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NEUROENDOCRINE DISRUPTION: THE EMERGING CONCEPT

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On July 10, 2010, the beautiful City of Rouen (France) hosted what we believe was the First International Symposium on the "Neuroendocrine Effects of Endocrine Disruptors (NEED)." Several laboratories had been working independently on the possibility that endocrine-disrupting chemicals were having major effects on the central nervous system. Luckily, an opportunity to assemble this group came about when Dr. Hubert Vaudry, the main organizer of the 7th International Congress of Neuroendocrinology (ICN) 2010, sent out a request for proposals for satellite symposia to be associated with the main event. One of us (O.K.) proposed the idea for this symposium, and it was readily endorsed by the ICN. Moreover, the idea to publish a special volume came soon after. This idea was also very well supported by Dr. Sam Kacew, editor-in-chief of the *Journal of Toxicology and Environmental Health*.

We thank the generous sponsors and volunteers who helped to finance and run this symposium. They are l'Université de Rennes 1, INRA, INERIS, CNRS, la Ville de Rouen, Science Action Haute-Normandie, and the ANR program "NEED." We also appreciated the critical help of many other people, in particular, Philippe Chan Tchi Song (l'Univeristé de Rouen), Monsieur Hébert from la Ville de Rouen, Emmanuelle Guiot, Cyril Gabbero, Arianna Servili, Maria Rita Pérez, and Chiara Piccinetti from the UMR-CNRS 6026 at l'Université de Rennes 1, and Jan Mennigen and Andrew Waye from the University of Ottawa. There are also the dozens of expert reviewers of the articles presented here, who must also be acknowledged with appreciation. The rigor of their reviews and their constructive yet critical comments most certainly helped all the authors.

Unfortunately, a number of excellent researchers were not able participate in the event. However, despite the cuts in funding experienced by researchers in many countries in this period in history, and with the strong support already noted, we still managed to bring together around 85 scientists from about 20 different countries. They were the substrate and catalyst for this dynamic, interactive symposium in the original sense of the word.* We had a wonderful day of science and discussion in a friendly atmosphere. The entire group enjoyed the fine food and wines of the host country, and new friendships and collaborations have most certainly resulted from this meeting.

Historically, the concept of endocrine disruptors emerged in the late 1980s and early 1990s from field studies pointing out a series of problems related to reproduction in wild populations. Major problems recorded decades earlier, such as disruptions in human development caused by diethylstilbestrol (DES), and reproductive problems in birds around

*See http://oxforddictionaries.com/view/entry/m_en_gb0837900#m_en_gb0837900 for the definitions of "symposium."

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the Great Lakes cause by dichlorodiphenyltrichloroethane (DDT) preceded formal recognition of endocrine disruption as the cause. Even at this early stage, hormone upsets were the suspected cause of these developmental effects. The concept then moved from the field to the laboratory and many teams worldwide started to decipher the mechanisms underlying potential effects of endocrine-disrupting chemicals (EDCs). Early studies mainly focused on peripheral organs, notably the gonads and liver. For many years, there was a deficit regarding the effects of EDCs on the neuroendocrine circuitry controlling peripheral hormonal functions and behaviors. Neuroendocrine disruption extends the concept of endocrine disruption to include the full breadth of integrative physiology; that is, neuroendocrine disruption is more than upsets in a few hormones (see Figure 1). It is possible that pollutants disrupt numerous neurochemical pathways, both temporarily or permanently altering diverse physiological and behavioral processes to affect an animal's capacity to reproduce, grow, or deal with stress and other challenges. This can happen in both invertebrates and vertebrates. It appears from discussions at meeting, as illustrated in the figure, that a parallelism exists between the developmental disorders of the reproductive system and disturbances of energy balance and central nervous system function. Also, endocrine disruption is mirrored by other insults such as hypoxia, stress, and poor nutrition. All of these factors impinge upon common mediators in the hypothalamus, providing the mechanistic basis of neuroendocrine disruption.

