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Research Article

Maternal salt overloading increases placental oxidative stress and impairs foetal survival ability in Wistar rats

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ABSTRACT

Background: Maternal nutritional during pregnancy is essential in foetal growth and development. **Objective:** This study is aimed at investigating the effects of high and low salt diet on foetal survival at different stages in pregnancy.

Methods: Seventy-five female rats (150-200g) with regular oestrus cycle (n=25) were mated at ratio 2:1 female to male on proestrus. Rats confirmed pregnant were fed a standard diet of 0.3% NaCl, low salt diet (LSD), 0.15% NaCl or high salt diet (HSD) of 8% NaCl. On days 6, 8, 12, and 19 of pregnancy, serum and urine electrolytes (Na^+, Cl^-, K^+), serum progesterone and oestrogen were measured using ELISA. Implantation sites, resorption sites, foetal number and weight were recorded. The remaining rats were allowed to deliver; litter number and birth weight were documented. Malondialdehyde (MDA) and antioxidant enzymes: catalase (CAT), glutathione reductase (GSH), and superoxide dismutase were measured in both serum and placenta homogenate. Histopathological changes in the placenta tissues were also studied.

Results: Serum oestrogen and progesterone were lower ($p<0.05$) in HSD than control. There were fewer ($p<0.05$) implantation sites, foetal number, and more resorption sites in HSD while foetal weight was significantly lower in LSD. In the serum and placenta, homogenate, CAT, GSH, and SOD were lower ($p<0.05$), while MDA was higher ($p<0.05$) in HSD compared to control. Histology of the placenta in LSD and HSD shows a distorted decidua cell, haemolysis of the fibrous tissue and cell necrosis.

Conclusion: Intake of high salt decreases the foetus's survival by increasing oxidative stress and disrupting the morphology of placenta.

INTRODUCTION

The growth of placenta and foetus is susceptible to maternal nutrition status,[1] therefore, most problems related to nutrition form the staple of many issues in pregnancy.[1,2] Basically, pregnancy is a time of intense physiological changes from conception till birth. As such, nutritional requirements increase during pregnancy to maintain maternal metabolism while supporting foetal growth and development.[2,3] Thus, nutrition enhances foetal growth at different stages in pregnancy and prevents the occurrence of diseases later in life.[4]

Micro and macronutrients are essential in promoting foetal growth and intrauterine survival. These nutrients are essential to human health but can also be associated with adverse effects when consumed in excess.[5] Salt is an essential micronutrient in pregnancy that ensures adequate birth weight and optimal functioning of foetal organs.[3] On the other hand, inadequate salt intake restricts blood volume and negatively impact foetal growth.[6] The placenta is physiologically susceptible to oxidative stress, but as it adapts to its new high oxygen-rich environment, a rise in antioxidant activity is also important.[7] Reactive oxygen species (ROS) sometimes harm placental development. Hence, it is important to have a balance between the generation of ROS or nitrogen species (RNS) and their clearance by defensive antioxidants.

Antioxidants such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase/reductase (GSH) are available in the placenta for this defence.[7] These ultimately prevents the foetal growth distortion that varies with each trimester.[8] Over the years, high salt intake has been a common feature of Western dietary patterns, associated with metabolic changes favourable to impaired mitochondrial function that promotes redox state imbalance in the new-born.[9]

Evidently, high salt diet increases oxidative stress,[10] but there is a paucity of data on the oxidative status of the placenta and its morphology following the high and low salt diet. Therefore, this study is aimed at investigating the outcomes of high and low salt diet on placenta oxidative status and its risk on foetal survival ability as pregnancy progresses.

MATERIALS AND METHODS

Experimental Animals and Diet

Seventy-five nulliparous female Wistar rats at 7-8 weeks old with a regular oestrus cycle of 4 to 5 days were obtained from the animal house at Lagos State University, College of Medicine, Ikeja, Lagos, Nigeria. The rats were kept in plastic cages lined with wood shavings. They were acclimatized for two weeks and maintained under a standard condition of a 12 hour light and dark period with an ambient temperature between 28°C to 30°C. Standard laboratory chow from Sabina feeds, Lagos, Nigeria, and water were provided *ad libitum*. The research protocol was certified by Lagos State University College of Medicine Ethics and Research Committee guidelines.(AREC/2022/056)

