Developmental Programming of Neuroendocrine and Behavioral Pathologies

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Preface

This monograph addresses a critical domain of contemporary medical science—functional teratology, the study of predominantly functional disorders in the offspring of mothers exposed to adverse exogenous or endogenous influences during pregnancy or other vulnerable stages of early postnatal development. Such disruptions may remain latent at birth yet manifest later in life as chronic physiological or behavioral pathologies. Research in this field is inherently multidisciplinary, integrating physiology, pathophysiology, endocrinology, psychology, obstetrics and gynecology, as well as internal medicine.

It is now well recognized that the absence of overt teratogenic abnormalities in the newborn does not ensure a normal trajectory of subsequent development and health. This is especially pertinent for the neuroendocrine system, metabolic regulation, behavior, adaptive responses to homeostatic challenges, endocrine gland function, reproductive health, and immune competency. When such disorders become evident only in adulthood, reconstructing the maternal conditions during pregnancy and identifying the likely etiological factors is exceedingly difficult. Experimental studies in animals with relatively short life spans (rats, mice, hamsters) therefore provide a valuable opportunity to model early-life exposure to adverse influences and to assess their consequences for offspring health.

The first and second trimesters of human pregnancy are considered the most sensitive periods for intrauterine development. The first trimester is characterized by embryonic morphogenesis, whereas the second is marked by the maturation of the fetus's regulatory physiological systems, particularly the neuroendocrine system. One of the most critical processes occurring during this developmental window is the sexual differentiation of the brain, which programs sex-specific behavioral patterns, neuroendocrine regulation of gonadal function, and responses to stress.

Taking these considerations into account, the author and collaborators focused their research primarily on the final week of gestation in rats and, in some cases, on the first postnatal week. This strategy is justified by the fact that, compared with humans, early ontogenesis in rodents is developmentally delayed, with maturation of the neuroendocrine system occurring largely during this period.

I wish to express my deep respect and sincere gratitude to my colleagues for their professionalism and their dedicated contributions to the development of the scientific

field to which this monograph is devoted. Without their committed and productive efforts, the present work would not have been possible.

Alexander Reznikov

Table of Contents

Abbreviations

AGD - anogenital distance

AVP – arginine vasopressin

COMT - catechol-O-methyltransferase

CRH – corticotrophin-releasing hormone

E_2D - estradiol-17β diacetate

GABA - γ-aminobutyric acid

GD - gestation day

GnRH - gonadotropin-releasing hormone

HPA - hypothalamic-pituitary-adrenal

HPG - hypothalamic–pituitary–gonadal

LH - luteinizing hormone

LHRH - LH-releasing hormone

MBH - medial basal hypothalamus

MDA - malone dialdehyde

m.m. - molecular mass

MPN - medial preoptic nucleus

PCOS – polycystic ovary syndrome

PND – postnatal day (s)

SCN - suprachiasmatic nucleus

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