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**Original article**

## **Effect of maternal alcohol consumption on the growth rates of accessory sex organs in neonatal mice**

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### ABSTRACT

The effect of maternal alcohol consumption on the growth rate of accessory sex organs (seminal vesicles, prostate and bulbourethral glands ) in neonatal mice was investigated using 120 male mice offspring obtained from 36 female mice divided into three groups, 1, 2 and 3 of 12 mice each. The pups of group 1 served as control while those of groups 2 and 3 were given 30% ethanol (v/v) during pregnancy (P) and during pregnancy and lactation (PL) respectively. At 3, 4, 5 and 6 weeks of age, 10 male pups were randomly selected from each of the three groups and sacrificed. After sacrifice, the accessory sex organs were dissected out and their weights determined. Thereafter, the relative growth rates of the organs were determined. The results showed significant increase ( $p < 0.05$ ) in the growth rates of accessory sex organs in P and PL when compared with the control. The investigation has therefore demonstrated that maternal alcohol intake during pregnancy and during pregnancy and lactation affects the growth rate of accessory sex organs in the neonates and that the organs attempted catch-up growth with the control.

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## **1. Introduction**

It has been established that alcohol has a potential teratogenic effects (Schwartz and Carey, 2005) and Assadi (2007) described a pattern of congenital abnormalities in children born to women with severe alcoholism. These abnormalities include retardation of weight and length of such infants at birth and noted lack of catch-up growth in them. Human and non-human primate studies on brain structure and function strongly suggest that consumption of alcohol during pregnancy can affect fetal brain structure and function (Guerri, 1998). As a result of this effect, the hypothalamic-pituitary-gonadal axis regulation which plays a critical role in control of reproduction (Grober et al, 1998) could be disrupted. The hormone from this axis plays a critical role in stimulating the growth and activity of the reproductive system (Young and Heath, 2004).

The effects of maternal alcohol intake on the growth and development of some parts of the body in both human and experimental animals have been reported (Li and Kim, 2003; Onu et al, 2011). In the reproductive system, reduced growth has been reported on the testes (Fakoya and Caxton-Martins, 2004) penis (Onu et al, 2004) and epididymis (Onu et al, 2010). The possible effect of alcohol intake during pregnancy and during pregnancy and lactation on the growth rate of accessory sex glands have not been elucidated, hence this investigation.

## **2. Materials and methods**

The experimental procedures employed in this study were similar to those of Lee and Leichter (1980). Thirty six (36) virgin female and 9 immature male mice used in this study were randomly selected at the weaning age of 21 days from a colony of locally in-bred mice maintained for research in the animal house of the Faculty of Veterinary Medicine, University of Nigeria, Nsukka. The virgin female mice were divided into three groups of 12 each. The offspring of group 1 served as control (C), while those of group 2 and 3 were exposed to alcohol during pregnancy only (P) and during pregnancy and lactation (PL) respectively. The mice were housed separately in cages with screened tops and were acclimatized for three weeks before the beginning of the study. Water and commercial feed (Bendel Feed and Flour Mills, Nigeria Plc.) were allowed ad libitum throughout the period of the investigation. At the beginning of the 7th week of age, the female mice in group 2 and 3 were given 10% ethanol (v/v) in drinking water for 2 weeks and 20% ethanol (v/v) for another 1 week. Thereafter, the female mice in the three groups were then bred overnight by introducing 1 male mouse into a cage housing 4 female mice. Day 1 of pregnancy was presumed after observation of vaginal plug the following morning. Following diagnosis of pregnancy, the female mice in P and PL were given 30% ethanol (v/v) till delivery. After delivery, the alcohol for P was replaced with drinking water while PL continued to receive the 30% ethanol (v/v) in drinking water until their pups were weaned at 21 days of age. At the age of 3, 4, 5 and 6 weeks, 10 male pups were randomly selected from control, P and PL and sacrificed. A total of 120 pups were used in the investigation.

### **2.1. Quantitative measurements**

After the sacrifice, the accessory sex organs were dissected out, trimmed of extraneous tissue and weighed using Metler's Analytical Balance (BFN-390). After this, the relative growth rates of the organ were calculated thus, the mean of logarithms of the weights at week 3 was calculated and then used to divide the logarithms of their respective weights at week 4, 5 and 6 (Nwaogo and Ihemelandu, 1999).

### **2.2. Statistical analysis**

Means and standard errors were calculated for each group. One-way analysis of variance (ANOVA) was used to examine whether there were significant differences in the parameters measured amongst the three groups. Following the observation that there were significant differences amongst the three groups, Duncan's Multiple Range Test (DMRT) (Duncan, 1955) was used to determine which groups differed. For the statistical test,  $p < 0.05$  and  $p < 0.01$  were considered statistically significant.

