Obesity from a Primal Health Research Perspective

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Full Text: Headnote ABSTRACT: Obesity has become an epidemic in the United States, threatening the health of millions of Americans. President Clinton has been a major supporter of addressing the problem of obesity, especially in children. To date this condition has been challenging to both understand its origins and to treat. This article reviews the pre- and perinatal literature and related medical literature and suggests that intrauterine undernutrition (famine-like) conditions during the first trimester shows a promising area for further research to explore childhood and adult obesity. KEY WORDS: Obesity, famine, pre- and perinatal period, insulin resistance, type 2 diabetes. INTRODUCTION When Bill Clinton announced the launch of a 10-year initiative to combat childhood obesity, he said: "We've got to change the eating habits of America's young people". He was concisely expressing the predominant attitudes and questions regarding the morbidly fat for thousands of years, from the Hippocratic concept of 'regimen' up to William Banting's famous pamphlet: 'A Letter On Corpulence Addressed To The Public'. Recent scientific advances demand our attention to both new and fundamental questions. We must first wonder how and when the systems that regulate our appetite and our capacity to store fat adjust themselves and reach their 'set point levels'. In general, in the field of health, we should first be concerned with the programming of our biological computers during the primal period of life (Odent, 1986). Our study of obesity will combine data provided from two perspectives. First, we'll explore the 'Primal Health Research Data Bank', which includes dozens of studies detecting risk factors for obesity during the primal period (from conception until the first birthday). In order to interpret the results of such studies, we'll also consider what we are learning today about recently-discovered components of the 'primal adaptive system', in particular how adipose tissue functions as an endocrine organ, even the largest one, if we consider its mass. We shall not review data provided by the genetic perspective, since our intent is to throw light on the sudden widespread epidemic of obesity. Our current knowledge of the genetic factors that predispose to obesity cannot be useful: the gene pool of human populations cannot have changed sufficiently in the past fifty years to explain the current epidemics. We must therefore look at environmental factors. However, we must keep in mind how artificial it is to attempt to separate genetic factors and environmental factors, when we already understand that the expression of our genes is influenced by early environmental factors. We shall not consider the association between obesity and birth order either, although it is worth mentioning, while family size continues to decrease, that being first-born appears as a risk factor. LESSONS FROM THE PRIMAL HEALTH RESEARCH DATA BANK One of the oldest and most valuable studies in our database was precisely about risk factors for obesity. It was published as early as 1976 in an authoritative medical journal (Ravelli, Stein, &Susser, 1976). From October 1944 to May 1945 an acute famine affected the western Netherlands. The authors combined information about prenatal and early postnatal status at the time of the famine with weight and height at the age of 19 on examination of 300,000 men for military service. The main conclusion was that deprivation during the first half of pregnancy was related to significantly higher obesity rates at age 19, while deprivation during the last trimester of pregnancy and the first months after birth, was associated with lower obesity rates. This historical study opened the way to further research about the long-term effects of being in the womb during the Dutch famine. In one of these studies, published in 1999, the authors measured the body size of 741 people born at term between November 1943 and February 1947 in Amsterdam. They compared people exposed to famine in late, mid, or early gestation with those born before, or conceived after, the famine period. It appeared in particular that maternal malnutrition during early gestation was associated with higher Body Mass Index and

