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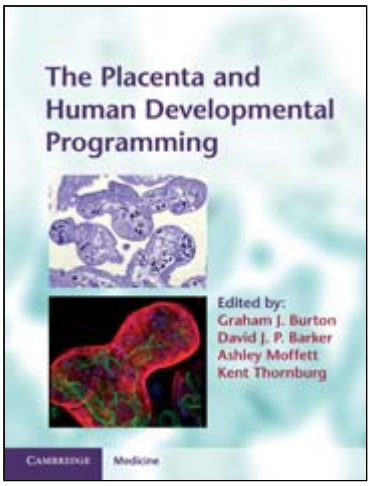

The Placenta and Human Developmental Programming

Edited by: Graham J. Burton, Department of Anatomy, University of Cambridge

Edited by: David J. P. Barker, MRC Epidemiology Resource Centre, University of Southampton

Edited by: Ashley Moffett, Department of Pathology, University of Cambridge

Edited by: Kent Thornburg, Department of Obstetrics and Gynecology, Oregon Health and Sciences University, Portland, OR

View All Contributors

Graham J. Burton, David J. P. Barker, Ashley Moffett, Kent Thornburg, Johan G. Eriksson, Eero Kajantie, Saleh H. Alwasel, Caroline H. D. Fall, Tessa J. Roseboom, Clive Osmond, Alan A. Jackson, Graham Burdge, Karen Lillycrop, Tom P. Fleming, Eric Jauniaux, Benjamin Tycko, Rosalind John, Elizabeth J. Radford, Anne C. Ferguson-Smith, Robert Pijnenborg, Lisbeth Vercruysse, Anthony M. Carter, Irene Cetin, Emanuela Taricco, Lorna G. Moore, Thomas Jansson, Theresa L. Powell, Owen R. Vaughan, Alison J. Forhead, Abigail L. Fowden, Gordon C. S. Smith, Perry F. O'Tierney, Terry Morgan, Samantha Louey, Cyrus Cooper, Laura Goodfellow, Nicholas Harvey, Susie Earl, Christopher Holroyd, Zoe Cole, Elaine Dennison, Robert Boyd, Richard Boyd

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Developmental programming is a rapidly advancing discipline of great importance to basic scientists and health professionals alike. This text integrates, for the first time, contributions from world experts to explore the role of the placenta in developmental programming. The book considers the materno-fetal supply line, and how perturbations of placental development impact on its functional capacity. Chapters examine ways in which environmental, immunological and vascular insults regulate expression of conventional and imprinted genes, along with their impact on placental shape and size, transport, metabolism and endocrine function. Research in animal models is integrated with human clinical and epidemiological data, and questions for future research are identified. Transcripts of

Chapter 2

The maternal and placental origins of chronic disease

David J. P. Barker, Johan G. Eriksson, Eero Kajantie, Saleh H. Alwasel, Caroline H. D. Fall, Tessa J. Roseboom and Clive Osmond

Introduction

Fetal programming is the process whereby environmental influences acting during development alter gene expression and program the body's structures and functions for life. There is now a body of evidence showing that chronic diseases of adult life, including cardiovascular disease, type 2 diabetes and certain cancers, originate through this process. These diseases are initiated by adverse influences during development [1]. These adverse influences may also slow the growth of the fetus and reduce its body size at birth and during infancy. Compared with people whose birth weights were towards the upper end of the normal range, those whose birth weights were towards the lower end have (a) reduced functional capacity, including fewer nephrons in the kidney; (b) altered metabolic settings, including insulin resistance; and (c) altered production of hormones, including stress and sex hormones. In a recent review the National Institute of Child Health concluded that 'coronary heart disease, the number one cause of death among adult men and women, is more closely related to low birth weight than to known behavioural risk factors' [2]. To date, most epidemiological observations that have demonstrated this link between prenatal life and later disease have used birth weight as the marker of early life. A baby's birth weight reflects its success in obtaining nutrients from its mother [3]. The source of these nutrients is not only the mother's current diet, but her nutritional stores and metabolism, which are the product of her lifetime's nutrition [4]. A baby's birth weight also depends on the placenta's ability to transport nutrients to it from its mother [3]. The placenta seems to act as a nutrient sensor, regulating the transfer of nutrients to the fetus according to the mother's ability to deliver them and the fetus's demands for them [5]. At the outset it is reasonable to assume that how the placenta programs the fetus will depend on the mother's lifetime nutrition.

This review uses new epidemiological data to examine the role of materno-placental interactions in initiating chronic disease in the offspring. It has been possible to pursue this issue epidemiologically because placental function and maternal nutrition are reflected in placental size at birth and in maternal body size, and these can be related to the later occurrence of disease.

Measurements of placental size and shape that reflect its function

The size, weight and shape of the placenta are all subject to wide variations [6]. Its size reflects its ability to transfer nutrients [7]. Small babies generally have small placentas but, in some circumstances, an undernourished baby can expand its placental surface to extract more nutrients from the mother [8]. Low placental weight at birth has been shown to predict hypertension and coronary heart disease in later life [9,10]. The weight of the placenta, however, does not distinguish its surface area from its thickness. In order to increase the surface for nutrient and oxygen exchange, the placenta can increase the surface of its villi, expand its invasion across the surface of the uterine lining or invade the maternal spiral arteries more deeply. The long-term consequences of these three responses may be different.

Placental shape

In the last century the surface of the placenta was described as being either 'oval' or 'round' [11–13]. In order to describe the extent to which the surface was more oval than round, two so-called 'diameters' of the surface were routinely recorded in some hospitals, a maximal diameter (the length of the

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