Primal Health Research: Four Essays

Author: Odent, Michel

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Abstract: None available.

Full Text: Michel Odent, M.D. I. Primal Health Research: A New Era in Health Research INTRODUCTION Even in the age of the Internet, a dinner table is more suitable than a transatlantic exchange of emails for initiating fruitful projects. During my last trip to the East Coast I met Cathy Daub and Debra Mendelson, from Birth Works®. It appeared (at dinner time) that by merging our websites we could offer free access to the Primal Health Research Data bank. Thanks to the expertise of Paul, Debra's husband, it took only some days to introduce a real data bank on the web. It is already useable. It will be improved in the near future. This bank will be first a tool for researchers interested in the health of the unborn generations. It will contribute to induce a new awareness of the long-term consequences of early experiences. Today it is easy to have an overview of all the (apparently) unrelated references and abstracts we brought together. This overview can convince anyone that our health is to a great extent shaped in the womb. Of course the impressive proportion of studies detecting correlations between fetal life and health later in life must be interpreted. There are simple and practical explanations. For example we must take into account the fact that it is easy (and politically correct) to introduce in a computer such indicators of fetal growth as birth weight. But we must go beyond such explanations: the point is that the studies we detected in the scientific and medical literature originate from countless disciplines: cancerology, neurology, cardiology, dentistry, reproductive medicine, endocrinology, gerontology, ophthalmology, psychiatry, etc. This means that today, when we refer to the environmental factors that determine the health and behaviour of human beings (versus the genetic factors), we must focus on the intrauterine environment. The time has come for a radically new vision of human development. The main implications of this new generation of research is that fetal growth and fetal development must become major public health preoccupations. In the current scientific context some of the factors that influence the quality of prenatal life are better understood. This is the case of the emotional factors. Pregnant women always had the intuitive knowledge that the development of their baby in the womb was influenced by their emotional state. Today physiologists can interpret this influence. For example when a pregnant woman is not happy because she feels dominated by somebody (e.g. an authoritarian boss) or by a situation (e.g. unwanted pregnancy) she has a tendency to release high levels of hormones such as cortisol. Yet cortisol is an inhibitor of fetal growth. The more aware we are of the importance of the emotional states of pregnant women, the more we will take into consideration the possible "nocebo effect" of antenatal care. It seems that many health professionals involved in antenatal care have not realized that one of their roles should be to protect the emotional state of pregnant women. In the issues of autumn 1994 (vol. 2, no. 2) and spring 1995 (vol. 2, no. 4), I had already introduced the concept of nocebo effect of antenatal care. Five years later I find it urgent to reintroduce the topic. ANTENATAL SCARE I constantly receive phone calls from pregnant women who are in a state of anxiety, even panic, after an antenatal visit. I usually reassure them by transmitting the sort of hard data that is easy to find at the age of evidence-based medicine. Having analyzed the most common reasons for these phone calls, I have realized that in general, ignorance is the basis of the widespread nocebo effect of antenatal "care". Most practitioners seem to be unable to scan the abundant medical literature for valuable epidemiological studies. I found that this sort of blindness is related to a deep rooted cultural misunderstanding of one of the most vital functions of the placenta, that is the placenta as an advocate of the baby: the placenta is constantly manipulating maternal physiology for fetal benefit. The placenta can send messages to the mother via hormones such as HCG or Human Placental Lactogen. It is as if the placenta is telling the mother, for example: "please dilute your blood

and make it more fluid, so that it can more easily go where it is urgently needed". The placenta can also ask the mother: "please, increase your blood pressure because we need more blood". It can also tell the mother about an increased need for glucose: this leads to a transitory modification of the metabolism of carbohydrates. The results of epidemiological studies are eloquent reminders of these functions of the placenta. Let us illustrate these interpretations by looking at three main reasons for panicky phone calls after a prenatal visit. First Example: "My Haemoglobin is 9:1 am Anaemic" When a woman has a haemoglobin concentration in the region of 9.0 or 9.5 at the end of her pregnancy, there are two possibilities. More often than not she will meet a practitioner (doctor or midwife) who is not interested in epidemiological studies and who thinks that iron deficiency in pregnancy can be detected via the haemoglobin concentration. She will be told that she is anaemic and she will be given iron tablets. She will understand that there is something wrong in her body that needs to be corrected. It can happen, on the other hand, that a pregnant woman with a similar haemoglobin concentration meets a practitioner who is aware of the most significant epidemiological studies and who is interested in placental physiology. This practitioner has digested the huge and authoritative study by a London team about the relation between maternal haemoglobin concentration and birth outcomes (1). Birth outcomes of 153,602 pregnancies were analysed (the haemoglobulin measurement used in the study was the lowest recorded during pregnancy). They found that the highest average birth weight was in the group of women who had a haemoglobin concentration between 8.5 and 9.5. Their main conclusion was that "the magnitude of the fall in haemogloblin concentration is related to birth weight". A similar pattern occurred in all ethnic groups. Furthermore it appeared that when the haemoglobin concentration fails to fall below 10.5, there is an increased risk of low birth weight and preterm delivery. Similar conclusions have been reached by other, yet smaller, epidemiological studies (2,3). This sort of practitioner is also probably aware of the many studies that fail to demonstrate that iron supplementation may improve birth outcomes (4). When such a practitioner suspects anaemia, he or she prescribes specific tests such as erythrocyte protoporphyrin, transferrine saturation or serum ferritin. The pregnant woman who has access to this evidence-based ante-natal advice will be offered reassuring explanations. It will be explained that the blood volume of a pregnant woman is supposed to increase dramatically, and that the haemoglobulin concentration indicates the degree of blood dilution. She will understand that the results of her tests are suggestive of effective placental activity and that her body is responding correctly to the instructions given by the placenta. She will be given good news. The antenatal visit will have had a positive effect on her emotional state and therefore on the growth and development of her baby. All over the world millions of pregnant women are wrongly told that they are anaemic and are given iron supplements. There is a tendency to overlook the side effects of iron (constipation, diarrhoea, heartburn, etc., plus the fact that iron inhibits the absorption of such an important growth factor as zinc (5). This misinterpretation of haemoglobin concentration in pregnancy is widespread beyond belief. A Japanese lady spent the first half of her pregnancy in London, before going back to Tokyo. One of her European friends (who had four babies) warned her long in advance that at the end of her pregnancy she will be told that she is anaemic and given iron tablets. Guess the end of the story. An authoritative British team of epidemiologists published a study about third stage of labour in a prestigious medical journal. In order to concentrate on low risk pregnancies they eliminated all women whose haemoglobin was below 10.(6) Finally, the average concentration in the population they studied was 11.1. Afterwards I was given an opportunity to indicate some of the limitations of this study (7). A lack of interest in placental physiology is at the root of such misinterpretations. There is a tendency to confuse a transitory physiological response (blood dilution) with a disease (anaemia). Obstetrics is dangerous when it is not evidence-based. Second Example: "They Are Giving Me Drugs to Treat My High Blood Pressure". In late pregnancy many women have an increased blood pressure. Once more there are two possibilities. More often than not this will be presented as bad news. What's more, certain women will be given antihypertensive drugs. The message is that there is something wrong that needs to be corrected. However there are practitioners who will not present an increased blood pressure as bad news. These

