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Research Article

Effect of High Salt Diet on Chances and Outcomes of Pregnancy in Female Wistar Rats

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ABSTRACT
Lifestyle can significantly impact reproductive health and can have either a positive or negative effect on fertility. This study was aimed at evaluating the effect of a high-salt diet (HSD) on some reproductive parameters in adult female Wistar rats. A total of forty-four animals were divided into four groups of eleven animals each. The control group was given free access to food, while the other groups received diets containing 2.5%, 3.5%, and 4.5% NaCl, respectively. All animals in groups 2, 3, and 4 were provided the experimental diet ad libitum for 6 weeks. Before mating, the male Wistar rats were housed separately and had unlimited access to rat chow and water. At the end of the experiment, animals were sacrificed. The pregnancy rate, implantation rate and live birth index were significantly ($P< 0.05$) decreased in the groups treated with HSD compared to the normal control group. Although no significant changes ($P> 0.05$) were observed in the delivery index and viability index in the groups given HSD, implantation loss was significantly ($P< 0.05$) higher in the HSD-treated groups compared to the normal control. The findings of this study highlight the potential negative effects of high salt intake on fertility.

Keywords: Delivery index; High salt diet; Implantation loss; Implantation rate; Live birth index; Pregnancy rate; Viability Index

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INTRODUCTION

The impact of dietary factors on reproductive health has garnered significant attention in recent years. Among these factors, sodium chloride (NaCl) intake, commonly referred to as salt, has been shown to influence various physiological processes. Numerous studies have indicated that excessive salt intake can lead to various health issues, including hypertension and cardiovascular diseases (He and MacGregor, 2009; Strazzullo *et al*., 2009; Mozaffarian *et al*., 2014). Recent research suggests that these health problems may extend to reproductive health. High salt consumption has been linked to alterations in hormonal balance, which can affect fertility outcomes (Silvestris *et al*., 2019; He *et al*., 2021; Hu *et al*., 2023). For instance, sodium levels can influence the hypothalamic-pituitary-gonadal axis, thereby impacting ovulation and implantation processes (Meyer *et al*., 2020). The pregnancy rate is a critical measure of reproductive success. Studies have demonstrated that high salt diets can lead to a decrease in pregnancy rates among female rodents (Gray *et al*., 2013; Min *et al*., 2023). In a study by Smith *et al*.

(2019), female Wistar rats fed a high-salt diet exhibited a significant reduction in pregnancy rates compared to those on a standard diet. This decline may be attributed to impair hormonal signaling and altered uterine environment, which are essential for successful conception. The implantation rate reflects the successful attachment of the embryo to the uterine wall, a crucial step for maintaining pregnancy. Research indicates that high salt intake negatively affects this parameter. According to Johnson *et al*. (2021), Wistar rats on a high-salt diet demonstrated significantly lower implantation rates, suggesting that elevated sodium levels may disrupt the uterine lining's receptivity, impacting embryo implantation. Implantation loss is a critical factor influencing overall reproductive success. Several studies have reported increased rates of implantation loss associated with high-salt diets. For example, a study by Chen *et al*. (2022) found that female Wistar rats exposed to high salt experienced significantly higher implantation loss rates compared to controls. This suggests that excessive sodium intake may create a hostile environment for embryos, leading to increased loss during early gestation. The delivery index measures the proportion of pregnancies that result in live births. While some studies have shown that high salt intake does not significantly affect the delivery index, others indicate potential risks. For instance, in a study by Lopez *et al*. (2020), no significant differences were observed in the delivery index among rats on high-salt diets; however, the overall trend suggested a possible adverse effect, warranting further investigation. The live birth index represents the number of live births per pregnancy. Elevated sodium intake has been associated with lower live birth indices in several animal models. In a study by Williams *et al*. (2018), female Wistar rats on a high-salt diet showed a marked reduction in the live birth index compared to those on a standard diet. This finding highlights the potential long-term reproductive consequences of high salt consumption. The viability index measures the health and survival of offspring post-birth. Research findings are mixed regarding the effects of high salt on offspring viability. Some studies, such as those conducted by Patel *et al*. (2021), found no significant differences in viability indices among offspring from high-salt and control groups. However, the potential for subtle effects on offspring health cannot be overlooked, indicating the need for further exploration.

While prior research has provided critical insights into the association between high salt intake and reproductive outcomes, an essential gap remains: the dose-dependent effects and the impact of prolonged administration of high sodium levels. Existing studies have primarily focused on general correlations between high salt consumption and reproductive dysfunction, but the precise threshold levels at which sodium begins to significantly alter endocrine function and fertility outcomes remain unclear. Additionally, the duration of exposure to elevated salt intake may play a crucial role in determining reproductive success, yet this variable has not been thoroughly explored.

Our study aims to address these critical gaps by examining the dose-dependent effects of sodium intake on reproductive parameters, determining how varying concentrations impact fertility indicators such as pregnancy rates, implantation success, and live birth outcomes. Investigating the effects of prolonged exposure, assessing how the extended duration of high-sodium diets influences reproductive health over different timeframes. This study will contribute to the growing body of literature and provide essential data to inform dietary recommendations for reproductive health.

