Prolonged exposure to acetaminophen during pregnancy reduces testosterone production by the human fetal testis

Mehmet Gokhan Culha¹, Ege Can Serefoglu²

¹Department of Urology, Istanbul Training & Research Hospital, Turkey; ²Department of Urology, Bagcilar Training & Research Hospital, Turkey

Correspondence to: Ege Can Serefoglu, MD, FECSM. Department of Urology, Bagcilar Training & Research Hospital, Turkey.
Email: egecanserefoglu@hotmail.com.

Provenance: This is a Guest Editorial commissioned by Section Editor Hongcheng Zhu, MD, PhD (Department of Radiation Oncology, The First Affiliated Hospital of Nanjing Medical University, Nanjing, China).


Submitted Nov 02, 2016. Accepted for publication Nov 08, 2016.
doi: 10.21037/atm.2017.01.57
View this article at: http://dx.doi.org/10.21037/atm.2017.01.57

Congenital anomalies are a major cause of infant morbidity and mortality, and a significant proportion of these anomalies involve urogenital malformations (1). As fetal testosterone has crucial importance in the development of the male reproductive system, any disruption in its production can result in disorders such as hypospadias, cryptorchidism or testicular cancer (2).

The risk of congenital malformation, including urogenital abnormalities, is increased by the administration of medical treatments during pregnancy (3). Weak analgesics, particularly acetaminophen containing over-the-counter drugs, are widely used among pregnant women in Europe and North America (4). However, recent studies have demonstrated that these analgesics may impair fetal testicular hormone production (5,6) and increase the risk of cryptorchidism in the offspring (7). Moreover, some have reported that this risk is even further promoted by the combination of acetaminophen-containing medications with other analgesics (5).

In an elegant study, van den Driessche et al. (8) demonstrated that protracted exposure of xenografted human testes to a therapeutic dose of acetaminophen decreased the plasma levels of testosterone, as well as decreased the weight of seminal vesicles in castrated host mice. After recording a significant reduction in both Cyp11a1 and Cyp17a1 mRNA expression after the final dose of acetaminophen administration via reverse transcription polymerase chain reaction, the authors concluded that the acetaminophen-induced testosterone reduction may result from the reduced quantities of these key steroidogenic enzymes (8).

With these important findings, the study of van den Driessche et al. (8) sheds light to the unknown aspects of the impact of endocrine disrupting compounds on fetal testosterone production. The detrimental effect of widely used acetaminophen containing analgesics on testosterone production must be taken into consideration by policy makers in order to heighten public awareness regarding the potential teratogenic effects of these over-the-counter analgesics. Meanwhile, researchers should corroborate the findings of this study in order to develop further preventive measures, if necessary.

Acknowledgements

None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

References


Cite this article as: Culha MG, Serefoglu EC. Prolonged exposure to acetaminophen during pregnancy reduces testosterone production by the human fetal testis. Ann Transl Med 2017;5(10):218 doi: 10.21037/atm.2017.01.57