Experimental design and Animal grouping

The rats were paired at the ratio of two (2) female to one (1) male on the evening of proestrus. The presence of spermatozoa in the vaginal smear the next day indicated the first day of pregnancy. Only the pregnant rats were selected for the study and were randomly allotted into three groups of 25 rats each as follows; Group 1 (control) was fed a normal salt diet which contained 0.3 % NaCl. Group 2 (salt restriction) was fed a low salt diet (LSD) which contained 0.15% NaCl, and Group 3 (salt overload) was fed high salt diets (HSD) with 8% NaCl.[11] The pregnant rats in each group were further subdivided into five groups (n=5) according to the procedures on pregnancy days 6, 8, 12, 19 and at birth (Table 1).

Table1: Subgroups in pregnant Wistar rats

Pregnancy days/Groups	Control (number of rats)	HSD (number of rats)	LSD (number of rats)
Day 6	5	5	5
Day 8	5	5	5
Day 12	5	5	5
Day 19	5	5	5
At birth	5	5	5

Urine, blood, and tissue sample collection

Urine samples were collected in the rats before pregnancy and on days 6, 8, 12, 19 of gestation. The animals were isolated in plastic metabolic cages for 24 hours, with free access to food and water *ad libitum*. The urine collected was pipette into the measuring cylinder to determine volume in millilitres (ml), and electrolytes were assayed. Blood samples were collected from each rat on days 6, 8, 12, and 19 of gestation through the retro-orbital sinus after anesthetizing with 1 mg/kg of ketamine. The blood samples were allowed to clot and centrifuged at 3000 rpm for 20 minutes. The serum was collected and refrigerated at -20°C.

The placentas were excised on days 12 and 19 of gestation following 1 mg/kg ketamine intraperitoneally and cervical dislocation. Some of the placenta tissue were immediately weighed and fixed in formosaline to process for histology. The remaining tissues were homogenized inphosphate buffer to determine the activities of CAT, GSH, SOD, and MDA.

Determination of Urine and Plasma Sodium, Potassium and Chloride

The plasma and urine collected were assayed for Na^+, Cl^- and K^+ using SFRI ISE 6000 electrolyte analyser.

Determination of oestrogen and progesterone

Serum oestrogen and progesterone were estimated using the enzyme-linked immunosorbent assay (ELISA) method. Oestrogen and progesterone ELISA kit AccuBind Inc. USA were used following the manufacturer's instructions.

Determination of serum and placenta Superoxide Dismutase (SOD), catalase (CAT), reduced glutathione (GSH), and Malondialdehyde (MDA) activities

Superoxide dismutase activities were determined by method based on the ability to inhibit the auto-oxidation of epinephrine as determined by an increase in absorbance at 480nm.[12] Catalase activities were determined according to the method of Aebi,[13] which was based on the fact that dichromate in acetic acid is reduced to chromic acetate when heated in the presence of H_20_2 with the formation of perchromic acid as an unstable intermediate. Activities of GSH were determined by using Ellman's reagent, which is a method based on the development of the relatively stable yellow complex formed due to the reaction between Ellman's reagent and free sulfhydryl groups.[14] Malondialdehyde which is an index of lipid peroxidation, was determined using the method by Buege and Aust.[15]

Counting of Implantation Sites, foetal number, and Number of litters

Rats in subgroups 6 and 8 (gestation) days were injected with 1 mg/mL Evans blue through the tail vein. After 15 minutes of injection, the rats were sacrificed following cervical dislocation. The implantation sites were carefully observed and counted in the uterus of the rats. Rats in sub-groups 12 and 19 (gestation) days were also sacrificed by cervical dislocation. Their conceptus was carefully separated from the uterus and counted. Their placenta were also carefully separated and weighed. The litter numbers were counted in the group of rats left to deliver their pups.

Histopathological Studies

The placenta tissues were embedded in molten paraffin wax and cut into sections of 5 µm thick. The tissue sections were further stained with Haematoxylin and Eosin (H&E), and then Photomicrograph was shot at ×400 under a microscope.

Statistical Analysis

The data collected were analysed using graphed pad prism 5.0 Statistical software. Data were expressed as mean ± Standard error of the mean (SEM) and analysed using one-

way analysis of variance (ANOVA). Student Newman-Keuls was used as post-hoc test to identify differences between individual mean. The confidence interval was placed at 95% so that in all cases, P-value < 0.05 was considered significant.