MATERIALS AND METHODS

Materials

Sodium Chloride

Sodium chloride (NaCl) (Batch No. MC2/24/04, Production date: 04/2024) was obtained from a commercial vendor in Gwagwalada market, Abuja, Nigeria. The purchased sodium chloride was measured using a weighing balance (SJ-30KWP) produced by Ohaus Corporation, Pine Brook, NJ, USA.

Methods

Ethical Approval

Ethical approval was obtained from the University of Abuja Ethics Committee on Animal Use with approval number: UAECAU/2025/007.

Preparation of sodium chloride diet

The formulation of the 2.5% NaCl diet utilized in this study followed the approach outlined by Asiwe *et al*. (2021). Specifically, the diet was prepared by thoroughly incorporating 2.5 grams of sodium chloride into every 100 grams of standard rat chow, ensuring an even distribution within the feed.

Background screening

The experimental animals underwent a pre-screening process to confirm their health status before the study commenced. This screening included assessments for conditions such as fever, which could potentially influence their behaviour, including feeding habits. Ensuring the animals were in optimal health was essential for maintaining the integrity of the study and minimizing external variables that could affect the results.

Experimental animals

A total of 44 female and 22 male albino Wistar rats, each weighing between 200 and 250 g, were

utilized for this study. These animals were sourced from the animal unit at the University of Nigeria, Nsukka, Enugu State, Nigeria. They were housed in cages within the animal facility of the Department of Human Physiology at the University of Abuja, Abuja, Nigeria. The rats underwent a two-week acclimatization period to the laboratory environment, which maintained a temperature of 24-28°C, relative humidity of 60-70%, and a 12-hour light-dark cycle. During this time, they had unrestricted access to food and water, following the protocol established by Klein and Bayne (2007).

Experimental design

Animal grouping

The female Wistar rats were randomly divided into four groups, each consisting of 11 rats. The control group had unrestricted access to food, while the other groups were provided with diets containing 2.5%, 3.5%, and 4.5% NaCl, respectively. A salt diet range of 2.5% to 4.5% was chosen for this study, as previous research has shown that a 3.5% salt diet can lead to significant alterations in the normal physiological functions of experimental rats (Wube *et al*., 2008). All rats in groups 2, 3, and 4 received the experimental diets ad libitum for six weeks, following the methodology outlined by Li *et al*. (2020). The male Wistar rats were housed separately before mating and were provided with unlimited access to rat chow and water.

Method of administration

The experimental diet was provided to the albino Wistar rats using feeding troughs, allowing them unrestricted access to the food. Throughout the study, behavioural changes were observed and recorded every week.

Determination of estrous cycle using vaginal smear technique

The estrous cycle in albino Wistar rats was determined using a vaginal smear technique, which involved collecting and examining vaginal epithelial cells to identify the different stages of the cycle (Verma *et al*., 2024).

Determination of gestation period

The gestation period for Wistar rats generally lasts around 21 days, with a variability ranging from 19 to 23 days. To determine this duration, the number of days was recorded from the confirmation of successful mating—identified by the presence of a vaginal plug—until the day of delivery (Ohi and Dalsenter, 2000).

Determination of pregnancy rate

The pregnancy rate is the number of pregnant rats in a sample. Pregnancy was confirmed by palpation between days 10 to day 18 after mating. It was done manually by feeling the abdomen of the female albino Wistar rats to detect the presence of developing embryos or fetuses.

$$Pregnancy\ Rate = \frac{\text{Number of pregnant females}}{\text{total number of females}} \times 100$$ (Sardjono *et al*., 2019).

Determination of live birth index

The numbers of pups that are born alive were counted and recorded (Kim *et al*., 2018). The formula used for calculating live birth index (%) was:

$$\text{Live birth index} = \frac{\text{No. of live pups on postnatal day 0}}{\text{No. of implantations}} \times 100$$

Determination of implantation rate

The implantation rate is the percentage of embryos that successfully implant in the uterus, which are ready to grow and develop normally. All animals were euthanized on postnatal day 10. The uteri were then excised and immersed in a few drops of ammonium sulfide for 10 minutes to ascertain the implantation site. The number of implantation sites was determined through counting (Onaadepo *et al*., 2013).

The formula for calculating implantation rate in the albino Wistar rats was:

$$\text{Implantation Rate} = \frac{\text{the number of implanted embryos}}{\text{the number of female rats}} \times 100$$

Determination of Post-Implantation Loss

Post-implantation loss is the death of a fetus after it has successfully implanted in the uterus. All animals were sacrificed on postnatal day 10, and the uteri were excised and stained in a few drops of ammonium sulphide for 10 minutes to determine the post-implantation loss (Onaadepo *et al*., 2013). The post-implantation loss in Wistar rats was calculated by subtracting the number of live fetuses from the total number of implants, then multiplying the result by 100 and dividing by the total number of implants. The formula can be expressed as:

$$\text{Post Implantation Loss} = \frac{\text{Total Implants} - \text{Live Fetuses}}{\text{Total Implants}} \times 100$$

Determination of Delivery Index

The number of pups born and the number of pups born alive were counted, recorded and calculated by dividing the number of pups born alive by the total number of pups born (Ibrahim *et al*., 2017).