practitioners can perceive and explain the fundamental differences between a gestational hypertension ("pregnancy-induced hypertension") as a physiological response and the disease pre-eclampsia. They can easily offer a reassuring analogy such as: "when you have a brain tumour, you have a headache; but when you have a headache it does not mean that you have a brain tumour". In the same way when you have pre-eclampsia you have a high blood pressure, but an increased blood pressure in late pregnancy does not mean pre-eclampsia. The explanations given by such practitioners are supported by several epidemiological studies. The most significant study from this regard is an examination of perinatal mortality over two years in the obstetric population at the Nottingham City Hospital (8). It demonstrated clearly that the best possible outcomes are among women with gestational hypertension compared with the overall population and, of course, compared with the pre-eclamptic women. Similar results, with smaller numbers, have been presented by Naeye (9), by Kilpatrick (10) and by Curtis (11). The misinterpretations of the fluctuations of blood pressure in pregnancy are as widespread as the misinterpretations of the fluctuations of haemoglobin concentrations. A recent review article identified 45 controlled trials that randomly allocated women with mild-to-moderate hypertension to antihypertensive treatment (12). This endless repetition of studies has been called "circular epidemiology". Of course the main effects of an antihypertensive treatment during pregnancy is to restrict fetal growth and to increase the number of low weight babies. Practitioners who have understood placental physiology would not even think of treating with drugs what is a physiological response and would anticipate the dangers. Third Example: "I Am Diabetic!" Many practitioners do not realize how powerful the nocebo effect of the term "gestational diabetes" can be! When a woman is given this diagnosis she tends to confuse what is a transitory response to fetal needs with a serious chronic disease. Such a term can transform overnight a happy pregnant woman into a sick person. The point is that this diagnosis is useless. Professor John Jarrett, from London, claims that gestational diabetes is a "non-entity" (13). In a letter to the American Journal of Obstetrics and Gynecology it has been called "a diagnosis still looking for a disease". Today there is a debate on whether pregnant women should be screened for glucose tolerance (14). This diagnosis is useless because, when it has been established, it leads to simple recommendations that should be given to all pregnant women, such as: avoid pure sugar (soft drinks, etc.); prefer complex carbohydrates (pasta, bread, rice, etc.); have a sufficient amount of physical exercises. We could write volumes about the nocebo effects of antenatal care. Three examples were enough to measure the amplitude of an intriguing phenomenon that is basically the same all over the world. An overview of the Primal Health Data Bank gives an opportunity to realize how serious this topic is. WHAT IS CUL-DE-SAC EPIDEMIOLOGY? An overview of our data bank can reveal other intriguing phenomena. One is the contrast between circular epidemiology and cul-de-sac epidemiology. The best way to explain the meaning of these phrases is to offer a reprint of a text I recently published in the Lancet, Between circular and cul-de-sac epidemiology. Lancet 2000, 355 (April 15): 1371. In her Feb 12 feature (p.556) Marilynn Larkin reports that Lewis Kuller condemned the continuation of epidemiological studies "beyond the point of reasonable doubt"(1). I have been intrigued for many years by the opposite of what Kuller calls "circular epidemiology". I call it cul-de-sac epidemiology. This framework includes research about topical issues. Despite the publication of this research in authoritative medical or scientific journals, the findings are shunned by the medical community and the media. "Cul-de-sac" epidemiological studies are not replicated, even by the original investigators and they are rarely quoted after publication. The first example I can offer is a Swedish study, published in 1990 by Bertil Jacobson, leading to the conclusion that certain obstetric drugs are risk factors for drug addiction in adult offspring (2). The results have never been confirmed or invalidated by further research. Yet drug addiction is one of the main preoccupations of our time. Another example is about obstetric medication as a possible risk factor for autism. At the end of his life, the Nobel prize winner Niko Tinbergen studied autistic children with the methods of a field ethologist. He came to the conclusion that there are risk factors for autism in the perinatal period, such as anaesthesia during labour and induction of labour. His observations inspired only one study. Ryoko Hattori (Kumamoto, Japan) found that the "Kitasato University's method" of delivery is a risk

factor for autism. This method is characterized by a combination of sedative, anaesthetic agents, and analgesics, together with a planned delivery induced a week before the due date. The curiosity of researchers has not been stimulated by this Japanese study, published in 1991 (3). From my conversations and correspondence with these researchers (including a trip to Kumamoto), I became aware of their similar comments on epidemiology. They all realized afterwards that research may be "politically incorrect." A pessimistic analysis focusing on the difficulties of epidemiology may inspire the simplistic conclusion that politically correct research leads to "circular epidemiology" and that politically incorrect research leads to "cul-de-sac epidemiology". An optimistic analysis would stress that it is possible to break through the dead end of a cul-de-sac and open an avenue. In other words the limits of political correctness are not immutable. Let us welcome break-through epidemiology. Editor's Note: These essays of unique importance in the field of prenatal and perinatal psychology and health, are reprinted with permission of Michel Odent, Director, Primal Health Research Centre 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 four essays as an annual feature in the pages of this Journal. For information about subscribing to the current volume of the newsletter please email info@birthworks.org or telephone: 609-953-9380. Free access to the Primal Health Research Data Bank is provided at: www.birthworks.org/primalhealth. Email for Dr. Odent: modent@aol.com REFERENCES References to Lancet article: 1. Larkin M. Epidemiological studies: Overdone or underappreciated? Lancet 2000; 355: 556. 2. Jacobson B, Nyberg K, Gronbladh L, et al. Opiate addiction in adult offspring through possible imprinting after obstetric treatment. BMJ 1990; 301:1067-70. 3. Hattori R, Desimaru M, Nagayama I, &Inoue K. Autistic and developmental disorders after general anaesthetic delivery. Lancet 1991; 337:1357-58. References to the Main article: 1. Steer P, Alam MA, Wadsworth J, &Welch A. Relation between maternal haemoglobin concentration and birth weight in different ethnic groups. BMJ 1995; 310: 489-91 2. Roller O, Sandvei R, &Sagen N. High hemoglobin levels during pregnancy and fetal risk. Int J Gynaecol Obstet 1980; 18:53-56. 3. Garn SM, et al. Maternal hematologie levels and pregnancy outcome. Semin Perinatol 1981; 5:155-62. 4. Hemminki E. &Starfield B. Routine administration of iron and vitamins during pregnancy. Br J Obst Gynaecol 1978; 85: 404-410. 5. Valberg LS. Effects of iron, tin, and copper on zinc absorption in humans. Am J Clin Nutr 1984; 40:536-11. 6. Rogers J, Wood J, et al. Active versus expectant management of third stage of labour: the Hinchingbrooke randomised controlled trial. Lancet 1998; 351:693-99. 7. Odent M. Active versus expectant management of third stage of labour. Lancet 1998; 351: 1659. 8. Symonds EM. Aetiology of pre-eclampsia: a review. J R Soc Med 1980; 73: 871-75. 9. Naeye EM. Maternal blood pressure and fetal growth. Am J Obstet Gynecol 1981; 141: 780-87. 10. Kilpatrick S. Unlike pre-eclampsia, gestational hypertension is not associated with increased neonatal and maternal morbidity except abruptio. SPO abstracts. Am J Obstet Gynecol 1995; 419: 376. 11. Curtis S, et al. Pregnancy effects of non-proteinuric gestational hypertension. SPO Abstracts. Am J Obst Gynecol 1995; 418: 376. 12. Von Dadelszen P, Ornstein MP, et al. Fall in mean arterial pressure and fetal growth restriction in pregnancy hypertension: a meta-analysis. Lancet 2000; 355: 87-92. 13. Jarrett RJ. Gestational diabetes: a non-entity? BMJ 1993; n306: 37-38. 14. Jarrett RJ, Castro-Soares J, Dornhorst A. &Beard R. Should we screen for gestational diabetes? BMJ 1997; 315: 736-39. II. Understanding Health: From Fetal Vulnerability to Adult Adaptability FETAL VULNERABILITY The six months following conception undoubtedly represent the phase of human life with the highest risks of death. This is one aspect of fetal vulnerability. Today we are in a position to understand another aspect of this vulnerability: anything happening during the phase of intra-uterine formation can have irreversible and life long effects. In other words we learned in the 1990s that our health is to a great extent shaped in the womb. The subscribers of our newsletter and those who have had an overview of our data bank know that in any field of medicine there have been studies detecting links between what happened during fetal life and health conditions in adolescence, adulthood and old age. If you are not yet familiar with the concepts of primal health research, just explore the data bank via key words as diverse as, for example, life expectancy, obesity, prostate cancer,

