Metabolic programming and early nutrition.

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Abstract

Recent studies on the early programming of adult metabolic disease have shed light on the molecular effects of early environment on long-term health. Studies on the placental interface, maternal nutrition, oxygen exposure, hazardous events, and infection are included, as are studies examining the roles of intrauterine food availability and the early postnatal environment. The epidemiological evidence for the programming of metabolic disease will be examined in this review, along with an overview of the numerous research that has used animals to simulate the results of metabolic phenotypic outcomes. Evidence supporting the suggested molecular pathways and the possibility of intervention will also be covered. Fasting hyperglycaemia, impaired fasting glucose, impaired glucose tolerance, central obesity, systemic inflammation, hypertension, decreased HDL cholesterol, and raised triglycerides are only a few of the symptoms that make up the metabolic syndrome. There is no disputing the widespread nature of the metabolic syndrome, with prevalence in the US estimated at 25% of the population, despite some controversy over causality and the proportional weight given to each of these criteria in the formal classification of the condition.

Keywords: Metabolic disease, Maternal nutrition, Hyperglycaemia.

Introduction

One of the key elements of metabolic syndrome is poor glucose homeostasis, which is just one of the numerous characteristics mentioned. Type-2 diabetes is largely caused by islet beta cell failure and/or resistance to insulin's effects in skeletal muscle, the liver, and adipose tissue. Beta cell dysfunction is caused by insufficient insulin generation and secretion by the islets' beta cells, whereas insulin resistance is defined by insufficient uptake and storage of glucose and amino acids for use as fuel. Although type-2 diabetes is typically diagnosed in people over the age of 40, it can also develop in specific ethnic groups, such as South Asians and African-Caribbean, after the age of 25 [1].

Principle of programming

The amount and quality of nutrition acquired through the maternal-fetal interface, or placenta, that is, during early blastocyst cell division, implantation into the maternal endometrium, trophoblastic invasion, and subsequent patterning, are all controlled by this interface. The trajectory of foetal growth can be hampered if foetal needs are not satisfied by maternal food supply, which could have an impact on long-term health. On the other hand, maternal substrate availability that is more than foetal needs can potentially superimpose variations from typical growth patterns and so confer disease outcomes over the course of a person's life. Fetal programming is the term used to explain the event that occurs in utero and involves the hardwiring of these abnormalities into every cell of the organism, hence determining future disease risk [2].

Genes: Fatal insulin hypothesis

Rare glucokinase gene mutations have been linked to low birth weight and the emergence of maturity onset diabetes of the young, a rare monogenic form of diabetes. This information served as the foundation for the "Fetal Insulin Hypothesis," which postulates that gene variations or polymorphisms that affect insulin production may contribute to both lower foetal growth and an increased risk of type 2 diabetes [3].

Role of the placenta

The placenta regulates foetal growth and development by enabling nutrition and gas transport, as well as by synthesising and secreting steroid and peptide hormones. Placental disruption is suggested as a key cause of human IUGR and programming of adult cardiovascular and metabolic disease. The growth and differentiation of the trophoblast, which comprises receptors, transporters, and enzymes, as well as vascular development and blood flow, determine how successfully it accomplishes this. Epidemiological data has connected foetal programming to low birth weight and low placental weight. Pregnancies complicated by diabetes, IUGR, and preeclampsia with hyperglycemia in the first trimester leading to up-regulation of glucose transporters and accelerated foetal growth in late gestation have been found to alter the expression of glucose and amino acid transporters in the placenta [4,5].

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It also includes:

- Maternal calorie restriction
- Maternal protein restriction
- Catch-up growth
- Maternal micro-nutrient restriction
- Maternal hypoxia
- Intrauterine artery ligation
- Maternal glucocorticoid exposure
- Gestational diabetes
- Maternal high fat feeding/over-nutrition

A programming of epigenetics

Epigenetic pathways offer a fascinating way to explain how a person's early environment may impact their vulnerability to long-term chronic diseases. More and more studies link epigenetic dysregulation to cancer, and numerous cancer forms exhibit defining epigenetic alterations such as histone modifications, hyper methylation of particular genes, and hypomethylation of the entire genome. Global hypomethylation is one of these and is thought to begin early in transformation, as well as being crucial for tumour development. Multiple investigations that found widespread hypomethylation in the nearby healthy tissue support this theory, indicating that this shift is an early stage of the transformation process that leads to karyotypic instability and other carcinogenic events.

Conclusion

If evaluated out of a broad perspective, it is amazing to see that, without exception, every single animal model of a substandard early life eventually leads to an offspring phenotype of metabolic disease. Our understanding of disease pathways has been enhanced by each of these models, which intersect at a point of metabolic dysregulation and proceed to linked diseases such as cardiovascular disease, hypertension, and cancer from this point. The discovery of deep sequencing is just one of many recent technological developments that should help to provide solutions to some of the outstanding mechanistic issues. We remain a long way from any form of human intervention, despite recent animal studies' suggestion that maternal food supplementation with methyl donors and cofactors in late pregnancy can mitigate the metabolic effects of growth restriction.

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