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Lactational Exposure To An Aqueous Extract Of *Hibiscus Sabdariffa* (Hs) Accelerates Offspring's Early Postnatal Growth In Sprague-Dawley Rats

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ABSTRACT

This study was designed to evaluate whether or not lactational exposure to HS affects offspring's postnatal growth.

Three groups (five rats per group) of pregnant Sprague-Dawley (SD) rats were used for this study. Group C had tap water while groups A and B had 0.6g and 1.8g HS extract respectively in 100ml tap water to drink throughout pregnancy. All groups had normal rat chow ad libitum. On the day of birth, birth weights were recorded and four pups from each dam in group C were substituted for 4 pups (two pups each) from dams in groups A and B. Thereafter, weights were recorded at 10days, 14days, 20days and 34 days postpartum.

Results of the present study show a statistically significant growth increase ($p < 0.05$) in group A pups at all periods of measurement compared with groups B and C pups while group B pups showed decreased growth at 10 days, comparable growth at 14 days and increased growth ($p < 0.05$) at 20 and 34 days postpartum compared with group C pups.

From the present study, we conclude that lactational exposure to an aqueous extract of HS accelerates offspring's early postnatal growth through a mechanism not yet known.

Key words: lactational exposure, Hibiscus Sabdariffa, Postnatal growth

Extracts of Hibiscus Sabdariffa (HS) have been used in folk medicine in the treatment of several complaints including high blood pressure, liver diseases and fever (Ali et al, 2005; Tseng et al, 1997; Usoh et al, 2005).

Zobo drink (a sweetened water extract of the dry petals of HS) is commonly produced, sold and consumed in Nigeria without caution by both males and females. It is consumed as a substitute for carbonated drinks and fruit juices and not necessarily for medicinal reasons. Some people have been observed consuming zobo drink during lactation.

Studies in both humans and experimental animals addressing the 'developmental origins of adult health and disease' hypothesis have established a relationship between environmental perturbations during periods of developmental plasticity in postnatal life and offspring's disease in adult life (Armitage et al, 2004; Armitage et al, 2005a; Armitage et al, 2005b). This relationship has been demonstrated to be due to permanent changes in organ structure and metabolism and/or alterations in homeostatic regulatory mechanisms in the offsprings. This may be the aetiopathogenetic basis of some adult diseases such as cardiovascular diseases, obesity and diabetes mellitus (Armitage et al, 2004; Ross and Desai, 2005).

We have observed in our earlier study that in utero exposure to HS severely attenuated early postnatal growth (unpublished). This study was designed to evaluate whether or not lactational exposure to HS affects offspring's postnatal growth.

MATERIALS AND METHODS

The procedure used in our laboratory for the extraction of HS was as follows: 30g of the dry petals of HS was brewed in 400ml of boiled tap water for 45min. The resulting decoction was filtered using a filtration sieve. 10ml of the filtrate was evaporated to dryness and yielded 0.36650.002g, giving a concentration of

0.036650.002g/ml.

The concentrations in the exposed groups (groups A and B) below were derived as follows: 10mls of filtrate was added to 48mls of tap water to make approximately 0.6g/100ml tap water (group A) while 10mls of filtrate was added to 9mls of tap water to make approximately 1.8g/100ml tap water (group B).

Three groups (five rats per group) of pregnant Sprague-Dawley (SD) rats were used for this study. Group C had tap water while groups A and B had 0.6g and 1.8g HS extract respectively in 100ml tap water to drink throughout pregnancy. All groups had normal rat chow ad libitum. On the day of birth, birth weights were recorded and four pups from each dam in group C were substituted for 4 pups (two pups each) from dams in groups A and B. Thereafter, weights were recorded at 10days, 14days, 20days and 34 days postpartum.

Statistical Analysis

Results are expressed as mean s.e.m. (standard error of mean). Statistical difference was calculated by the students' t-test with 0.05 taken as the level significance.

RESULTS

Table 1. Postnatal Weights (g)

Groups	Postnatal weights (g)				
	At birth	At 10 days	At 14 days	At 20 days	At 34 days
A	5.0±0.1	15.9±0.4*$	20.2±0.3*$	28.0±0.4*	71.5±1.4*$
B	5.3±0.3	13.3±0.3*	19.2±0.3*	28.7±0.8*	64.6±2.3*
C	6.1±0.1	17.0±0.2	21.9±0.2	30.4±0.4	50.4±0.9

*=p<0.05 vs control (C)
$ =p<0.05 vs B

Table 2. Postnatal Weight Gain

Groups	Postnatal weight gain			
	At 10 days	At 14 days	At 20 days	At 34 days
A	10.9±0.4$	15.2±0.3*$	23.0±0.4*	66.5±1.4*$
B	8.0±0.5*	13.9±0.2*	23.4±1.0	59.3±2.3*
C	10.9±0.2	15.8±0.2	24.3±0.4	44.3±0.9

*=p<0.05 vs control (C)
$ =p<0.05 vs B

Results of the present study show a statistically significant reduction in pup weights at 10, 14, and 20days postpartum with group B pups having a more significant weight reduction when compared with group A pups (table 1 and 2).