Stem cells, neurons, and glia may be targets of EDCs. It is clear from several in vitro and in vivo model systems that disruption of developmental processes can affect neurogenesis and differentiation of key circuits. This can lead to structural, major behavioral, and thus permanent alternations in the central nervous system. Epigenetics and the concept of reprogramming nervous system development must also now be considered as part of neuroendocrine disruption. Moreover, there is also evidence that some pollutants can affect long-term

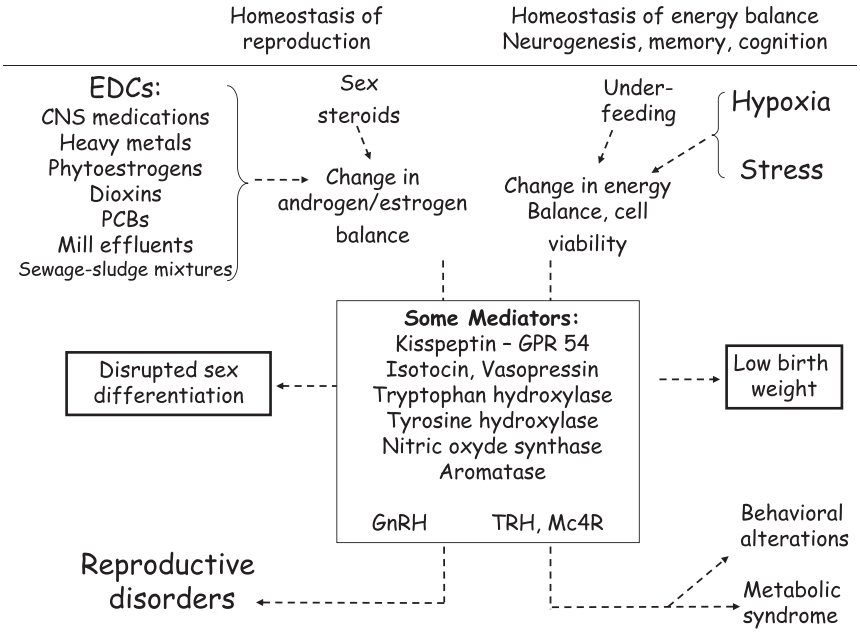

FIGURE 1. Summary of the main concepts and topics covered in this special volume on neuroendocrine disruption. Please refer to the main text and the individual articles for details. CNS, central nervous system; EDCs, endocrine-disrupting chemicals; GnRH, gonadotropin-releasing hormone; Mc4R, melanocortin receptor 4; PCBs, polychlorinated biphenyls; TRH, thyrotropin-releasing hormone.

potentiation, learning, and memory, or induce neurodegeneration. Also emerging from these studies is the idea that both industrial chemicals and pollutants from natural sources can interact with other environmental stressors, thereby disrupting multiple physiological systems. Effects of chemicals can be direct on enzymes involved in classical and gaseous neurotransmitter synthesis and metabolism. The production of neuropeptides is also affected by a diverse array of EDCs. In additional to effects on reproductive processes, EDCs can have effects on neuroendocrine feedback loops of all types, and on mechanisms of cross-talk pathways between neuroendocrine axes. There is also the obesogen hypothesis—that endocrine disruption at both central and peripheral sites may cause metabolic upsets, thereby contributing to etiology of obesity. The concepts of developmental sensitivity and critical periods of exposure along with major differences in species sensitivities to EDCs are also covered in the articles presented here.

Many of the effects of EDCs seen in teleost, amphibian, avian, and mammalian model systems may also occur in humans. By developing this integrative symposium covering the array of organisms and systems affected by neuroendocrine disruptors, it is our hope that these data will help scientists move from the laboratory to translational research at the clinical and ecosystem levels. Only in this way will the complete picture of the consequences of EDC exposure emerge.