breast cancer, recurrent miscarriages, schizophrenia, colonie diverticulosis, coronary heart disease, handedness, Parkinson's disease, cancer in childhood, dental caries, etc. If your research is prospective, you can use key words famine, emotional state in pregnancy, seasonality of birth, birth weight, alcohol in pregnancy, anoxia antepartum, abdominal circumference at birth, antipyretics, et cetera. In the current scientific context it is clear that all organs and functions have critical periods of development. It appears that many so-called adult diseases are in fact the long term consequences of early developmental defects. For example if there was something wrong during the period that is critical for the development of the pancreas, the main consequence may be, 50 years later, a non-insulin dependent diabetes. From an overview of the data bank one can conclude that the growth and development of the fetus is influenced by a great variety of factors such as the emotional state of the pregnant woman, her nutritional habits, and, today, the amount of fat-soluble synthetic chemicals which are stored in her adipose tissues. The very first weeks following conception represent a phase of extreme vulnerability. We previously mentioned an evaluation of urinary gonadotrophin levels during the two weeks following implantation (1). According to this study, the incidence of "post implantation loss" is as high as 43%. A recent animal experiment suggests that the maternal diet during the preimplantation period has long-term irreversible effects (2). Female rats had a low protein diet during only the four days following mating, the preimplantation period. They had a normal diet for the remainder of gestation. There were effects on birth-weight, postnatal growth rate, blood pressure and organ/body-weight ratios in either male or female offspring. Preimplantation embryos collected from dams displayed significantly reduced cell numbers, apparently induced by a slower rate of cellular proliferation. The low protein diet reduced insulin and amino acid levels, and increased maternal glucose blood levels by day 4 of development. Male fetuses are more fragile than female fetuses. According to an evaluation dated 1983, for 100 abortions of chromosomally normal females there are 132 males (3). Since that evaluation the male/female ratio of fetal deaths has been continuously increasing. This has been well demonstrated in Japan (4). An analysis of the vital statistics of Japan looked at the male/female ratio of miscarriages between 12 and 15 weeks gestation, that is an age when it is possible in most cases to identify the sex of the fetus by routine examination of external genitalia. This ratio increased from 2.52 in 1966, to 3.10 in 1976, to 6.16 in 1986, to 10.01 in 1996! The only interpretation one can offer for this alarming phenomenon is intrauterine pollution by hormonal disruptors and more precisely by oestrogen mimickers. Hormonal disruption is the effect, in particular, of many families of fat-soluble synthetic chemicals. Many of them are polychlorinated substances. That is why there is a spectacular increase of male fetal losses and also, among the survivors, multiple signs of impaired development of the genital tract: lower sperm counts (5,6), more undescended testicles (7), more abnormalities of the penis (8) and also more cancers of the testicle (9). It seems that this sort of cancer is in most cases the long term visible effect of intrauterine developmental defects. The specific vulnerability of male embryos and fetuses has been explained by the fact that all the fetuses have always been exposed to far higher levels of oestrogens than their pregnant mothers. This implies that testicles must develop much faster than ovaries, so that they can produce male sex hormones before their masculinity becomes submerged by high levels of oestrogens. It now seems that sex differentiation begins at conception, and the Y chromosome is involved in accelarating the growth of the male (XY) embryo (10). There is an advantage to win a race so that the testicles are differentiated before there is a high level of oestrogens. In other words male embryos must have a faster growth, that is a higher metabolic rate than females. It has been shown that in pre-implantation bovine embryos total glucose metabolism was twice as high in males compared with females (11). Among humans, after in vitro fertilization, the likelihood of a liveborn male is greater than for a female if at the time of transfer the number of cells per embryo is four or above (12). Finally the main difference between the sexes is that male embryos have a faster metabolic rate. Speed increases vulnerability. This new awareness of the importance of prenatal environmental factors has many practical implications. For example when one considers the issue of pollution and health one must keep in mind that intrauterine pollution should be the main preoccupation. This new awareness also leads to re-interpretation of old studies, such as twin studies:

twins share or do not share the same genes, but they always develop in the same uterus of a mother the same age, with the same emotional state, the same diet, the same blood pressure, etc. On the other hand both twins are not exactly in the same intra-uterine environment, because they are not in the same posture; one of them can receive more blood than the other, etc. In the past, when there were discussions about the comparative role of genetic and environmental factors at the root of a disease or a behaviour, it was as if the environmental factors start at birth. Today the prenatal enviromental factors are considered so crucial that we do not even need long and comprehensive studies of the specific vulnerability of the fetus. ANECDOTES OF ADULT ADAPTABILITY In contrast with fetal vulnerability, the capacity adults have to recover from adverse circumstances is amazing. During World War II, tens of thousands of human beings were held captive in such conditions of extreme privation that a great proportion of them could not survive. Half a century later we have at our disposal a sufficient amount of data to evaluate the health and the longevity of the survivors. In a previous newsletter (Winter 1993, Vol. 1, No. 3) we studied the biographies of well known people who survived quasi-experimental extreme deprivation during world war II, then came back to active life and died after the age of ninety. These thirteen anecdotes provided sufficient information to demonstrate the extreme adaptability of certain human adults. Because the systematic studies that are emerging now are about men, I find it useful to recall the main points of the biography of the two women we included originally in our list. Dr Cicely Williams, born in Jamaica and educated in England, was primarily responsible for the identification, in the 1920s, of Kwashiorkor, a nutritional deficiency which has ravaged children in third-world countries. After many years in Africa she went to Malaya. She was conducting a health survey in the remote province of Trengganu when Pearl Harbor was attacked. It took her weeks of danger and deprivation to reach Singapore where she arrived just as the Japanese invaded. She was imprisoned at the notorious civilian Changi gaol. After two years of near starvation, she was taken to the headquarters of the Kempe Tai, the equivalent of the Gestapo. After 'interrogation' she was put in a series of cages which, for the next four months, she shared with dead and dying men, forced to crouch down, and starved. After the war she went to America and did a post-graduate study at Johns Hopkins University. In 1948 she became the first advisor in maternal and child health to the World Health Organization. In her nineties she still spoke in Israel, Nepal, and Pakistan. Retired-Except on Demand was the title of her biography published by Sally Craddock. Cicely Williams died aged ninety-eight. Tsola Dragoicheva was a member of the Bulgarian communist party when she was arrested in September 1923 during an ill-prepared revolt. She was sentenced to fifteen years in prison, amnestied in 1924, but found herself once again behind bars after the communists had blown up one of Sophia's cathedrals, causing the death of one hundred and twenty prominent Bulgarians. After torture, she was condemned to death and saved from the gallows because she was pregnant. She spent the next nine years in prison, keeping her son with her. The child was later entrusted to party colleagues, who sent him to Moscow. After her release Dragoicheva studied in Moscow and came back to Bulgaria where she was elected to the Central Committee of the Bulgarian Workers' party. When the Germans invaded the U.S.S.R. in 1941 Dragoicheva was arrested again and sent to the women's detention camp at Sveta Nikola. From there she escaped. After the war she became a prominent figure in party and state and also wrote three substantial volumes of memoirs. She died aged ninety-nine and nine months. The son whose conception had saved her life became a heart surgeon. He should be about seventy-six now. It would be interesting to trace him. Since our winter 1993 newsletter there have been many deaths of famous people whose biographies demonstrate the huge capacity to recover from adverse situations in adulthood. The biography of Cardinal Ignatus Kung, Bishop of Shanghai, is typical of recent examples. In September 1955, Kung, along with several hundreds priests, was arrested and imprisoned. Several months later he was paraded in front of an angry mob at Shangai's pre-revolution dog racing stadium. In 1960 he was sentenced to life imprisonment. He was eventually released in 1987 (after 32 years of severe privation in Chinese prisons) and given the permission to travel abroad. After settling in Stamford, Connecticut, and recovering a better health, he traveled to Rome for a private audience with Pope John Paul II. He died in March 2000, aged 98. All these

