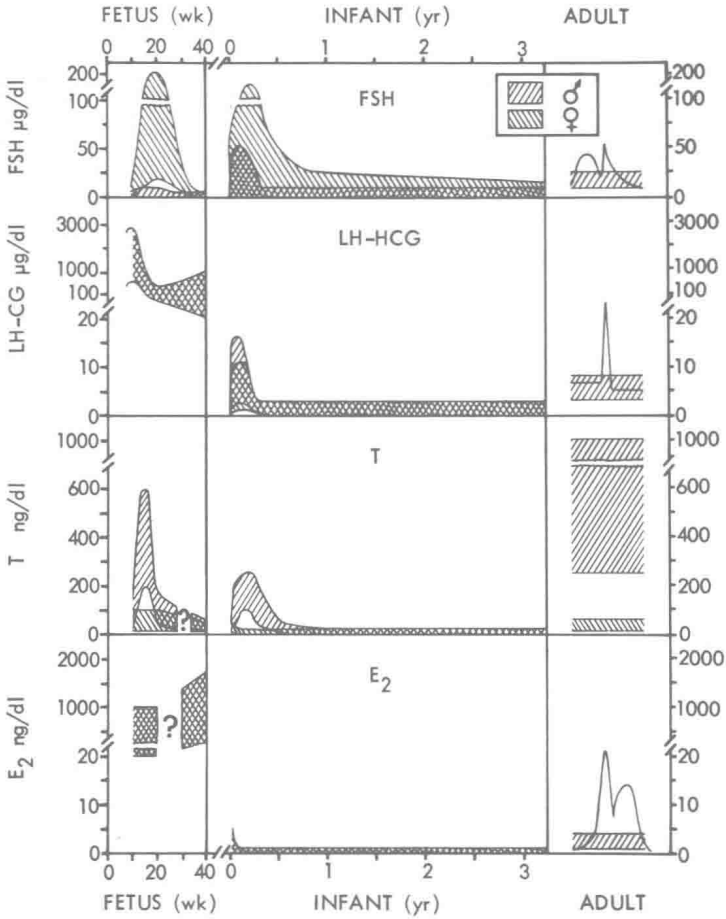

Figure 1-1. A schematic summary of the changes in serum concentrations of FSH, LH (plus HCG during fetal life), testosterone (T), and estradiol (E_2) from conception to adult life in males and females. Serum FSH and LH concentrations are in µg LER-907/dl. The adult segment compares the male range with female values during one typical menstrual cycle.

gonadotropin these secrete testosterone in amounts sufficient to raise serum levels to the adult male range and to induce masculine differentiation of the internal and external genitalia (1). The fetal ovaries develop somewhat later and do not play a similar essential role in female genital differentiation; however, by midpregnancy the fetal ovary contains large numbers of follicles and is producing some sex steroids.

As the gonads develop, so too do those structures which will ultimately regulate their function. The fetal pituitary begins to secrete FSH and LH by 10 weeks gestation. Serum concentrations of these hormones rise to a peak at 16 to 18 weeks and then decline; levels in midpregnancy are

considerably higher in females than in males. At the same time one can identify in the hypothalamus neurons containing gonadotropin-releasing hormone (GnRH), the axons of which terminate in the median eminence. Hypothalamic levels of GnRH increase during the first half of pregnancy in parallel to the rise in gonadotropin secretion, but no sex difference such as that seen with FSH and LH is observed (2). Recent studies in our laboratory have demonstrated that functional capillary connections exist between the median eminence and the anterior pituitary as early as 11 weeks gestation, while other studies have shown that the fetal pituitary is responsive to GnRH stimulation. These observations suggest that fetal pituitary gonadotropin secretion is under hypothalamic regulation from its inception. In turn, fetal pituitary gonadotropins appear to be essential for normal gonadal endocrine function and germ cell maturation.

After midpregnancy there is a decline in serum FSH and LH concentrations which likely reflects maturation of a negative feedback system responsive to the high levels of placental estrogens, and/or progesterone. The lower values of serum gonadotropins in male fetuses during the second trimester suggest that even at this early stage fetal pituitary function is modulated in part by testicular androgens.

The Infant

By the time of birth the reproductive endocrine system is structurally complete, although the mechanisms that regulate GnRH and gonadotropin secretion will continue to mature through childhood and adolescence. The disappearance from the neonatal circulation of placental estrogens removes their negative feedback influence and triggers in all infants a brisk increase in gonadotropin secretion (3). In males the testis responds with a parallel rise in testosterone secretion, which in turn somewhat blunts the gonadotropin rise (Figure 1-1). However in infant girls the ovarian steroidogenic response is less prominent; female serum gonadotropin levels often reach the postmenopausal range during the first year of life and may not return to the usual prepubertal range until 3 or 4 years of age.