waist circumference in 50 year-old women but not in men. Another study (published in 1998) looked at the glucose tolerance of adults who had been exposed either to the famine during fetal life, or who were born in the same area the year before the famine, or who had been conceived after the famine. Glucose tolerance-which is closely related to the tendency to obesity-was significantly decreased among adults who spent their intrauterine life during the period of starvation. The siege of Leningrad also exposed the entire population of a well-defined area to a severe famine. Among those exposed to malnutrition the influence of obesity on blood pressure was stronger. Today the keyword 'obesity' (and the related keywords 'insulin resistance' and 'diabetes type 2') leads to about 25 entries in our data bank (www.birthworks.org/primalhealth). From an overview of all these studies we can easily draw the conclusion that the risks of being obese in childhood and adulthood are to a great extent already determined before the end of the 'primal period'. Many studies researched the risks for these diseases in relation to birth weight and confirmed the results of the Dutch studies. Smoking in pregnancy was always found to be a risk factor, as were the long-term effects of drugs given to pregnant women. For example, it appears from one study that betamethasone (a glucocorticosteroid) given to a pregnant woman in order to prevent a respiratory distress of her newborn baby might result in insulin resistance of that child 30 years later. There have been many studies evaluating the prevalence of obesity in childhood, adolescence and adulthood in relation to infant feeding. In general, it seems that breastfeeding has a protective effect. However, the associations between breastfeeding, its duration, and the risks of being overweight in childhood, adolescence and adulthood have not been confirmed by large authoritative studies extending to adulthood such as the 1958 British birth cohort. It appears from several of these studies that the weight of the mother is a stronger predictor of obesity than the mode of infant feeding: a big mother will tend to produce a big baby. Such data suggest that the metabolic profile of a pregnant woman has more long-term influence than the kind of food consumed by a baby. Interestingly, from studies that focus in particular on the first week following birth, we can conclude that the period between birth and age 8 days is a critical window for nutritional programming. One study looked at the weight gain during this critical period of adults aged 20 and 32 who had been bottle-fed. Another one examined the first week of extra uterine life of children of diabetic mothers. From this 2005 overview of the Primal Health Data Bank we can conclude that the risks of being obese are to a great extent determined by pre- and perinatal environmental factors. WHEN FAT CELLS BECAME ENDOCRINE GLANDS Until recently adipose tissue was considered an inert energy store. The turning point occurred in 1994 when leptin (from the Greek word leptos, which means 'thin') was identified as a hormone released by fat cells (adipocytes), the absence of which resulted in morbid obesity in the ob/ob mouse (Zhang, Proenca, Maffei, et al., 1994). Today leptin may be regarded as one of the many 'adipokines'-hormones that signals changes in fatty tissue and energy status to control fuel usage. (This new framework includes adiponactin, resistin, plasminogen activator inhibitor-1, tumor necrosis factor-alpha, visfatin, retinal binding protein 4). The relative roles of all these hormones in modifying appetite and insulin resistance are the subjects of intense research. While leptin is also secreted by the placenta, the mammary gland, and the stomach, adiponectin seems to be exclusively secreted by adipose tissue into the bloodstream; its levels are inversely correlated with body mass index. The word resistin was chosen because of the observed insulin resistance after injections of this hormone. Visfatin mimics the effects of insulin. Hormones released by the fat cells are not the only signals that communicate the state of energy balance in the body to the brain. The recently-discovered gastric hormone ghrelin increases hunger through its action on hypothalamic centers. Blood ghrelin concentration increases during fasting. Humans injected with ghrelin report sensations of intense hunger. A gastric bypass operation tends to reduce the levels of this hunger-inducing hormone. In the current scientific context we are achieving a radically new vision of energy homoeostasis. We used to visualize the brain as being in control of the body. Today, we have to visualize the continuous exchanges of signals between peripheral parts of the body (fat cells, stomach) and the brain centers. The concept of a 'primal adaptive system' is ever more useful when referring to the basic adaptive systems involved in what we commonly call health. Originally, I suggested this term to avoid the artificial