anecdotes are valuable in that they inspire further research. In fact, more than fifty years after the end of World War II, we are reaching a time when relevant systematic studies can be published. SYSTEMATIC STUDIES In the months after the Japanese invasion of the Malay Peninsula in 1942, more than 140,000 Allied servicemen were captured by the Japanese. Conditions in the prison camps were so harsh that by the time the war ended between 25% and 40% had died. These prisoners experienced malnutrition, infections, exhaustion, severe psychological stress and diverse neurological syndromes. There have been concerns that the prisoners may have been exposed to slow-acting neurotoxins from plants such as cycads (13), which could have made them more liable to develop degenerative neurological disease. That is why the first large follow-up study regarding these prisoners of war focused on the risk of Parkinson's disease, without ignoring the death rates from other diseases. Using records held by the War Pensions Agency, a British team of researchers abstracted data on 11,915 British former prisoners of war held captive by the Japanese (14). 11,134 were traced. The numbers of deaths in this group between 1952 and 1997 were compared with those expected from national rates for the male population of England and Wales. The first amazing conclusion is that the overall mortality was significantly lower than expected: 8,796 deaths were expected but there were in fact 7,474 of them. Death rates from Parkinson's disease were slightly below the national average, though this difference was not statistically significant (35 deaths versus 43 expected). A similar pattern was seen for other degenerative neurological disorders (motorneuron disease, multiple sclerosis and dementia). The former prisoners had significantly lower than expected mortality from all major causes of death (coronary heart disease, cerebrovascular disease, all sorts of cancers and respiratory disease). They also had below average rates of death from tuberculosis and suicide. The only exception was the increased mortality from diseases of the liver such as cirrhosis and primary cancer of the liver. This fact can be easily interpreted. A large proportion of prisoners had hepatitis B and probably hepatitis C. Yet it is well known that the chronic carriers of these sorts of hepatitis have a greatly increased risk of both cirrhosis and liver primary cancer. This British study is by far the most authoritative one regarding the long term consequences of periods of severe malnutrition with frequent infections, exhaustion and intense psychological stress in adulthood. All the other studies were based on much smaller numbers. For example a recent Australian survey of World War II veterans aged 66-86 years compared former prisoners and non-prisoners (15). This study looked at the data regarding only 208 men and therefore could not provide statistically significant results. The overall conclusions of these anecdotes and systematic studies are easy to summarize. Those who were originally in a state of health good enough to survive the period of adverse circumstances finally had a longer life and were more healthy afterwards than the rest of the population. In other words a period of highly unfavourable lifestyle in adulthood does not alter significantly the life expectancy and the capacity to remain free of diseases. The contrast between fetal vulnerability and adult adaptability is a key for understanding the nature of health. What really matters is our 'Primal Health', that is the basic state of health in which we are at the end of the 'primal period' (16). This basic state of health is determined by a combination of genetic and early environmental factors. A new understanding of the word health, adapted to the current scientific context, indicates what the main preoccupations should be in the future. For those who are interested in the health of the unconceived generations, certain issues should prevail over all the others. This is the case of preconceptional preparation, intrauterine pollution, nutrition during pregnancy and other factors influencing the health and well being of pregnant women, factors influencing the physiological processes in the period surrounding birth, factors influencing the duration of breastfeeding, milk pollution, and long-term effects on health of early multiple vaccination, in particular. When looking at the most common topics developed in health magazines and the media in general, it is clear that the recommendations given to adults are presented as important. Adults are offered countless articles on their ideal weight, the best way to take exercise or the latest best diet. Yet we are in a position to claim that the lifestyle in adulthood has only minor effects on health. It will take time to change the focus because health magazines are read by adults and many adults are mostly interested in their own health. I came to similar conclusions when considering the comments inspired by my

book The Scientification of Love (17). One of the main themes of the book is that recent scientific advances inspire new questions regarding the development of the capacity to love. They also indicate that early experiences, particularly in the period surrounding birth, are critical in the development of the capacity to love. However many readers only remember the chapter on romantic love. We are in an adult-centered society. Perhaps the current research can bring adult attention to the critical issue and importance of the fetal environment. REFERENCES 1. Clarke CA, Mittwoch U. Changes in the male to female ratio at different stages of life. Brit J Obstet Gynecol 1995; 10: 677-79. 2. Kwong WY, Wild AE, Roberts P, Willis AC &Fleming TP. Maternal undernutrition during the preimplantation period of rat development causes blastocyst abnormalities and programming of postnatal hypertension. Development 2000; 127 (19): 4195-1202. 3. Hassold T, et al. Sex ratio in spontaneous abortions. Ann Hum Genet 1983; 47: 3947. 4. Mizuno R. The male/female ratio of fetal deaths and births in Japan. Lancet 2000; 356: 738-739. 5. Carlsen E, Giwercman A, Keiding N, &Skakkeback NE. Evidence for decreasing quality of semen during the past 50 years. BMJ 1992; 305: 609-13. 6. Auger J, Kunstmann, JM, Czyglik F, Jouannet P. Decline in semen quality among fertile men in Paris during the past 20 years. N Engl J Med 1995; 332: 281-5. 7. Jackson MB. John Radcliffe Hospital cryptorchidism research group. The epidemiology of cryptorchidism. Horm Res 1988; 30: 153-56. 8. Paulozzi LJ, Erickson D, &Jackson RJ. Hypospadias trends in two US surveillance systems. Pediatrics 1997; 100: 831. 9. Forman D, &Moller H. Testicular cancer. Cancer Surv 1994; 19-20: 323-41. 10. Burgoyne PS. A Y-chromosomal effect on blastocyst number in mice. Development 1993; 117: 341-45. 11. Tiffin GL, et al. Glucose and glutamine metabolism in pre-attachment cattle embryos in relation to sex and stage of development. J Reprod Fert 1991; 93: 125-32. 12. Pergament E, et al. Sexual differentiation and preimplantation cell growth. Human Reprod 1994; 9: 1730-32. 13. Spencer PS. Guam ALS/parkinsonism-dementia: a long-latency neurotoxic disorder caused by 'slow toxin(s)'in food? Can J Neur Sci 1987; 14: 347-57. 14. Gale CR, Braidwood EA, Winter PD, &Martyn CN. Mortality from Parkinson's disease and other causes in men who were prisoners of war in the Far East. Lancet 1999; 354: 2116-18. 15. Creasey H, Sulway MR, et al. Is experience as a prisoner of war a risk factor for accelerated age-related illness and disability? J Am Geriatr Soc. 1999; 47: 60-64. 16. Odent M. Primal Health. Century-Hutchinson London 1986. 17. Odent M. The Scientification of Love. Free Association Books. London 1999. III. Vaccinations: Prevention of Disease Can Be a Cause of III Health THE YEAR 2000 IN RETROSPECT The year 2000 will be remembered as a turning point in the history of vaccinations. Concepts that have been traditionally ignored in vaccination research became familiar. Terms such as 'ill health', 'good health' or 'non-specific effects on health' were suddenly widely used. Until recently the only questions raised in the medical literature have concerned the effectiveness and the specific side effects of a particular vaccine. These questions were often the basis of endless discussions between the pro- and anti-vaccination groups. The difficulty to surpass such a narrow viewpoint is rooted in the time when babies were offered only one or two vaccinations. Today the main question should concern the interactions between the great number of vaccines routinely offered to modern babies. Researchers, practitioners and parents should think first in terms of good health and bad health. This is not easy to do especially when you have been brainwashed with the dangerous concept of'preventive medicine', which suggests that health is the absence of disease and that the longer the list of diseases you prevent, the healthier you are. The year 2000 has offered opportunities to realize that the prevention of disease may itself be a cause of ill health. On the one hand we have learned from studies of the 'Gulf war syndrome'. On the other hand we have learned from studies about child mortality in the third world. SHOTS IN THE DESERT Everybody heard about the so-called 'Gulf war syndrome'. It is now well accepted that there were increased rates of ill health in those who served in the Gulf around 1990. The veterans reported such a great diversity of symptoms that doctors were obliged to create the new entity of 'CDC multisymptom illness' (CDC = Centers for Disease Control and Prevention). The important point is that researchers had to review their concepts and their usual vocabulary in order to explore the "role of vaccinations as risk factors for ill health in veterans of the Gulf war"(1). This was the exact title of a large study published in May 2000 in the