RESULTS

Effects of high and low salt diets on electrolyte levels in the serum and urine of pregnant Wistar rats

In the serum of high salt diet, sodium level was significantly increased on days 6 and 8 (P<0.05) as well as days 12 (P<0.001) and 19th of pregnancy (P<0.05) when compared with the control diet. Sodium level in the serum of low salt diet shows a significant decrease (Table 2). Also, in the urine (Figure 2), sodium level was significantly high in rats on high salt diet at pregnancy day 6 and 12 (P<0.0001) as well as day 19 (P<0.001). In LSD rats, it was significantly low on day 6 (P<0.05), day 8 (P<0.001) and day 12 (P<0.0001). (Table 2)

Chloride ion in the serum of HSD, showed a significant increase on pregnancy days 6, 8, 12 and 19 (P<0.05). In LSD, Chloride ion was reduced on the 6, 8, 12 (P<0.05) and 19th day of pregnancy (Figure 3). Chloride ion in the urine was also high in HSD on days 6, 8, 12 and 19 (P<0.05) of pregnancy while chloride ion in LSD was reduced on days 6, 8, 12 (P<0.05) and 19 of gestation (P<0.001). (Table 2)

Potassium ion in the serum of HSD was significantly lower in HSD on day 19 of pregnancy (Table 2) while potassium ion in the urine was significantly higher in HSD rats on day 12 of pregnancy (Table 2).

Outcomes of high and low salt diets on oestrogen/ progesterone level in the serum of pregnant Wistar rats

Oestrogen level in the serum was reduced in HSD on days 6, 8, 12 and 19 of pregnancy (P<0.05) (Figure 1). Progesterone in HSD was reduced on day 8 (P<0.05), 12 (P<0.0001) and day 19 (P<0.01) of pregnancy (Figure 2) when compared with rats. In LSD, progesterone was reduced on day 12 (P<0.05) and 19 (P<0.01) of pregnancy (Figure 2) when compared with control.

Maternal high and low salt diets on antioxidant enzymes and lipid peroxidation level in the serum and placenta of Wistar rats

In the serum, there was no significant difference in the CAT across the groups when compared with control. GSH was significantly low in LSD on day 12 and also low in HSD on days 12 and 19. On days 12 and 19 of gestation, SOD was reduced (P<0.005) in LSD and HSD when compared with the control. In HSD rats, MDA was higher on day 12 (P<0.001) and 19 (P<0.05) when compared with the control (Table 3).

In the placenta tissues, CAT level was reduced in LSD (P<0.05) and HSD (P<0.005) on day 12 while it was also reduced in HSD on day 19th (P<0.01) of pregnancy. The level of GSH in HSD was reduced on day 12 and 19 (P<0.005) while MDA was high on day 12 and 19th (P<0.05) of pregnancy than the control (Table 3).

Outcomes of maternal high and low salt diet on foetal, placenta and litter weight in Wistar rats

Foetal weight on day 19 was significantly decreased in LSD and increased in HSD than in control. Placenta weight was decreased in LSD on day 19 (P<0.05) and increased in HSD. At birth, the litter weight was significantly decreased in Low salt diet rats when compared with both control and High salt diet. (Table 4)

Outcomes of Maternal high and low salt diet on implantation and resorption sites, foetal numbers, Litter numbers and length of gestation

Implantation was reduced in HSD on day 8 (P<0.05) while resorption site was also reduced in HSD on days 12 and 19 of pregnancy when compared with control (Table 5)

Foetal number was significantly reduced in HSD on day 12 and 19 (P<0.05). At birth, litter number was decreased in HSD (P<0.05). (Table 5)

Table 2. Effect of salt diet on electrolyte levels in the serum and urine of pregnant Wistar rats

