PRIMAL HEALTH RESEARCH

A NEW ERA IN HEALTH RESEARCH

Published quarterly by Primal Health Research Centre
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Summer 2005 Vol.13 No.1

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OBESITY

FROM A PRIMAL HEALTH RESEARCH PERSPECTIVE

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 expressing in a concise way what have been for thousands of years the main attitudes and questions regarding the morbidly fat, from the Hippocratic concept of regimen up to William Banting's famous pamphlet *A letter On Corpulence Addressed To The Public.*

Recent scientific advances lead us to give the priority to 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, the main questions today are about how our biological computers are programmed during the primal period of life.[1]

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. 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 what we are learning about the adipose tissue as an endocrine organ, even the largest one, if we consider its mass.

We shall not look at data provided by the genetic perspective, since our ulterior motive 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 epidemic. We must therefore look at environmental factors. However we must keep in mind how artificial it is to separate genetic factors and environmental factors, at a time when we 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 families are smaller than ever, 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 included in our database was precisely about risk factors for obesity. It was published as early as 1976 in an authoritative medical journal.[2] From October 1944 to May 1945 an acute famine affected the Western Netherlands. The authors could combine 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-y-old women but not in men. Another study, published in 1998, looked at the glucose tolerance of adults who had been either exposed 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. The 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 has also exposed the whole population of a well-defined area to a severe famine. It appeared that 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. From an overview of all these entries 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'. There are many studies looking at the risks in relation to birth weight and confirming the results of the Dutch studies. When researchers looked at smoking in pregnancy, they always found that it was a risk factor. We can learn also from long-term effects of drugs

is more useful than ever when referring to the basic adaptive systems involved in what we commonly call health. Originally I suggested this term as a way to get rid of the artificial separations between the nervous system, the immune system, and the endocrine system. Today, while we are learning that the fat cells, the heart and the digestive tracts are endocrine glands, the network we call the 'primal adaptive system' appears larger and much more complex than we could imagine 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 led to obesity in adulthood.[4] Among humans it has been demonstrated that breastfed babies have higher leptin values than bottle fed babies in the first four months of life.[5] Furthermore, maternal milk of small-for-gestational, large-for-gestational, and appropriate-for-gestational babies have different amounts of leptin, especially during the first month of life. More rapid growth is shown in the small-for-gestational age group during the first postnatal 15 days, compared with the others, and leptin levels are significantly reduced in that group.[6]

Although the details are complex, the point is that there is a lot of activity during the *critical perinatal period.*

Obesity and schizophrenia

There are many other fruitful ways to explore the 'Primal Health Research Data Bank'. One of them is to put together diseases that share the same risk factors in the primal period. Where obesity is concerned, this leads to look at the possible links with schizophrenia (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 becoming schizophrenic later on in life. This has been confirmed recently by an evaluation of the rates 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 noticeable that the well-known correlation between obesity and *schizophrenia predates the availability of modern antipsychotics.*[7] *It is also noticeable* that the prevalence of glucose intolerance and diabetes type 2 is high among schizophrenic cohorts.[8] 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 human development. We must also consider that certain brain structures (such as hippocampus) and the pancreas share the same basic nutritional needs during their critical periods of development, for example their needs in zinc. It is still premature to claim that obesity, different expressions of insulin resistance, and schizophrenia are several aspects of one disease that can express itself via a great variety of dominant symptoms, according to genetic individual predispositions. But it is not premature to claim that the concepts of 'Primal adaptive system' and 'Primal Health Research' lead to smash the conventional barriers between scientific and medical disciplines.

Further research

The primal health research perspective indicates the sort of research we are expecting in order to explain the worldwide epidemic of obesity (and diabetes type 2). We must look at what makes the diet of modern mothers-to-be special, compared with the diet of the previous generations, whatever the geographical context. This leads to focus on soft drinks and trans-fatty acids. We must also look at the modern ways to deviate from the physiological model in the perinatal period. Today the most common ways to interfere with the physiological processes are the widespread practice of labour induction, the increased rates of non-labour (elective) caesarean, and the association of epidural anaesthesia with drips of synthetic oxytocin. Bill Clinton has a lot on his plate if, in his mind, 'young people' include first and foremost mothers-to-be.

Michel Odent

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.

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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 the newborn baby might result 30 years later in insulin resistance of her child.

There have been many studies evaluating the prevalence of obesity in childhood, in adolescence and in adulthood in relation to the mode of infant feeding. In general it seems that breastfeeding has a protective effect. In fact 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 such as those following until adulthood the members of 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 effects than the kind of food consumed by a baby.

Interestingly, from the studies that look in particular at 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 of these studies looked at the weight gain during this critical period of adults age 20 and 32 who had been bottle-fed. Another one looked at 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 the fat cells (the adipocytes), and whose absence resulted in morbid obesity in the ob/ob mouse.[3] Today leptin may be presented as one of *the many 'adipokines', that is to say one of the hormones that signal changes in fatty-*tissue mass and energy status so as 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 from 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' functions to increase hunger through its action on hypothalamic centers. There is an increased blood ghrelin concentration 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 reaching 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 the peripheral parts of the body (fat cells, stomach) and the brain centers. The concept of 'primal adaptive system'