## **3. Results**

### 3.1. Growth rates of accessory sex organs

#### 3.1.1. Seminal vesicles

The mice offspring in P grew faster ( $p < 0.01$ ) than the control throughout the period of the investigation. At week 4, the growth rate of the control and the mice offspring in PL were similar ( $P > 0.05$ ). At week 5, the control significantly grew faster ( $p < 0.01$ ) when compared with PL, while at week 6, the latter significantly grew faster ( $p < 0.01$ ) when compared with the former (TABLE I).

#### 3.1.2. Bulbourethral gland

The mice offspring in P grew significantly faster ( $p < 0.01$ ) than the control throughout the duration of the investigation. Control and PL were similar ( $p > 0.05$ ) at week 4 while the latter significantly grew faster ( $p < 0.01$ ) at week 5 and 6 when compared with the former (TABLE II).

#### 3.1.3. Prostate gland

The growth rate of the control and P were similar ( $p > 0.05$ ) while PL significantly grew faster ( $p < 0.01$ ) when compared with the control throughout the period of the investigation (TABLE III).

## 4. Discussion

This study has demonstrated that maternal alcohol intake during pregnancy and during pregnancy and lactation reduced the growth rate of the accessory sex organs in mice. This is evident when growth rates were used as growth indices. The result indicated that the two alcohol-exposed groups had greater growth rates when compared with the control.

**Table 1**

Growth rate of seminal vesicle of mice exposed to alcohol during pregnancy (P) and during pregnancy and lactation (PL).

Age (weeks) at sacrifice	Control (n = 10)	P (n = 10)	PL (n = 10)
4	1.44±.10a	1.83±0.04b	1.60±0.04a
5	2.02±0.10a	2.75±0.10b	1.51±0.10b
6	2.70±0.04a	3.54±0.01b	3.67±0.10b

Values represent means±SEM for each measurement

Means with different superscript on the same row are significantly different at  $p < 0.05$

P, Male mice exposed to alcohol during pregnancy only

PL, Male mice exposed to alcohol during pregnancy and lactation

**Table2**

Growth rate of bulbourethral gland of mice exposed to alcohol during pregnancy (P) and during pregnancy and lactation (PL).

Age (weeks) at sacrifice	Control (n = 10)	P (n = 10)	PL (n = 10)
4	1.56±0.01a	2.03±0.05b	1.79±0.10a
5	2.19±0.04a	2.70±0.30b	3.06±0.10b
6	2.74±0.03a	3.16±0.10b	3.30±0.04b

Values represent means±SEM for each measurement

Means with different superscript on the same row are significantly different at  $p < 0.05$

P, Male mice exposed to alcohol during pregnancy only

PL, Male mice exposed to alcohol during pregnancy and lactation

**Table 3**

Growth rate of prostate gland of mice exposed to alcohol during pregnancy (P) and during pregnancy and lactation (PL).

Age (weeks) at sacrifice	Control (n = 10)	P (n =10)	PL (n = 10)
4	1.29±.01a	1.37±0.03a	13.52±0.40b
5	1.84±0.03a	1.93±0.10a	9.04±0.01b
6	2.15±0.02a	1.79±0.10a	17.88±1.13b

Values represent means±SEM for each measurement

Means with different superscript on the same row are significantly different at  $p < 0.05$

P, Male mice exposed to alcohol during pregnancy only

PL, Male mice exposed to alcohol during pregnancy and lactation

However, the growth rates of seminal vesicle and bulbourethral gland showed similarity between control and PL while that of prostate gland also showed similarity between the control and P at week 4. The similarity of growth rates at week 4 between the control and PL in seminal vesicle and bulbourethral gland and between the control and P in prostate gland was an indication of an attempt by those exposed to alcohol to catch-up growth with the control. This observation indicated that feeding of alcohol during pregnancy and lactation following prenatal exposure did not suppress completely the attempt of the accessory sex organs to catch-up growth with their control counterpart. This observation is similar to that of Onu et al, (2004) on penile growth.

The mechanism by which alcohol intake during pregnancy and during pregnancy and lactation reduced the growth rate of accessory sex organs was not determined in this study. However, Murillo-Fuentes et al, (2001) observed that alcohol consumed during pregnancy adversely affects the regulatory mechanism of growth of the pups and the effect persists after birth. It is well established that severe structural damage occurs to the brains of infant exposed to alcohol during pregnancy (Guerri, 1998). Zhang et al, (2005) also observed that alcohol impairs fetal hormonal system.. Since the endocrine mechanism in the fetuses is similar to that of the adults (Mennella et al., 2005), the alcohol consumed during pregnancy and during pregnancy and lactation could have disrupted the neuroendocrine functions of the fetuses by its action on hypothalamus sequel to destruction of brain structure and function (Guerri, 1998). This action on the hypothalamus could lead to disruption of the hypothalamic-pituitary-gonadal axis as suggested by Onu and Ezeasor, (2001) which plays significant role in reproduction (Grober et al, 1998). The hormone from this axis plays a critical role in the growth and activity of the reproductive system (Young and Heath, 2004) of which the accessory sex organs are part.

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