Sodium ion level (mmol/L)				**Chloride ion level (mmol/L)**			**Potassium ion level (mmol/L)**		
Serum				**Serum**			**Serum**		
Gestation	**Control**	**LSD**	**HSD**	**Control**	**LSD**	**HSD**	**Control**	**LSD**	**HSD**
Day 6	125.6±1.9	112.3±2.4**	139.0±2.9*	92.0±3.4	70.9±2.9*	116.0±4.8*	6.4±0.6	6.4±0.7	6.1±0.5
Day 8	129.0±3.2	114.1±3.1*	140.1±2.4*	90.9±2.8	75.7±3.5*	120.9±1.6*	6.2±0.4	7.4±0.6	5.5±0.4
Day 12	128.1±2.7	117.8±3.7*	146.8±2.3**	95.0±5.6	75.2±3.9*	17.4±3.4*	6.7±0.6	7.7±0.5	5.7±0.2
Day 19	125.0±2.2	112.4±2.3*	146.2±5.4*	93.4±3.3	71.2±3.8**	118.1±4.4*	6.4±0.6	7.3±0.5	4.5±0.3*
Urine				**Urine**				**Urine**	
Gestation	**Control**	**LSD**	**HSD**	**Control**	**LSD**	**HSD**	**Control**	**LSD**	**HSD**
Day 6	49.5±2.7	31.0±2.1*	59.3±3.1***	40.1±3.0	30.4±2.4*	41.5±2.8*	42.1±2.1	44.4±3.9	43.7±3.2
Day 8	51.3±5.1	32.4±2.8**	60.8±2.5	38.5±4.1	30.1±1.7*	40.9±2.2*	44.0±8.4	44.1±4.6	39.4±1.4
Day 12	50.1±2.4	34.5±1.4**	63.6±2.3***	40.6±3.7	28.6±3.4*	37.8±2.3*	39.2±2.2	47.7±2.5*	37.6±2.0
Day 19	48.5±3.4	32.1±3.1 5	5.6±4.1**	38.2±3.0	28.4±2.6**	38.3±2.8*	40.2±2.7	52.4±3.6	41.4±2.9

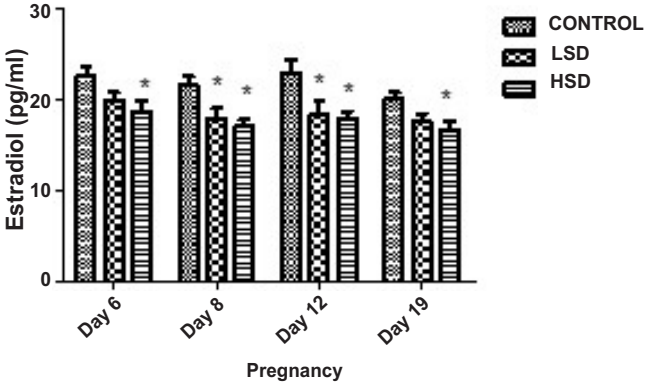

Figure 1. Oestrogen level in the serum of Control, High and Low salt diet in pregnant Wistar rats*P<0.05 when compared with control, n=5

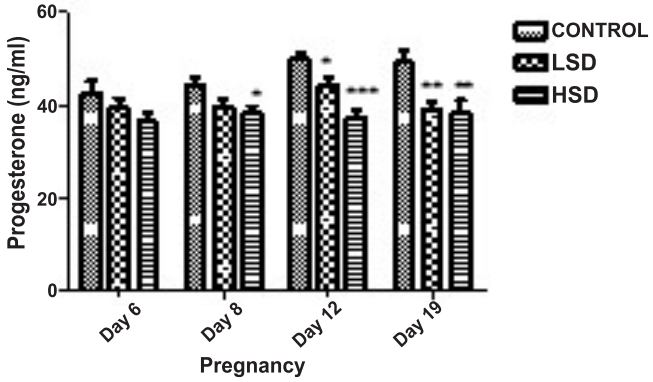

Figure 2. Progesterone level in the serum of Control, High and Low salt diet in pregnant Wistar rats *P<0.05, **P<0.001, ***P<0.0001 when compared with control, n=5

Table 3: Control, High and Low salt diet on antioxidant enzymes and lipid peroxidation level in the serum and placenta of Wistar rats

