MedCrave
Step into the World of Research
International Journal of Pregnancy & Child Birth

Mini Review
Open Access
CrossMark

Fetal harmful environments and the related consequences: the answers to our queries

Abstract

The fetal origin of adult disease (FOAD) hypothesis makes the physician reflect on how fetuses that have not reached their genetical growth potential (intrauterine growth retardation, IUGR) due to different nutritional deprivation situations, may be at risk of later adult diseases.In this mini review, we try to characterize how these IUGR fetuses make roll a series of metabolic mechanisms with the aim of protecting themselves in low nutritional-oxygenation states, and how consequently these mechanisms may interact leading to the FOAD explanation. We have been working in the study of different maternal-fetal complications for more than 20 years, dealing with different pregnancy disorders and prenatal diagnosis, such as: fetal lung maturation, maternal infections, endometriosis, computerized cardiotocography, maternal platelets alterations, color flow velocimetry, etc.[1–4] At a certain point, one of my team leaders asked: what is the future of these IUGR fetuses? He passed the way a few years ago. Nevertheless, that question remained fixed in my mind. The fetal origin of adult disease (FOAD) was an argument of one medical Congress a couple of years ago, and I have observed the gap of knowledge concerning this interesting theory in the international literature worldwide. Since that time, I went deep in this theory, and these pages represent a brief comment regarding the FOAD hypothesis.

Keywords: short term, long term consequences, nutritional, pregnancy disorders

Volume 4 Issue 6 - 2018

Juan Piazze
ASL Frosinone (Casa della Salute di Ceprano-Ospedale SS Trinità di Sora), Italy

Correspondence: Juan Piazze, ASL Frosinone (Casa della Salute di Ceprano-Ospedale SS Trinità di Sora), Italy, Email clowdpia@me.com

Received: October 08, 2018 | **Published:** November 13, 2018

The low weight fetal adaptation

David Barker, an epidemiologist, in more than one decade has evidenced throughout anthropological and epidemiological studies, that during fetal life, during rapid cell division stages, tissues and vital organs enter the so called "critical periods" of development. Regarding the FOAD theory, the nutritional derangement during fetal life may cause the reprograming of the regulation of the circulatory system, the insulin resistance in the muscle tissues and in the liver, with the aim of directing fetal resources to brain and heart.[5,6] Economists have also been active in demonstrating that various environmental factors can have negative impacts on the developing fetus, even at levels previously thought harmless. Later-life impacts extend to "bread and butter" economic outcomes, including educational attainment and wages.[7] Recent studies suggested that the balance of maternal protein and carbohydrate intakes during pregnancy is related to blood pressure in the offspring.[8]

Low birth weight and adult cardiovascular disease

By using 1911-1930 birth records for one English county (Hertfordshire), Barker showed that lower birth weight and weight at one year were associated with an increased risk of death from CHD and stroke.[9–12]

Cardiovascular function

Different authors have considered the potential use of arterial intima media thickness and carotid stenosis in order to identify in IUGR babies the asymptomatic vascular disfunction.[13–16]

Obesity

People who were heavier at birth tend to become 'fatter' adults as measured by body mass index. However, this may reflect increased lean mass rather than adiposity. Further, gestational diabetes produces a U- shaped or J- shaped relationship between birth weight and adult type2 diabetes.[17]

Neurodegenerative disease

Fetal growth restriction plays a fundamental role in the short term/ long term outcomes, determining a high incidence of cerebral deficit and also of neurological development, other than the lower weight. These consequences may not be evident up to late infant development, and may lead to a low scholastic situation, memory deficit linguistic and visual defects and even in the motorial development. Infants with a low cranial circumference show a high risk of cognitive derangement[18] and it has also been observed that a low birth weight may be related to a lower QI in infants at 11 years.[19]

Kidney glomerular disfunction

In IUGR babies, nephrons are reduced in number, mostly because of shorter kidneys. However, the kidneys glomerular flow is increased, and this in order to compensate a regular glomerular filtration velocity. This may lead to hypertension and glomerular hypertrophy, leading to a systemically hypertension and to an increased sodium resorption and finally to glomerular damage with consequent albuminuria and glomerulosclerosis.[20]

Endothelial disfunction

An endothelial disfunction is an event that precedes atherosclerosis that precedes the structural changes in the vascular wall. Different groups hypothesize that the atherosclerosis process begins in the infant period, and it developed in a silent way, before events such as myocardial infarction or ictus verify.[21]

May "fetal origin of adult disease" change our points of view?

Since the first description of "critical periods" of development,

David Barker opened a path leading to understand how fetuses in danger and with deranged growth, that develop the "brain sparing" effect in order to redirect the resources to brain and heart in a survival maneuver, may reprogram the whole system explaining further adult diseases. Recently, Zanetti et al, assessing the causal relationship of different associations using even Mendelian randomization, stated that that lower birthweight, used as a proxy for IUGR, is causally related with increased susceptibility to coronary artery disease and Type 2 Diabetes Mellitus.[22] Chronic diseases are not the inevitable lot of humankind. They are the result of the changing pattern of human development. Prevention of chronic disease and an increase in healthy ageing require improvement in the nutrition of girls and young women.[23]

FOAD hypothesis offers us a new view regarding the management of IUGR fetuses lives. If my friend and tutor were alive, we would continue to chase for the links that may lead to correlate prenatal, natal and post natal life mechanisms by which metabolism, body composition and growth may permanently be affected in later life.