However, a comparison of the percent postnatal weight gain (defined as weight gain/birth weight x 100) shows a statistically significant weight increase in group A pups at all periods of measurement compared with group B pups and control pups (table 3) while group B pups showed decreased weight gain at 10days postpartum, comparable weight gain at 14days postpartum and increased weight gain at 20 and 34days postpartum compared with control pups.

Table 3. % Postnatal Weight Gain

Groups	% Postnatal weight gain			
	At 10 days	At 14 days	At 20 days	At 34 days
A	219.9±10.2*$	305.1±9.0*$	462.7±16.8*	1339.8±61.9*$
B	154.1±16.6*	265.0±14.7	449.1±40.1*	1129.1±65.7*
C	180.6±5.9	261.2±6.5	400.9±9.8	728.8±17.9

*=p<0.05 vs control (C)
$ =p<0.05 vs B

Results also show a more significant weight gain in the first 10 and 14 days postpartum relative to the second 10days and last 14 days respectively (table 4 and 5). At the second 10days postpartum, there was no significant difference in percent weight gain between groups A and C pups whereas group B pup gained significantly more weight compared with both group A and C pups signifying tremendous growth at this period (table 4).

In the last 14days of measurement, groups A and B pups exhibited more percent weight gain compared with control pups (table 5) with group A pups exhibiting more weight gain relative to group B pups.

Table 4. Postnatal Weight Gain In The 1st And 2nd 10 Days Postpartum

Groups	Postnatal weight gain				
	Birth weight (g)	1st 10 days (birth-10 days)		2nd 10 days (11 days-20 days)	
		Weight gain (g)	% weight gain	Weight gain (g)	% weight gain
A	5.0±0.1	10.9±0.4$	219.9±10.2*$#	11.5±0.7*$	71.8±6.0$
B	5.3±0.3	8.0±0.5*	154.1±16.6*#	15.4±0.7*	115.8±4.9*
C	6.1±0.1	10.9±0.2	180.6±5.9#	13.4±0.4	78.9±2.6

* = p<0.05 vs control (C)
$ = p<0.05 vs B
\# = p<0.05 vs 2nd 10 days

Table 5. Postnatal Weight Gain In The 1st And Last 14 Days Postpartum

Groups	Postnatal weight gain				
	Birth weight (g)	1st 14 days (birth-14 days)		Last 14 days (21 days-34 days)	
		Weight gain (g)	% weight gain	Weight gain (g)	% weight gain
A	5.0±0.1	15.2±0.3*$	305.1±9.0*$#	43.8±1.1*$	158.5±3.8*$
B	5.3±0.3	13.9±0.2*	265.0±14.7#	35.9±2.3*	125.7±9.5*
C	6.1±0.1	15.8±0.2	261.2±6.5#	19.9±0.9	65.9±3.5

* = p<0.05 vs control (C)
$ = p<0.05 vs B
\# = p<0.05 vs last 14 days

DISCUSSION

In the first 10 days of life, group A pups exhibited tremendous growth rate typified by the significantly increased percent weight gain relative to group B and control pups, whereas group B pups exhibited less growth compared with control pups. However, at the second 10days of life, there was no significant difference between the growth of pups in groups A and C but a tremendously increased growth in group B pups relative to group A and C pups. This probably accounted for the similar growth at 14days postpartum in groups B and C pups and similar growth at 20days postpartum in groups A and B pups. These observations suggest that lactational exposure to an aqueous extract of HS accelerates postnatal growth (through a mechanism not yet known) and that at a concentration of 0.6g/100ml water, HS exerts a more growth accelerating effect than at a concentration of 1.8g/100ml water.

This was unexpected because greater growth acceleration was expected for group B pups since they were exposed to more HS. This observation may suggest that the magnitude of the growth acceleration induced by lactational exposure to HS may be concentration dependent to the extent that beyond a certain critical concentration, there may be a point of diminishing return at the expense of growth acceleration. This may probably explain the growth restriction in the first 10days of life in group B pups relative to group C pups.

The reason for the decreased weight gain in the first 10days postpartum in group B pups relative to group C pups is not known. However, at the second 10days postpartum, there was a tremendous growth in group B pups resulting in similar growth patterns in groups A and B pups that is significantly greater than control pups at 20days postpartum. This observation strongly suggests that the reduced growth in group B pups in the first 10days of life may have induced a 'catch-up' growth (through an unknown mechanism) that starts early in the second 10days postpartum in these pups resulting in the

tremendously increased growth at the end of the second 10 days in a manner similar to the observations of Ciafarani and Colleagues (1999), Hales and Ozanne (2003) and Desai and Coworkers (2005). This probably resulted in the similar growth patterns in groups A and B pups that is significantly greater than the control pups at 20days postpartum.

From the foregoing, we conclude that lactational exposure to HS accelerates offspring's early postnatal growth and that the magnitude of this growth acceleration may be concentration dependent to the extent that beyond a certain critical concentration, there may be a point of diminishing return at the expense of growth acceleration.

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