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Research

My research has shown that undernutrition and intense physical activity can have a limiting effect on female fertility. Women who have too little body fat, because of injudicious dieting and/or intensive physical activity, also have disruption or impairment of their reproductive ability; they are infertile due to hypothalamic dysfunction which is correlated with weight loss or excessive leanness. The effect is reversible with gain in weight and fatness. Fat is an active metabolic tissue that makes estrogen, and as recently discovered, a hormone, leptin. Human reproduction has a metabolic cost: it takes about 50,000 calories over the daily caloric maintenance cost to grow a human infant to term. Lactation costs about 500-1000 calories a day. I hypothesized that a critical, minimum amount of body fat is necessary for, and directly influences, female reproduction.

My research results are predictive and are now used clinically for evaluation of nutritional infertility and the restoration of fertility. Research on the long-term health of 5,498 U.S. college alumnae showed that moderate athletic regular activity resulted in a lower risk of breast cancer and cancers of the reproductive system and a lower risk of late onset diabetes. Environmental factors of nutrition, physical activity and disease can affect each reproductive milestone from menarche to menopause, hence as I have documented the natural fertility of populations.

In accord with the connection fatness and fertility is the 1994 discovery of J. Friedman et. al. that body fat produces a hormone, leptin, which has receptors in the hypothalamus, the ovary and the testis. Leptin controls appetite and energy metabolism, and the hypothalamic control of reproduction. My book "Female Fertility and the Body Fat Connection" (2002 & 2004 paperback, University of Chicago Press) details this research.

Education

1943, University of Wisconsin, Ph.D. (Genetics)
1940, Columbia University, M.A. (Zoology)
1939, Smith College, B.A.
(Magna cum laude, Phi Beta Kappa, Sigma Xi)

Awards and Honors

2005, Professor Emeritus Award of Merit, Harvard School of Public Health, Boston, MA.

2003, Rally Day Medal for Medical Research and Reproductive Health, Smith College, Northampton, MA.
1997, Fellow, American Academy of Arts and Science, Cambridge, MA.
1993-1994, Fellow, Bunting Institute, Cambridge, MA.
1992, Appointed Associate Professor of Population Sciences Emerita.
1988-1991, Research Grant Award, National Institute of Child Health and Human Development.
1988, Appointed Sigma Xi National Lecturer 1988-1990
1975-1976, Fellow, John Simon Guggenheim Memorial Foundation

<http://www.hsph.harvard.edu/faculty/rose-frisch/>

Publications

Frisch, RE, and McArthur JW. Menstrual cycles: Fatness as a determinant of minimum weight for height necessary for their maintenance or onset. *Science*, 1974 185:949-951.

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Buchübersicht

Are girls entering puberty earlier than they used to? This question, which has been debated recently by doctors and scientists in the pages of "Time" magazine and the "New York Times," proves that there is still a great deal to learn about women's reproductive health. "Female Fertility and the Body-Fat Connection" is the record of one scientist's groundbreaking and decades-long work on the connections among fertility, body fat, and reproductive health in women.

Rose E. Frisch explains here how, in women, a certain amount of body fat is crucial to the reproductive system and sexual maturation. Women who are too lean are infertile and cannot conceive children; young girls who are too thin have a delayed onset of their first period. "Female Fertility and the Body-Fat Connection" illuminates how and why a "critical fitness" level underlies a woman's reproductive health. In the process Frisch gives readers a comprehensive view of the research done to date on the relationship between body composition and fertility and also describes her own journey as a woman scientist working to advance her critical-fitness hypothesis both to the general public and the scientific community. Frisch answers the questions every woman has about the desirable weight for health and fertility and even includes tables to help women find their own best weight. She also demonstrates how important diet and exercise are for the long-term reproductive health of women, and shows what factors influence the onset of puberty in girls.

Each milestone of the reproductive life span is affected by food intake and energy output, the factors affecting the storage of fat. "Female Fertility and the Body-Fat Connection" is a cornerstone to understanding the health of girls and women.

Eingeschränkte Vorschau - 2002 - 194 Seiten

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research-article

Body fat, menarche, fitness and fertility

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Many well-trained athletes, ballet dancers and women who diet excessively have secondary or primary amenorrhoea. Less extensive training or weight loss may result in anovulatory menstrual cycles, or a shortened luteal phase. These disruptions of reproductive ability are due to hypothalamic dysfunction, which is correlated with weight loss or excessive leanness. It is proposed that these associations are causal and that the high percentage of body fat (26–28%) in the mature human female may influence reproduction directly. Four mechanisms are known: (i) adipose tissue converts androgens to oestrogen by aromatization. Body fat is thus a significant extragonadal source of oestrogen; (ii) body weight, hence fatness, influences the direction of oestrogen metabolism to more potent or less potent forms; leaner women make more catechol oestrogens, the less potent form; (iii) obese women and young, fat girls have a diminished capacity for oestrogen to bind sex-hormone-binding-globulin; (iv) adipose tissue

can store steroid hormones. An indirect mechanism may be signals of abnormal control of temperature and changes in energy metabolism, which accompany excessive leanness. The hypothalamic reproductive dysfunction results in abnormal gonadotrophin secretion: there is an age inappropriate secretory pattern of luteinizing hormone (LH) and follicle stimulating hormone (FSH), resembling that of prepubertal children. The secretion of LH and the responses to LHRH are reduced in direct correlation with the amount of weight loss. Other evidence from non-athletic and athletic women and mammals is presented in support of the hypothesis that a particular, minimum ratio of fat to lean mass is normally necessary for menarche ($\approx 17\%$ fat/body wt) and the maintenance of female reproductive ability ($\approx 22\%$ fat/body wt). Nomograms are given for the prediction of these critical weights for height from a fatness index; these weights are useful clinically in the evaluation of nutritional amenorrhoea and the restoration of fertility in underweight women. Evidence is presented that undernutrition and hard physical work can affect the natural fertility of populations, by the delay of menarche, a longer period of adolescent subfecundity, a longer birth interval and an earlier age of menopause. Data from a study of the longterm reproductive health of 2622 former college athletes compared with 2766 non-athletes show that the former college athletes had a significantly lower lifetime occurrence of breast cancer and cancers of the reproductive system, and a lower lifetime occurrence of benign tumours of these tissues, compared with the non-athletes. Over 82% of the former athletes began their training in high school or earlier. A possible explanation may be that long-term, the former athletes had lower levels of oestrogen because they were leaner, and more of the oestrogen was metabolized to the non-potent catechol oestrogens. The observed reduction of cancer risk associated with physical exercise has potential for public health.

Key words: fatness/fertility/fitness/menarche/puberty

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