Maternal Anticancer Drugs and Fetal Neuroendocrine Dysfunction in Experimental Animals

Ahmed RG*
Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt

*Corresponding author: Ahmed RG, Division of Anatomy and Embryology, Zoology Department, Faculty of Science, Beni-Suef University, Beni-Suef, Egypt
Tel: 002-010-91471828; E-mail: ahmedragab08@gmail.com

Received date: November 16, 2017; Accepted date: December 12, 2017; Published date: December 28, 2017

Copyright: © 2017 Ahmed RG. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Anti-Cancer Drugs

Increase demand of gestational thyroid hormones (THs) is necessary for the fetal development [1-40]. Doxorubicin (DOX) is an anticancer drug that is used widely in treating leukemias, lymphomas, lung, breast, ovary and uterine cancers [41]. However, its injection during pregnancy initiated thyroid dysgenesis and disorganization [42,43], hypothalamic-pituitary-thyroid axis (HPTA) dysfunction [43,44], GH-resistance [45], growth retardation [46,47], and CNS damage in children or adults [48]. Furthermore, thyroid disorders in rats are commonly disturbed the inorganic phosphate (Pi) and Ca\(^{2+}\) enzymes system (Na+, K+-ATPase, Ca\(^{2+}\)-ATPase). The neuronal-oligodendrocyte functions [57]. Rats are commonly disturbed the inorganic phosphate (Pi) and Ca\(^{2+}\) homeostasis [49]. The impairment in the Ca\(^{2+}\)-ATPase function was observed in the hypothyroid rat brain [50,51].

Also, the neonatal hypothyroidism can cause the following: (1) impair the synaptic transmission [52,53]; (2) disturb the Na+, K+-ATPase activity [3,6]; (3) alter the kinetic properties of Na+, K+-ATPase [54,55]; (4) diminish the metabolic and electrical activities [52,53,56]; and (5) delay the neuronal-oligodendrocyte functions [57]. In general, the deficiency in the rate of ATP synthesis [50,58,59] and in the rate of AMP-activated protein kinase (AMPK) [60] was decreased significantly in the hypothyroid state. This reduction can inhibit fatty acid oxidation [60] and impair the energy production [61]. On the other hand, my group reported that the activities of fetal ATPase-enzymes system (Na+, K+-ATPase, Ca\(^{2+}\)-ATPase and Mg\(^{2+}\)-ATPase) were decreased in cerebrum and cerebellum of maternal DOX group [33]. In parallel, DOX decreased ATP [62] and ATP/ADP ratio [62,63], inhibited Na+/K+ pump [64] and sarcoplasmic reticulum Ca2+-ATPase [62], and increased apoptotic pathways [65]. Thus, DOX may act as endocrine- and neural-disrupting actions on the development of THs-brain axis. Thus, any insignificant alterations in the thyroid function during the development can cause brain damage [2,6,66]. Also, clinical studies displayed that maternal TH deficiency during the first trimester of pregnancy can affect the outcome of human neurodevelopment [67,68]. For these reasons, I recommend annual estimation for THs and growth rates for DOX-mothers and their neonates [33]. Obviously, investigations in this field are still in its beginnings, with numerous significant and challenging issues remaining to be addressed.

Conflict of Interest

The author declares that no competing financial interests exist.

References

20. Ahmed RG (2017c) Anti-thyroid drugs may be at higher risk for perinatal thyroid disease. EC Pharmacology and Toxicology 4:4: 140-142.


Ahmed RG (2017g) Antiepileptic drugs and developmental neuroendocrine dysfunction: Every why has A Therefore. Arch Med 9: 2.

Ahmed RG (2017h) Gestational prooxidant-antioxidant imbalance may be at higher risk for postpartum thyroid disease. Endocrinol Metab Syndr 6: 279.

Ahmed RG (2017i) Endocrine disruptors; possible mechanisms for inducing developmental disorders. Intern J of basic science in medic.


Citation: Ahmed RG (2017) Maternal Anticancer Drugs and Fetal Neuroendocrine Dysfunction in Experimental Animals. Endocrinol Metab Syndr 6: 281. doi:10.4172/2161-1017.1000281

Endocrinol Metab Syndr, an open access journal
ISSN:2161-1017
Volume 6 • Issue 6 • 1000281