British Medical Journal (BMJ). The mere title clearly indicates that the objective was to study the long term non-specific effects on health of a complex combination of vaccinations. Of course our interest, in the framework of primal health research, is not in the vaccinations of adults, but in the vaccinations of babies. However we must be aware of the studies of multiple vaccinations of adults, because they undoubtedly influence the way we raise questions about vaccinations during the primal period. Valuable studies of the Gulf war syndrome are possible because tens of thousands of servicemen participated in the conflict. For example the UK deployed 53,462 military personnel. Many of them received biological warfare vaccines (anthrax and plague). Whooping cough vaccine was always associated with plague as an adjuvant. They also received routine vaccines such as tetanus, cholera, poliomyelitis, typhoid, yellow fever, hepatitis B and IgG for hepatitis A. In 1997, Rook &Zumla offered theoretical reasons to implicate multiple vaccinations as a possible cause of ill health in Gulf war veterans (2). All these vaccines tend to unbalance the immune system and to deviate it "towards Th2". They also underlined that stress hormones (cortisol) and pesticides tend to exaggerate such deviations. According to the study published in BMJ, multiple vaccines received during deployment multiplied by 5 the risks of having the 'multisymptom illness'. This study should be remembered for its historical interest. The questions regarding vaccinations were not raised in terms of effectiveness and side effects, but in terms of non-specific effects on health. The concept of ill health, which implies the concept of good health, was introduced in the mainstream medical literature. LESSON FROM GUINEA-BISSAU Guinea-Bissau, in West Africa, is one of the world's poorest countries. It has one of the highest rates of mortality in childhood. In such a context it is possible to use child survival as a criterion of health. A Danish team of researchers looked at child survival in order to study the non-specific effects on health of different vaccines (3). The study involved 15,351 women and their children born during 1990 and 1996. The vaccination schedule recommended in Guinea-Bissau is BCG and polio at birth; diphtheria, tetanus, and pertussis and polio at 6, 10 and 14 weeks; and measles at 9 months of age. The mortality over periods of 6 months was evaluated. The findings show that both BCG and measles vaccines halved child mortality. The significant reduction in mortality was unrelated to tuberculosis or measles deaths: it appears that BCG and measles vaccines have a non-specific beneficial effect on health. On the other hand, children who received the combination of diphtheria, pertussis and tetanus (DPT) and polio vaccines had a risk of death multiplied by 1.84. The authors interpret their findings with caution because the inquiries were performed in difficult circumstances. However selection biases are unlikely, because different vaccines were associated with opposite tendencies. The report of this recent study should be instrumental in transmitting the concept of non-specific effects on health of early multiple vaccinations in infancy. In fact, there have been previous studies in the third world suggesting that measles vaccines influence child mortality. As early as 1991 a prospective randomised study (86.7%) among the non-vaccinated in rural Senegal detected an increased mortality at 41 months among children who had received an high-titre measles vaccine at 5 months, compared to those who had received a standard low-titre vaccine (4). None of these deaths were related to measles. Another study in rural Senegal, published in 1993, demonstrated a divergent mortality for male and female recipients of low-titre and high-titre measles vaccine (5). In 1995 an analysis of all studies comparing mortality of unimmunized children and children immunized with standard titre measles vaccine in developing countries lead to the conclusion that standard measles vaccine has a beneficial effect which is unrelated to the specific protection against measles disease (6). These studies were conducted in countries as diverse as Bangladesh, Benin, Burundi, Guinea-Bissau, Haiti, Senegal and Zaire. AND WHAT ABOUT BABIES IN WEALTHY COUNTRIES? The lessons from the Gulf and from the developing countries inspire inescapable questions. What about the possible long-term effects on health of the complex vaccination schedules offered to babies in wealthy countries? It is currently difficult, even impossible, to provide valuable answers to such questions. Child mortality rates are so low that they cannot be used as criteria. Also vaccination rates are so high that it is difficult to establish a control group to compare the health of a vaccinated group with the health of a non-vaccinated group. The non-vaccinated children often belong to families who are unconventional where their

attitude to health is concerned. This means that there are many possible confounding factors that only a randomisation might eliminate. Unfortunately we cannot learn either from epidemiological studies of certain aspects of ill health, such as asthma or diabetes in childhood, which are mysteriously frequent in the industrialized societies. Researchers who try to detect risk factors for such diseases take into account a great number of variables but they always forget to look at the immunization status (7,8,9). In spite of all these difficulties the results of our three-step inquiry suggest that the non-specific effects on health of early multiple vaccinations are real in wealthy countries as well. Furthermore there are striking similarities between the results of our inquiry and the results of studies conducted in other contexts, particularly in the context of Guinea-Bissau. We also came to the conclusion that whooping cough vaccination, and the vaccinations usually associated with whooping cough, have a negative effect on health (10), while BCG has a positive effect (11). For those who did not read our summer 1994 newsletter (vol. 2, no.1) or our autumn 1998 issue (vol. 6, no.2) let us recall that we first analysed criteria of health in a population of 446 children (mean age 8 years) that was homogeneous in terms of infant feeding (all children had been breastfed more than a year and had received only breastmilk during the first 6 months). None of them had received BCG. In this particular population there were significant differences when classifying the children according to whooping cough (pertussis) vaccination (12). When presenting the results we focused on pertussis vaccination (always associated with diphtheria and tetanus) as a risk factor for asthma in childhood. To the question: 'Has your child ever been diagnosed as asthmatic?' there were 26 positive answers in the immunised group (10.69%) compared with 4 in the non immunized group (1.97%). The difference is highly significant (95% confidence interval 1.93-15.30). We did not find the same difference between the two groups with respect to the diagnosis of eczema (13). In fact there were significant differences when other criteria of health were considered. Among the 243 pertussis-vaccinated, 130 had ear infections versus 59 among the 203 not vaccinated. We also looked at the time spent in hospitals as a criterion of health. Among the vaccinated children 173 (71.2%) had never been hospitalized versus 176 (86.7%) among the non-vaccinated. More precisely, 53 children have been hospitalized for less than 5 days and 17 for more than 5 days in the vaccinated group, versus 24 and 3 in the other group. When we considered "other diseases" (i.e., not ear infection, asthma, eczema and whooping cough), there were 84 cases in the vaccinated group (34.6%) versus 49 in the non vaccinated group (24.1%). From this inquiry we could conclude that children who are not immunized against whooping cough are in better health than those who are immunised. The second step of our inquiry is represented by our study of 274 pupils of British Rudolf Steiner schools. 125 of them had been immunized against whooping cough versus 149 non immunized. Among the 125 pupils vaccinated against whooping cough, 23 (18.4%) were diagnosed as asthmatic, versus 6 (4.02%). The difference was once again statistically significant. The third step is represented by an analysis of the medical records of the 210 pupils of the French Sterner school La Mhotte. Pupils of Steiner schools belong to families whose lifestyles are apparently similar, whatever the side of the Channel. However there are differences where vaccinations are concerned. French immunised children usually receive BCG at birth or a very early age. None of the children who had received both whooping cough vaccination and BCG have been diagnosed as having asthma. We came to the conclusion that BCG protects whooping cough immunized children against asthma. This protective effect of BCG helps to explain differences between countries. In countries with the highest prevalence of asthma, BCG is not routinely offered (e.g., UK, New Zealand, Australia, Republic of Ireland). Before the fall of the communist system, BCG during infancy was routine practice in Eastern Europe. The rates of asthma in childhood and adolescence in such countries is comparatively low. School children in Leipzig, East Germany, born three years before unification, still had a comparatively low rate of asthma in 1995-96, whereas the prevalence of atopic sensitization was already increasing (14). The first conclusion of our inquiries is that we detected negative effects on health of pertussis vaccination-and the usually associated vaccines-while we detected positive effects of BCG. The second conclusion is that we have a lot to learn about the interactions between vaccinations. Today it would be ethical to start long term prospective randomised controlled studies.