Childhood

The decade between infancy and puberty is characterized by low levels of gonadotropin and sex steroid secretion. However, both the pituitary

and the gonads of the child are capable of adult endocrine function; their relative dormancy results from the interaction of two mechanisms that serve to inhibit the pulsatile secretion of hypothalamic GnRH (Figure 1-2). The first of these is a poorly understood CNS inhibitory mechanism which may be analogous to that which mediates reproductive inactivity through a kind of reverse puberty in seasonal breeding species, such as the sheep. This neuroendocrine system serves as an intrinsic biological clock, but its function can be modulated by external influences such as nutrition, the light–dark cycle, and sex steroids.

The second mechanism involves feedback inhibition by sex steroids, to the action of which the prepubertal hypothalamus is remarkably sensitive. The interaction of these two mechanisms can be seen in agonadal children, who show high levels of serum gonadotropins in infancy, a decline during childhood, and then a final rise to adult castrate levels at about age 12 (4).

During late childhood there is a gradual increase in adrenal secretion of C-19 steroids such as dehydroepiandrosterone and androstenedione. This phenomenon, termed adrenarche, may reflect either changes in pituitary function or merely intrinsic changes in adrenal steroidogenesis as a function of growth (5). Some authors, having observed that excessive amounts of potent androgen can accelerate hypothalamic maturation and thus the onset of puberty, have postulated a similar physiological role for these weak adrenal androgens, but there is little clinical or experimental support for such a relationship.

Puberty

The onset of puberty is signalled by a reduction in central inhibition of pulsatile hypothalamic GnRH secretion, which is first reflected in episodic sleep-associated bursts of gonadotropin release (6). The endocrine system amplifies this initial neural signal with time, so the pituitary becomes increasingly responsive to GnRH, and the gonads more sensitive to gonadotropic stimulation. At the same time, but probably as a secondary phenomenon, the hypothalamus and pituitary become less sensitive to feedback inhibition by gonadal steroids.

As puberty progresses sleep-associated pituitary activity is replaced by the adult pattern of pulsatile gonadotropin secretion approximately every 2 hours throughout the day and night. Mean serum concentrations of FSH and LH rise (7) and in turn elicit rising levels of gonadal androgens and estrogens (Figures 1-3 and 1-4). These sex steroids not only cause the appearance of secondary sexual characteristics, but also

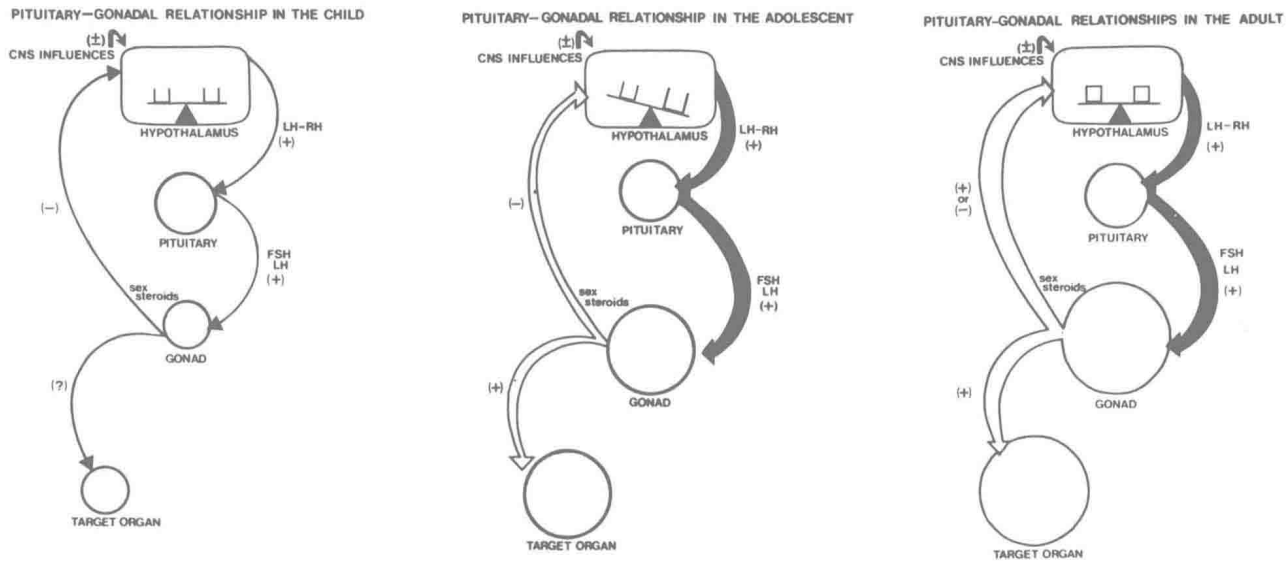

Figure 1-2. A schematic summary of the changes in hypothalamic–pituitary–gonadal interrelationships during postnatal development. The (+) sign indicates stimulation and the (−) sign inhibition. The width of each arrow reflects the secretion rates of gonadotropin-releasing hormone (LH-RH), gonadotropins (FSH and LH), and sex steroids. Reproduced with permission from J. S. D. Winter, C. Faiman, and F. I. Reyes, *Clinical Obstetrics and Gynecology,* **21,** 71 (1978).