SERUM								
	CAT(mg/ml)		**GSH (µmol/ml)**		**SOD (mg/ml)**		**MDA (nmol/ml)**	
Group	**Day 12**	**Day 19**	**Day 12**	**Day 19**	**Day 12**	**Day 19**	**Day 12**	**Day 19**
Control	6.78±0.89	6.61±0.72	6.00±0.61	5.36±0.47	8.15±0.87	6.64±0.93	0.02±0.003	0.03±0.01
LSD	4.40±0.77	4.51±0.60	3.86±0.59*	4.09±0.54	5.14±0.93*	4.35±0.67*	0.05±0.01	0.05±0.03
HSD	3.52±0.83	3.64±0.69	3.44±0.66*	3.24±0.51*	4.47±0.68*	2.35±0.33**	0.07±0.12**	0.103±0.03*
PLACENTA TISSUE								
	CAT(mg/ml)		**GSH (µmol/ml)**		**SOD (mg/ml)**		**MDA (nmol/ml)**	
Group	**Day 12**	**Day 19**	**Day 12**	**Day 19**	**Day 12**	**Day 19**	**Day 12**	**Day 19**
Control	7.011±0.38	5.30±0.58	6.32±0.43	4.54±0.41	8.80±0.12	6.72±0.73	0.021±0.004	0.044±0.007
LSD	6.46±1.06*	4.29±0.48	5.19±0.62	4.12±0.51	4.60±0.98	3.64±0.92	0.030±0.006	0.050±0.009
HSD	3.053±0.49**	2.60±0.53**	4.46±0.42*	2.62±0.39**	4.73±0.10	2.92±0.75	0.060±0.006	0.080±0.004*

*P < 0.05 when compared with control, **P<0.001when compared with Control n=5

Table 4: Effects of High and low salt diet on Foetal, Placenta and Litter weights in Wistar rats

Group	**Foetal weight**		**Placental weight**		**Litter weight**
	Day 12	**Day 19**	**Day 12**	**Day 19**	
Control	0.006±0.008	2.66±0.188	0.054±0.007	0.4±0.032	4.317±0.227
LSD	0.038±0.006	2.044±0.219*	0.040±0.008	0.280±0.020*	3.093±0.308*
HSD	0.068±0.0086#	3.040±0.106*#	0.084±0.012*#	0.560±0.051*##	4.967±0.303#

*P < 0.05 when compared with control, #P<0.05 when Compared LSD, ##P<0.001 when compared with LSD, n=5

Table 5: Effects of High and low salt diet on implantation sites, resorption sites, foetal number, Litter number and Length of gestation in Wistar rats

Group	**Implantation site**		**Resorption site**		**Foetal number**		**Litter number at birth**	**Length of Gestation**
	Day 6	**Day 8**	**Day 12**	**Day 19**	**Day 12**	**Day 19**		
Control	13.67±0.49	14.17±0.75	0.60±0.25	0.80±0.37	13.33±0.72	10.33±0.84	9.33±0.49	22.60±0.68
LSD	13.50±0.63	12.83±0.65	1.40±0.25	1.80±0.49	12.67±0.62	9.33±0.715	8.17±0.60	21.80±0.49
HSD	11.83±0.60	10.67±0.96*	2.20±0.37**	2.80±0.21**	9.33±1.02*#	6.50±0.805*	6.83±0.87*	20.80±0.37

*P < 0.05 when compared with control, **P<0.001when compared with Control, #P<0.05 when Compared LSD, n=5

Histopathological findings

The placenta tissue from the control group on day 12 (9A) showed normal decidua cells (DC) of the placenta with the normal cytotrophoblast (CY) and syncytiotrophoblast (SY) along with the chorionic villi (CV). Blood vessels are patent with nucleated RBC alongside narrowed lacuna (L). The placenta tissue from the control group on day 19 (9B) showed normal decidua cell of the placenta, cytotrophoblast (CY) and syncytiotrophoblast (SY) along with the chorionic villi (CV). Decidua cells (DC) are mildly disrupted and congested with blood vessels. The fibrous area is thus remarkable with haemolysis.

The placenta tissue from the LSD group on day 12 (9C) showed the labyrinth zone with the trophoblastic cells. Haemolysis is merged with the trophoblastic (CY and SY) cells with widened lacuna (L). In addition, placenta tissue from the HSD group on day 19 (9D) showed foetal blood vessels with either thin wall or vessels that are totally devoid of vascular wall. Interstitial oedema of the chorionic villi (CV) is also fully seen with necrosis of the decidua cells (DC). The placenta tissue from the HSD group on day 12 (9E) showed reduced decidua cells (DC). Chorionic villi (CV) is also reduced in size but presented with interstitial oedema and congested blood vessels with nucleated RBC. Furthermore, the placenta tissue from the HSD group on day 19 (9F) showed that blood vessels lack walls in many cells. Trophoblastic cells are reduced in size and number hence blood vessels also has RBC that are congested and nucleated.