separations between the nervous, immune, and endocrine systems. Today, as we are learning that fat cells, the heart and the digestive tract are all endocrine glands, the network we call the 'primal adaptive system' appears larger and much more complex than we could have imagined twenty years ago. Our study of obesity is an opportunity to formulate fundamental questions about when and how our basic adaptive systems develop, adjust and regulate themselves. It is probable that the recently discovered components of the primal adaptive system also reach their set point levels during critical phases of the primal period. This is suggested by animal experiments which have detected a neonatal leptin surge following intrauterine undernutrition that caused obesity in adulthood (Yura, Itoh, Sagawa, et al., 2005). Among humans it has been demonstrated that breastfed babies have higher leptin values than bottle-fed babies in the first four months of life (Savino, Nanni, Maccario, et al., 2004). Furthermore, breast milk of mothers of small-for-gestational, large-for-gestational, and appropriate-for-gestational babies has different amounts of leptin, especially during the first month of postpartum life. More rapid growth and significantly- reduced leptin levels are seen in the small-for-gestational age group during the first postnatal 15 days, compared with the others (Dundar, Anal, Dundar, et al., 2005). Although the details are complex, the point is that important activity occurs during the critical perinatal period. OBESITY AND SCHIZOPHRENIA There are many other fruitful ways to explore the 'Primal Health Research Data Bank'. One is to observe diseases that share the same risk factors in the primal period. Where obesity is concerned, this suggests possible links with schizophrenia (visit www.birthworks.org/ primalhealth and type the key word 'schizophrenia'). It is now well established that those who spent their prenatal life during the Dutch Hunger Winter were also at increased risks of developing schizophrenia later in life. This has been confirmed recently by an evaluation of the rates of onset of adult schizophrenia following prenatal exposure to the Chinese famine of 1959-1962, which involved a population of 62 million in the Wuhu region of the Anhui province. It is noteworthy that the well-known correlation between obesity and schizophrenia predates the availability of modern antipsychotic drugs (Allison, Fontaine, Heo, et al., 1999). It is also notable that the prevalence of glucose intolerance and diabetes type 2 is high among schizophrenic cohorts (Bushe &Holt, 2004). Today obesity, diabetes type 2, and schizophrenia may be interpreted as the long-term consequences of developmental defects during the pre- and perinatal periods of development. We must also consider that certain brain structures (such as the hippocampus) and the pancreas share the same basic nutritional needs during their critical periods of development, for example zinc. It is still premature to claim that obesity, different expressions of insulin resistance, and schizophrenia are different aspects of the same disease expressed in a great variety of dominant symptoms, according to individual genetic predispositions. But it is now time to claim that the concepts of 'Primal Adaptive System' and 'Primal Health Research' are dissolving the conventional barriers between scientific and medical disciplines. FURTHER RESEARCH The primal health research perspective suggests the sort of research that can help explain the worldwide epidemic of obesity (and diabetes type 2). We must compare the diet of modern mothers-to-be with the diet of previous generations, whatever the geographical context. For example soft drinks and trans-fatty acids are particularities of modern diet. We must also look at how modern management of the perinatal period deviates from the physiological model. Today, among the unprecedented ways to interfere with the physiological processes, the most common ones are the widespread practice of labour induction, the increased rates of non-labour (elective) caesarean, and the association of epidural anaesthesia with synthetic oxytocin infusion. Bill Clinton has a lot on his plate if, in his mind, 'young people' include-first and foremost-mothers-to-be. Sidebar Editor's note: These two essays are reprinted with permission of Michel Odent, MD, Director, Primal Health Research Center in London and the newsletter Primal Health Research, published in North and South America by Birth Works, Inc., Medford, NJ. APPPAH is pleased to support increased circulation by reprinting Dr. Odent's essays as a feature in the pages of this journal. Contact information: info@birthworks.org or (609) 953-9380. Free access to the Primal Health Research Data Bank is provided at: www.birthworks.org/primalhealth. Email for Dr. Odent is: modent@aol.com References REFERENCES We mention the references that preceded the recent development of Primal Health

Research, and those that cannot be found in the Primal Health Research Data Bank. Allison, D.B., Fontaine, K.R., Heo, M., et al. (1999). The distribution of body mass index among individuals with and without schizophrenia. Journal of Clinical Psychiatry, April, 60(4), 215-20. Bushe, C. &Holt, R. (2004) Prevalence of diabetes and impaired glucose tolerance in patients with schizophrenia. British Journal of Psychiatry (Suppl.) April, 47, S67-71. Dundar, N.O., Anal, O., Dundar, B., et al. (2005). Longitudinal investigation of the relationship between breast milk leptin levels and growth in breast-fed infants. Journal of Pediatric Endocrinology and Metabolism, February, 18(2), 181-7. Odent, M. (1986). Primal health. London: Century-Hutchinson. (2nd edition. Clairview 2002) Ravelli, G.P., Stein, Z.A., &Susser, M.W. (1976). Obesity in young men after famine exposure in utero and early infancy. New England Journal of Medicine, August, 12, 295(7), 349-53. Savino, F., Nanni, G.E., Maccario, S., et al. (2004). Breast-fed infants have higher leptin values than formula-fed infants in the first month of life. Journal of Pediatric Endocrinololgy and Metabolism, 17(11), 1527-32. Yura, S., Itoh, H., Sagawa, N., et al. (2005). Role of premature leptin surge in obesity resulting from intrauterine undernutrition. Cell metabolism, 1(6), 371-78. Zhang, Y., Proenca, R., Maffei, M., et al. (1994). Positional cloning of the mouse obese gene and its human homologue. Nature, 372, 425-32. AuthorAffiliation Michel Odent, MD, Primal Health Research Center, London, England

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