Acknowledgments

None.

Conflicts of interest

The author declares there are no conflicts of interest.

References

1. Piazze JJ, Anceschi MM, Maranghi L, et al. The biophysical/biochemical test. A new marker of fetal lung maturity in borderline cases. *J Reprod Med*. 1999;44(7):611–615.
2. Neerhof MG, Haney EI, Silver RK, et al. Lamellar body counts compared with traditional phospholipid analysis as an assay for evaluating fetal lung maturity. *Obstet Gynecol*. 2001;97(2):305–309.
3. Piazze J, Anceschi MM, Cerekja A, et al. Nuchal translucency as a predictor of adverse pregnancy outcome. *Int J Gynaecol Obstet*. 2007;98(1):5–9.
4. Meloni P, D'Angeli I, Piazze J, et al. First trimester PAPP-A levels associated with early prediction of pregnancy induced hypertension. *Hypertens Pregnancy*. 2009;28(4):361–368.
5. Barker DJP, Osmond C, Winter PDW, et al. Weight in infancy and death from ischemic heart disease. *Lancet*. 1989;2(8663):577–580.
6. Barker DJP. The fetal origins of an adult disease. *Fetal and Maternal Medicine Review*. 1994:671–680.
7. King H, Aubert RE, Herman WH. Global burden of diabetes, 1995–2005: prevalence, numerical estimates and projections. *Diabetes Care*. 1998;21(9):1414–1431.
8. Barker DJP, Osmond C, Simmonds SJ, et al. The relation of small head circumference and thinness at birth to death from cardiovascular disease in adult life. *BMJ*. 1993;306(6875):422–426.
9. Martyn CN, Barker DJP, Osmond C. Mother's pelvic size, fetal growth and death from stroke and coronary heart disease in men in the UK. *Lancet*. 1996;348(9037):1264–1268.
10. Frankel S, Elwood P, Sweerman P, et al. Birthweight, body mass index and incident coronary heart disease. *Lancet* 1996;348(9040):1478–1480.
11. Barker M, Robinson S, Osmond C, et al. Birth weight and body fat distribution in adolescent girls. *Arch Dis Child*. 1997;77:381–383.
12. Barker DJP. Adult consequences of fetal growth restriction. *Clin Obstet Gynecol*. 2006;49(2)270–283.
13. Goodfellow J, Bellamy MF, Gorman ST, et al. Endothelial function is impaired in fit young adults of low birth weight. *Cardiovas Res*. 1998;40(3):600–606.
14. Weyer C, Pratley RE, Lindsay RS, et al. Relationship between birthweight and body composition, energy metabolism and sympathetic nervous system activity later in life. *Obes Res*. 2000;8:559–565.
15. Gale CR, Martyn CN, Kellingray S, et al. Intrauterine programming of adult body composition. *J Clin Endocrinol Metal*. 2001;86(1):267–272.
16. Koklu E, Ozturk MA, Kurtoglu S, et al. Aortic intima-media thickness, serum IGF-I, IGFBP-3, and leptin levels in intrauterine growth-restricted newborns of healthy mothers. *Pediatr Res*. 2007;62:704–709.
17. Fall CHD, Barker DJP, Osmond C, et al. Relation of infant feeding to adult serum cholesterol concentration and death from ischemic heart disease. *BMJ*. 1992;304:801–805.
18. Fattal-Valevski A, Toledano-Alhadef H, Leitner Y, et al. Growth patterns in children with intrauterine growth retardation and their correlation to neurocognitive development. *J Child Neurology*. 2009;24(7):846–851.
19. Shenkin SD, J M Starr, A Pattie, et al. Birth weight and cognitive function at age 11: the Scottish Mental Survey 1932. *Arch Dis Child*. 2001;85:189–197.
20. Luyckx VA, Brenner BM. Low birth weight, nephron number and kidney disease. *Kidnet Int*. 2005;68:68–77.
21. Jarvisalo MJ, Jartti L, Nanto-Salonen K. Increased aortic intima-media thickness: a marker of preclinical atherosclerosis in high risk children. *Circulation*. 2001;104(24):2943–2947.
22. Zanetti D, Tikkanen E, Gustafsson S, et al. Birthweight, Type 2 Diabetes Mellitus, and Cardiovascular Disease: Addressing the Barker Hypothesis with Mendelian Randomization. *Circ Genom Precis Med*. 2018;11:e002054.
23. Barker DJP. Sir Richard Doll Lecture. Developmental origins of chronic disease. *Public Health*. 2012;126(3):185–189.