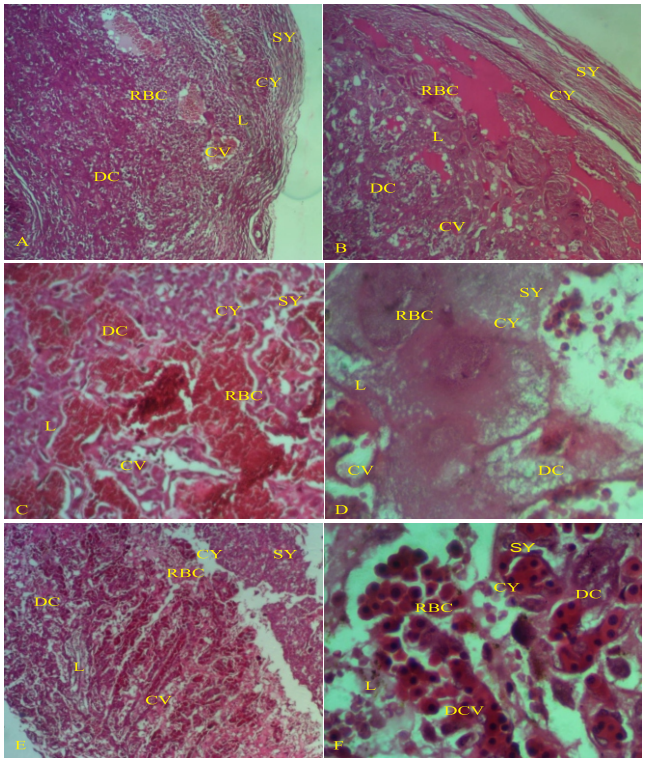

Figure 3A-F: The photomicrograph of the placenta in the Control Wistar rats on day 12 and 19 of pregnancy (A & B), Wistar rats on LSD on day 12 and 19 of pregnancy (C & D), and Wistar rats on HSD on day 12 and 19 of pregnancy (E & F). DC= Decidua cell, CY= Cytotrophoblast, SY= Syncytiotrophoblast, L= Lacuna, CV= chorionic villi, RBC= Red blood cell, H&E, X400.

DISCUSSION

Maternal nutritional status during pregnancy significantly impacts maternal blood composition, which influences placenta and foetal growth.[3] As shown in this study, serum and urine levels of sodium and chloride were increased in HSD and decreased in LSD. Sodium and chloride contributes to plasma osmolarity and create fluid homeostasis suitable for cell survival.[16] The imbalance in the electrolyte shown in this study also caused an excessive urine output which is in line with the finding of Jia et al.[17] High sodium intake stimulates thirst to dilute the extra sodium consumed into an isotonic environment.[18] Therefore, the high sodium reported in this study shows that sodium toxicity in the extracellular fluid also depends on exposure time, irrespective of the magnitude of salt loading.[19]

The pregnancy hormones observed in this study were oestrogen and progesterone, which are the main pregnancy steroid hormones,[20] that helps maintain a normal physiological environment during pregnancy.[21] This study recorded a decrease in serum oestrogen and progesterone in HSD. A decrease in the level of oestrogen and progesterone leads to complications in pregnancy.[22] which affects foetal survival. Physiologically, both oestrogen and progesterone level increases as pregnancy progress.[23] These hormones promote foetal development by maintaining the endometrium during implantation, preventing miscarriage and other undesirable complications.[24]

This present study also showed a decrease in implantation sites and increased resorption sites in rats on excess salt. Although, Drews et al.,[25] earlier reported that embryo resorption in rodent is triggered by endogenous embryonic apoptosis. Feng *et al*.,[26] also emphasized the contributions of poor maternal nutrition in placenta growth and foetal survival. However, factors that interfere with the secretion of hormones in pregnancy decrease implantation sites and increase foetal loss.[26] Furthermore, Progesterone is essential for the induction of uterine decidualization, which occurs during the peri-implantation period. On the other hand, oestrogen also plays a vital role in foetal development and maintenance of the endometrium.[27]

The placenta grows to provide an ever-larger surface area for materno-foetal exchange during pregnancy. Apart from transporting nutrients, these cells maintain specific ionic composition in the cytosol for normal cell turgor pressure, pH, and cellular metabolism. Electrolytes such as sodium and chloride ions are transferred across the placenta by passive diffusion and sometimes active transportation,[28] to maintain pregnancy homeostasis. The trophoblastic cell in the placenta maintains this relationship by allowing transportation of water and ions to the foetus making maternal and foetal ionic concentrations similar.[28]