This is the most reliable method to evaluate the ratio of benefits to risks for any medical procedure. The very first step is to divide a population into two (or more) groups by drawing lots (randomisation). One group is randomised to receive a treatment while another group is allocated to another treatment. Then there is a long period of follow-up, so that comparisons are possible. Where mass vaccinations are concerned, it would be unethical (or immoral) to continue the current programmes without starting prospective randomised controlled studies of the non-specific effects on health of different combinations of vaccines. HOW CAN INFORMED PARENTS DECIDE? It is difficult to play the role of parents at the dawn of the 21st century. Today parents are condemned to constantly make choices. Choices are more difficult in certain countries. There are differences between countries such as the UK, where vaccinations are not obligatory, and a country like France, where there is a list of obligatory vaccinations. Finally the questions raised by the parents are the same everywhere and the decisions are more or less open to choice. As long as early multiple vaccinations are not evaluated via the most reliable methods, the only hard data we have at our disposal are about effectiveness of a particular vaccine and possible short term adverse 'reactions'. For example nobody can evaluate the life expectancy of babies who received ten vaccinations compared with those who did not receive more than one or two. That is why parents (and health professionals) must take into account their beliefs, their intuition, their worries and their personal attitude regarding risk calculation. Meanwhile the only strategy one can suggest to parents is to try to shorten the list of vaccines the child will receive. They must look at the different vaccines one by one and take into account how serious is the disease the vaccine is supposed to prevent, how effective the vaccine is and what we know or suspect about the short- and long-term side effects. They must also take into account the geographical context. The risk of catching certain diseases depends on the place where people live Parents should begin by considering the components of the widely used combination diphtheria-tetanus-pertussis. They should focus first on pertussis (whooping cough) which is never obligatory but is routinely associated with the two others. It is not a very effective vaccine. There have been epidemics among vaccinated children (e.g. the Cincinnati epidemics). According to the Guinea-Bissau data and our data it has a detrimental effect on health. On the other hand the disease whooping cough may be life threatening during the first year of life. However, it is noticeable that in Japan and Sweden babies do not receive pertussis vaccination during the year following birth (in Sweden because this vaccine was excluded from the routine programme in 1979). Yet Japan and Sweden have the lowest infant mortality rates in the world (that is the rate of deaths before the age of a year). There is no doubt that the absence of pertussis vaccination is compatible with exceptionally low infant mortality rates. There is no risk of catching diphtheria in Western Europe or North America. The main reason parents still have to vaccinate against diphtheria is the desire to participate in a global effort to eradicate the disease. Tetanus vaccination is undoubtedly highly effective. It has long-lasting effects. The risks of adverse reactions are very low. On the other hand tetanus is a life threatening disease. Parents are in an uncomfortable situation when a child is injured or burnt. In the casualty department, the medical staff is obliged to inject an immune globuline (passive immunization), which can induce severe allergic reactions. They will start a vaccination at the same time. When the association of diphtheria-tetanus-pertussis is reduced to tetanus, it implies that the antigen load is minimal and the amount of ajuvants as well. These are aluminium hydroxide, a potent inducer of IgE response (15), a mercury derivative (but not in the USA), and formaldehyde. In industrialized countries the main reason to vaccinate against poliomyelitis is the desire to participate in a global effort to eradicate the disease. Today, in such countries, the only cause of paralytic polio is oral vaccination. In the USA, the last case of non vaccine induced paralytic polio occurred in 1979. The issues are different for those who plan to live in third world tropical countries. MMR (measles, mump, rubella) is highly topical. Many parents are cautious because they heard of MMR as a possible risk factor for chronic bowel diseases and autism. There is a lack of hard data. The absence of prospective randomised controlled studies leads to sterile discussions. The media made a misleading report of a Finnish study that was supposed to rule out 'categorically' the link between MMR and autism. In fact it was a non controlled study(16). This means that there was no possible comparison with a non

immunized population. This study was not designed to evaluate MMR as a possible risk factor for autism. One can understand parents who prefer to avoid MMR and also those who would like to do measles only. This is easy in certain countries (e.g. France). It is more difficult in the UK, because there is no monovalent vaccine available on the NHS. However it is possible. Parents can visit www.argonet.co.uk/users/jabs. Hib (Haemophilus influenza type b) is a rare cause of meningitis today and many adverse reactions have been reported. Mass vaccination against group C meningococci is a British phenomenon. Let us quote a 1999 commentary in the Lancet: "The introduction of any vaccine that targets only a fraction of the population of a bacterial pathogen should be viewed as a large-scale experiment in bacterial population biology." Let us recall that five of the 13 meningococcal serogroups commonly cause disease. The most common bacterial meningitis is related to group B, for which there is no vaccine. B meningococci disease has increased in 2000 (17). Why? The C vaccine called MMC ('meningococcal serogroup C conjugate vaccines') is undoubtedly effective compared with the plain C polysaccharide vaccines, but there is uncertainty about its long-term effects. Hepatitis B vaccine is included in the series routinely offered to babies. Parents who are themselves sero-negative may be reluctant to vaccinate their child. Let us recall that it is first a sexually transmitted disease. Certain health professionals can be at risk of being contaminated. Drug addiction with exchanges of needles is another risk factor. Contamination via pharmaceutical blood products is unlikely today. Finally parents have many reasons to postpone their decision, at least until puberty. Mass chicken pox is an American phenomenon. As for BCG, it is not routinely included in the vaccination programmes in English- speaking countries. According to the data we have at our disposal, it would not be wise to vaccinate against whooping cough children who have not previously received BCG. There is also the issue of children living in a family where there is a TB person. *** Our objective in this article is not to study in depth all the vaccines that can be used in infancy. It is just to suggest a strategy to parents who are obliged to make a choice. REFERENCES 1. Hotopf M, David A, et al. Role of vaccinations as risk factors for ill health in veterans of the Gulf war: cross-sectional study. BMJ 2000; 320: 1363-67. 2. Rook GAW, &Zumla A. Gulf war syndrome: Is it due to a systemic shift in cytokine balance towards Th2 profile? Lancet 1997; 349: 1831-33. 3. Kristensen I, Aaby P, &Jensen H. Routine vaccinations and child survival: Follow up study in Guinea-Bissau, West Africa. BMJ 2000; 321: 1435-9. 4. Garenne M, Leroy O, et al. Child mortality after high-titre measles vaccines: Prospective study in Senegal. Lancet 1991; 338: 903-07. 5. Aaby P, Samb B, Simondon F, et al. Divergent mortality for male and female recipients of low-titer and high-titer measles vaccines in rural Senegal. Am J Epidemiol 1993; 138: 746-55. 6. Aaby P, Samb B, Simondon F, et al. Non-specific beneficial effect of measles immunisation: Analysis of mortality studies from developing countries. BMJ 1995; 311: 481-5. 7. Isaac steering committee. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema. Lancet 1998; 351: 1225-1232. 8. Sears M. Epidemiology of childhood asthma. Lancet 1997; 350: 1015-20. 9. McKinney PA, Parslow R, et al. Perinatal and neonatal determinants of childhood type 1 diabetes. Diabetes Care 1999, 22 (6): 928-32. 10. Odent M. Long-term effects of early vaccinations. Primal Health Research Newsletter. Vol. 2. no. 1 (Summer 1994). 11. Odent M. Future of BCG. Lancet 1999; 354: 2170. 12. Odent M, Culpin E. &Kimmel T. Pertussis vaccination and asthma: Is there a link? JAMA 1994; 272: 592-3. 13. Odent M, Culpin E, &Kimmel T. Atopic eczema. Lancet 1994; 344:140. 14. Hurwitz EL, &Morgenstern H. Effects of diphtheria-tetanus-pertussis or tetanus vaccination on allergies and allergy-related respiratory symptoms among children and adolescents in the United States. J Manipulative Physiol Ther 2000 ; 23 (2): 81-90. 15. Von Muting E, Weiland SK, Fritzsch C, et al. Increasing prevalence of hay fever and atopy among children in Leipzig, East Germany. Lancet 1998; 351: 862-66. 16. Mark A, Bjorksten B, &Granstrom M. Immunoglobuline responses to diphtheria and tetanus toxoids after booster with aluminium-adsorbed and fluid DT-vaccines.Vaccine 1995, 13(7): 669-73. 17. Patja A, Davidkin I, et al. Serious adverse events after measles-mumps-rubella vaccination during a fourteen-year prospective follow-up. Pediatr Infect Dis J 2000; 19(12): 1127-34. 18. Ramsay ME, Andrews N, et al. Efficacy of meningococcal serogroup C conjugate vaccine in teenagers and toddlers in England. Lancet 2001; 357: 195-96. IV. Is Breast

Best? Beyond the Immediate Impassioned Responses "Outrageous!!!!" . . . "Arrogance of man" . . . "Tempest in a teacup" . . . "Breastfeeding promotion undermined yet again" . . . "Another breastfeeding obstacle" . . . No article in a serious medical journal has matched the uproar sparked by a study published in BMJ (British Medical Journal) about "Duration of breast feeding and arterial distensibility in early adult life" (1). Thirty-eight electronic responses appeared suddenly on the BMJ web site. A record! As a reference I looked at the number of electronic responses inspired by the previous paper published in the same issue of BMJ (2 responses) and by the following one (O response). All over the world the media mentioned the results of this study in a sensational mode. It makes me think of "The Satanic Verses" by Salman Rushdie, which has been dubbed "the most famous book most people will never read". Several of our subscribers asked me to comment on this paper, because they heard or read about it, without understanding exactly what is was about. In order to avoid a repeat of the Rushdie phenomenon our first step must be to reproduce in its entirety the abstract as it was published in BMJ on March 17, 2001. I even recommend you look at the full text: go to bmj.com in order to reach Pubmed/Medline. By typing the name of the first author (Leeson CPM) you'll have access to the full text. The researchers belong to The Medical Council Childhood Nutrition Research Centre, Institute of Child Health (London) and to The Vascular Physiology Unit, Great Ormond Street Hospital for Children (London). Objectives: To test the hypothesis that duration of breastfeeding is related to changes in vascular function relevant to the development of cardiovascular disease. Design: Population based observational study. Setting: Cambridge, England Participants: 331 adults (171 women, 160 men) aged between 20 and 28 years, born in Cambridge Maternity Hospital. Main outcome measures: Distensibility of brachial artery, type and duration of infant feeding, current lipid profile, and other cardiovascular risk factors. Results: The longer the period of breastfeeding the less distensible the artery wall in early adult life, with no sex differences (regression coefficient = 3.93 µm/month, 95% confidence interval 7.29 to 0.57, P = 0.02). However, in those breast fed for less than four months, arterial distensibility was not significantly reduced compared with an exclusively formula fed group. The vascular changes observed were not explained by alterations in plasma cholesterol concentration in adult life. Conclusions: Breast feeding in infancy is related to reduced arterial function 20 years later. These data should not alter current recommendations in favour of breastfeeding, which has several benefits for infant health. Further work is needed, however, to explore the optimal duration of breastfeeding in relation to cardiovascular outcomes. HISTORY A certain number of facts regarding the history of infant feeding must be emphasised before interpreting the results: * All the men and women participating in the study were born between 1969 and 1974. This period preceded the widespread use of "humanised formula", that is to say the introduction of vegetable fats in artificial milk. We must assume that the artificial milk consumed by most of the participants contained 100% cow's milk fats; it was therefore rich in saturated fat and contained a certain amount of trans fatty acids. * In the early 1970s it was common practice to discreetly top up breastfed newborn babies with artificial formula during the first days following birth. * In the early 1970s human milk pollution with polychlorinated chemicals had probably reached its maximum: PCBs had not yet been banned. All these synthetic chemicals are associated with lipoproteins and interact with lipid metabolism. Weak Points Some of the limitations and several weak points of this study appear when reading the full text. The authors sent invitations to 1526 people. 229 of them declined and 877 did not reply. Finally full details of vascular function, risk factors and infant feeding practice were available for only 331 subjects (21.7 % of those who were originally invited to participate). A retrospective study of a small sample of self-selected volunteers cannot be authoritative. There are contradictions between the two parts of "Results". In the first part we read that there was an inverse relation between duration of breastfeeding and arterial distensibility and also that, "as expected", reduced distensibility was related, in particular, to systolic blood pressure. In the second part we read that duration of breastfeeding was unrelated to blood pressure. If there is a relation between duration of breastfeeding and arterial distensibility and between arterial distensibility and blood pressure, it is difficult to believe that there is no relation between duration of breastfeeding and blood pressure. Arterial distensibility is