Similarly, a decrease in foetal and placenta weight was observed in LSD. An earlier report by Leandro et al.,[28] also indicated an intrauterine growth restriction following a low salt intake. Notably, our study also recorded weight reduction in HSD rats. Usually, the weight of the placenta is linked with its transport and metabolic ability, which successively affects the nutrient exchange between the placenta and foetus.[29] As a result, disruption in uterine-placenta perfusion affects foetal growth and may lead to diseases in adulthood.[6,29] According to Lim *et al*.[30] increased sodium in serum is

standard among preterm infants; however, this is usually due to unfavourable water balance, leading to isotonic dehydration of the extracellular fluid compartment and consequently foetal weight loss. Furthermore, micronutrients such as sodium also play an essential role in DNA synthesis, cell proliferation, and absorption of nutrients.[31] However, there must be balance in the intake of these salts to maintain normal plasma osmolarity.

Elevation of plasma and placenta oxidative stress is another way high salt intake altered foetal survival in this study. Physiologically, the placenta of normal pregnancy experiences a burst in oxidative stress which increases as pregnancy progresses.[32] These are usually returned to baseline upon surge in antioxidant activity by the foeto-placenta unit as the placenta gradually acclimates to the new oxidative surrounding.[33] Oxidative stress (OS) is a state characterized by an imbalance between pro-oxidant molecules, including reactive oxygen or nitrogen species, and antioxidant defences. It has been identified to play a vital role in the pathogenesis of fertility in males and females.[34] In this present study, reduction in antioxidants, CAT, GSH, and SOD and increase in malondialdehyde in the serum and placenta of HSD is evidence of oxidative stress.[35] Malondialdehyde (MDA) is a stable end product of lipid peroxidation and therefore can be used as an indirect measure of the cumulative lipid peroxidation. A physiological level of redox is essential in foetal development. Subsequently overproduction of ROS can result in an impaired intracellular milieu and a disturbed metabolism which endanger cell survival.[34] Therefore, the placenta has a complex system of the antioxidant response, such as superoxide dismutase (SOD) or catalase (CAT) enzymes, glutathione peroxidase (GSH), which usually maintains the action of ROS in balance. Although, specific physiological as well as pathophysiology conditions have been reported to reduce this antioxidants defence system.[36] Our study has also clearly observed the incessant reduction in antioxidants in both placenta tissues and serum in salt-loaded rats till the end of pregnancy.

Although, Placenta weight change and peroxidative damages are important indexes in placenta toxicity in reproductive and developmental studies. It is important to identify histopathological changes in the placenta for a more accurate evaluation.[37] In this study, the placenta histology showed that the decidua cells in the placenta of rats on HSD were reduced in size. Decidua cells act as a barrier that separates the implanting blastocyst from their maternal tissue. It prevents the embryo from being attacked by maternal immune cells.[38] Thus, this maternal component of the materno-foetal interface provides nutrition, gas exchange and produces hormones.[39] The relationship between the decidua cell and the foetus is established post-conceptus immediately. Hence, any defects in the decidua development at this stage result in loss of pregnancy or complications at gestational. The necrosis of the decidua cells seen in rats on LSD was evidence of complications that resulted in intrauterine growth restriction (IUGR), also recorded in this group. The reduced size of chorionic villi and interstitial oedema seen in HSD rats are also indications of distorted morphology, which contributed to reduced foetal survival in the HSD rats.

The relationship between the weight of the placenta and its histopathology can be linked with its transportability and the metabolic mechanism used to determine alterations in nutrient exchange for foetal growth. An increase in the weight of the placenta is induced by an adaptive response to circulatory disturbance, which leads to hypertrophy of both the labyrinth and basal zone that cause a reduction in the number of the foetus.[40] as seen in rats on HSD. On the other hand, a decrease in placenta weight is also induced by hypoplasia of the labyrinth zone, which results from necrosis of the trophoblast and usually leads to IUGR, which predisposes to diseases later in life.

CONCLUSION

In conclusion, high salt diet during pregnancy impairs maternal ionic composition in the serum during pregnancy. These however, negatively affect the morphology of the placenta and caused an increase in oxidative stress which decreases the survival rate of the foetus.

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