not a well established risk factor for cardiovascular diseases, although it appears to have biological plausibility. Its measurement implies sophisticated methods which use high resolution ultrasound. It has been experimented by only a tiny number of researchers. High blood pressure, on the other hand, is a well established risk factor. That is why I find it surprising that the authors did not mention and discuss the contradictions between their results and the results of three studies which are included in the Primal Health Research data bank. According to these studies blood pressure is raised among children and adults who were bottlefed. If you type Wilson AC' in the index by authors, you'll find the Dundee study, suggesting that blood pressure at the age of seven is significantly raised in those children who have been exclusively formula fed for the first 15 weeks of life compared to those who had received any breast milk (2). If you type 'Taittonen L' you'll find the results of a Finnish study suggesting that the blood pressure of post pubertal male and female subjects is reduced among those who were breastfed more than 3 months (3). If you type 'Singhal A' you'll find a unique prospective randomised control study of 216 premature babies who were given either donated banked breastmilk or formula (preterm formula or standard formula). Arterial blood pressure at age 13-16 years was lower in the 66 children assigned banked breastmilk (alone or in addition to mother's milk) than in the 64 assigned preterm formula. The proportion of intake as human milk in the neonatal period was inversely related to later arterial pressure. No differences were found between those who had term formula and those who had standard formula. The authors could conclude that their data "provide experimental evidence of programming of a cardiovascular risk factor by early diet and further support the long-term beneficial effects of breastmilk" (4). It is worth underlying that Professor A Lucas participated in both the banked breastmilk study and the arterial distensibility study. It is obvious that if such contradictions-which could not be ignored by the authors-had been highlighted and discussed in the report of the "distensibility study", the results of this study would not have been presented by the media as a sensational breakthrough and would not have sparked such an uproar. Despite That. . . Despite the many weak points of the study, its counterintuitive results, and the way it has been reported, however we must take into account the content of this paper. It suggests that, at least in industrialized countries, prolonged breastfeeding may lead to adverse cardiovascular outcomes later on in life. This is not the first study in the Primal Health Research data bank leading to such conclusions. Go to the authors index and type 'Fall CH'. You'll find a study about men born in Hertfordshire at the beginning of this century (5). Those who had still been breastfed aged 1 year had higher rates of coronary heart disease 60-70 years later compared with the expected rate for men of that age. If you type 'Mott GC' you'll find the report of one in a series of experimental studies with baboons by researchers in San Antonio, Texas (6). Exclusive breastfeeding throughout infancy, followed by a diet high in saturated fats, was associated with an abnormal lipid profile and more arterial fatty streaks in mature animals. The concordant conclusions of these studies from complementary perspectives must be looked at seriously. They are not implausible. One can hypothesize than prolonged breastfeeding tends to programme the metabolism of lipids in a way that is not an adaptation to the current dominant western diet, high in saturated fatty acids and trans fatty acids. Homo sapiens was originally adapted to a diet rich in long chain polyunsaturated fatty acids, with a high ratio of omega 3 to omega 6 and a certain balance between food from the land and food from the sea. Comparable conclusions may be inspired by another study included in our data bank. This study also reveals possible negative effects of breastfeeding on a particular aspect of human health in wealthy industrialised countries. The researchers collected data regarding 3856 three year-old children participating in health check up programmes in 60 Japanese municipalities between October to December 1997 (7). They divided the children in three groups according to the mode of infant feeding (breast milk, artificial milk and mixed feeding). The rate of atopic dermatitis was slightly higher in the breastfed group (odds ratio 1.16). Once more, in order to interpret the results, we must remember to which environment Homo Sapiens was originally adapted to. In a tropical wild environment one of the main roles of the human immune system is to adapt to a real symbiosis with a great variety of parasites, particularly intestinal worms. This implies the development of a family of antibodies called IgE. These IgE are more or less redundant in wealthy industrialized

countries. However their levels may remain too high for the main role they are originally supposed to play. They tend to over react in the presence of certain antigens. It is well known that atopy is associated with high levels of IgE. We should not overlook the positive aspect of all these studies. Even if they are not always authoritative they help realising that Homo Sapiens is not perfectly adapted to the 21st century dominant western lifestyle. THE OTHER SHJE OF THE COIN The bright side of the coin cannot be tarnished by a small number of data provided by retrospective studies difficult to interpret. It would take volumes to report a great variety of published studies confirming the positive effects of breastfeeding on vital aspects of health. Everybody knows about the spectacular positive effects of breastfeeding in third world countries where the rates of child survival can be used as criteria of health. There are countless references regarding the effects of breastfeeding on the risks of infections, particularly gastrointestinal infections (8, 9, 10, 11), ear infections (12, 13, 14), lower respiratory tract infections (15, 16, 17, 18), meningitis (19, 20), and necrotising enterocolitis (21). In the field of paediatric surgery, breasfeeding has a positive effect on the incidence of acute appendicitis (22), inguinal hernia (23), undescended testicles (24), and hypertrophie pyloric stenosis (25). The preventive effects of breastfeeding have been demonstrated in common chronic illnesses such as multiple sclerosis (26), rheumatoid arthritis (27) and insulin dependent diabetes (28, 29, 30). All these studies can be found in our data bank. When considering the long-term effects of breastfeeding, we must not think only in terms of disease prevention and even of health promotion, but also in terms of human development in general. A well-known study assessed the LQ. at 7 1/2 to 8 years of 300 children born preterm (31). All of them were fed by a tube passed through the nose to the stomach. Those who were fed mother's milk had an 8.3% advantage in IQ, even after adjustment for differences between groups in mother's education and social class. There have been many other studies demonstrating the benefits of human milk on intelligence quotient, speech abilities (32,33) and different aspects of cognitive development (34, 35). In the context of the year 2001, we must give a special importance to a series of Dutch studies whose objectives were to evaluate the effects on neurological, mental and psychomotor development of early exposure to polychlorinated chemicals (36, 37, 38). No negative effects of exposure to PCBs and dioxin through breast milk could be detected until the age of six years. Today, in spite of human milk pollution, the well known benefits of breastfeeding outweigh and even counteract the adverse developmental effects of PCBs and dioxins during fetal life. Intrauterine pollution is the main preoccupation. Similar conclusions were provided by an American study regarding women who consume fish from the lake of Michigan, highly polluted with PCBs (39). The children were followed up to age 11. An entire newsletter would be needed to summarize the known effects of breastfeeding on maternal health. There is strong evidence that women who breastfeed are at lower risk of breast cancer before menopause (40, 41, 42). An entire book has been written to emphasize the importance of developing the capacity to love during the short critical period when birth physiology connects with the physiology of lactation (43). LACTATION STARTS BEFORE THE BABY is BORN One of the most common reactions to the BMJ article is the fear that it might undermine breastfeeding promotion. There is still a tendency to focus on the role of promotion and to overlook more important issues. Today the main obstacle is not the failure of breastfeeding promotion. Talk to your hairdresser or your taxi driver and you'll realise that everybody knows that "breast is best". Today the main preoccupation is that most women who stop breastfeeding before two or three months would have liked to have continued for longer. Our understanding of the connections between birth physiology and the physiology of lactation helps understanding the current difficulties. We have at our disposal a sufficient amount of data to claim that the duration of breastfeeding is to a great extent determined during the period surrounding birth. In order to interpret a Swedish study, published in 1996, let us first recall that to be effective the hormone oxytocin must be released by fast pulsations rather than on a continuous mode. This study demonstrated that 2 days after birth, during a breastfeeding session, women who gave birth vaginally release oxytocin in a "pulsatile" way, compared with women who gave birth by emergency caesarean section (44). Furthermore there is a correlation between the "pulsatility" of oxytocin release and what the duration of exclusive breastfeeding will be. An Italian team demonstrated that beta-

endorphin concentrations in the milk of mothers who delivered vaginally are significantly higher four days after birth than levels of mothers who underwent caesarean section (45). It is probable that one of the effects of milk opiates is to induce a sort of addiction to mother's milk. The authors of this study are now evaluating the links between beta endorphin concentrations in colostral milk and the duration of breastfeeding. Physiological data are supported by clinical observation. A survey in Warwickshire (UK) indicates that women who had put their own babies to the breast for the first feed were more likely to be still breastfeeding (71%) at around six weeks than those who had someone else put the baby on for them (38%) (46). It is obvious that when women give birth in physiological conditions and the first contact between mother and baby is not disturbed, there is no need for somebody else putting the baby at the breast. If the duration of breastfeeding is to a great extent determined in the period surrounding birth it would be surprising that the current situation can easily be improved. We are at a turning point in the history of childbirth. Until recently, in order to have a baby, a woman was obliged to release a complex cocktail of hormones that play a key role in the initiation of lactation. Today, in many industrialised countries, for the first time in the history of mankind, most women give birth without releasing such hormones. Either they rely on substitutes for natural hormones (drip of synthetic oxytocin plus epidural anaesthesia) or they give birth by caesarean section. The issue of breastfeeding cannot be dissociated from the issue of childbirth. REFERENCES 1. Leeson CPM, Kattenhorn M, Deanfield JE, &Lucas A. Duration of breast feeding in early adult life: population based study. BMJ 2001; 322: 643-7. 2. Wilson AC, Stewart Forsyth J, Greene SA, Irvine L, Hau C, &Howie PW. 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