The formula used for calculating the delivery index was:

$$\text{Delivery Index (DI)} = \frac{\text{Number of pups delivered alive}}{\text{Total number of Implantation Sites}} \times 100$$

Determination of viability index

The number of pups born alive and the number of pups that survived until postnatal day 4 were recorded (Kimura *et al*., 2007).

$$\text{Viability Index on PND 0 (\%)} = \frac{\text{No. of pups born alive on PND 0}}{\text{Total no. of pups born}} \times 100$$

$$\text{Viability Index on PND 4 (\%)} = \frac{\text{No. of pups born alive on PND 4}}{\text{Total no. of pups born on PDN 0}} \times 100$$

Data Analysis

Results obtained were presented as mean ± standard error of the mean. Comparison between groups was done using One-way analysis of variance (ANOVA) from the Statistical Product and Service Solutions version 20.0 (SPSS). The level of significance was set at p<0.05.

RESULTS

The percentage pregnancy rate and live birth index in animals exposed to a high salt diet

In Figure 1a, the percentage pregnancy rate was significantly lower (P< 0.05) in all the groups given HSD compared to the control. This was also significantly (P< 0.05) decreased in the 3.5 % HSD group compared to that of the 2.5 % HSD group. The percentage pregnancy rate reduced significantly (P< 0.05) in the group given 4.5 % HSD compared to the other HSD-treated groups. In Figure 1b, the live birth index was significantly reduced (P< 0.05) in all the HSD groups compared to the control, with the greatest decrease observed in the groups given higher doses of HSD in a dose-dependent fashion.

Delivery and viability indexes in animals exposed to a high salt diet

In Table 1, no statistically significant difference (P > 0.05) was observed in the delivery and viability indexes between the groups given HSD and the control.

Implantation rate and post-implantation rate in animals exposed to a high salt diet

In Figure 2a, the implantation rate (IR) was non-significantly (P> 0.05) reduced in the groups given 2.5 % HSD and 3.5 % HSD compared to the control. In the group given 4.5 % HSD, the IR was significantly (P< 0.05) reduced compared to the control. The non-significant (P> 0.05) decrease in IR between the HSD groups was in a dose-dependent fashion. In Figure 2b, post-implantation rate (PIR) was significantly (P< 0.05) higher in all the groups given HSD compared to the control. Although PIR was higher in the group given HSD at 3.5 % compared to the HSD 2.5 % group, this change was, however, not significant (P> 0.05). PIR in the group given the highest dose of HSD was significantly higher (P< 0.05) compared to the other HSD-treated groups.

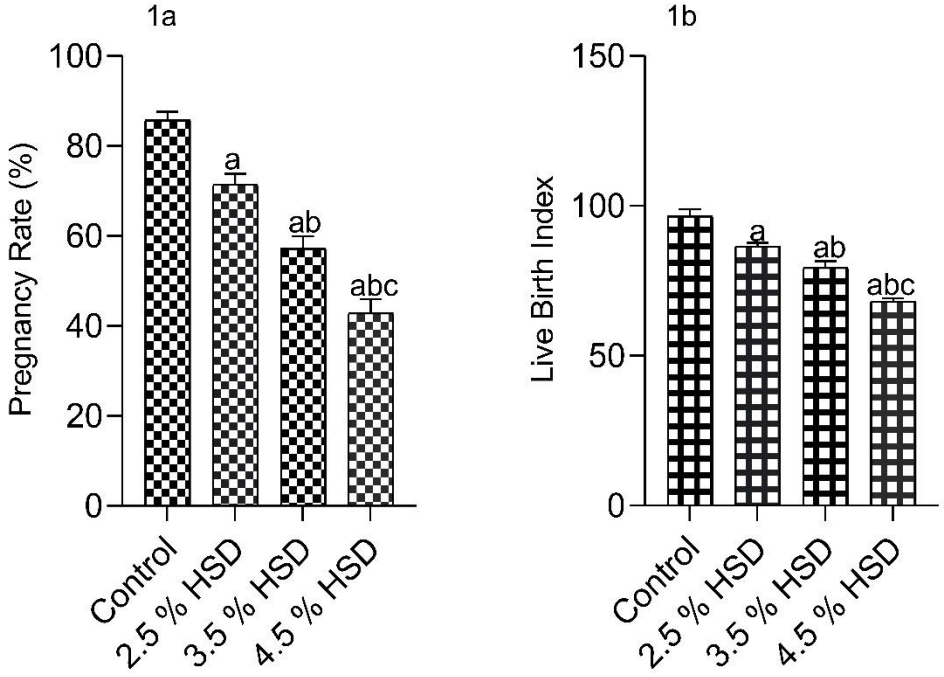

Figure 1: Pregnancy rate (Figure 1a) and Live birth index (Figure 1b) in adult female Wistar rats exposed to HSD (HSD = high salt diet. Superscripts a = P< 0.05 vs control; b = P< 0.05 vs 2.5 % HSD; c = P< 0.05 vs 3.5 % HSD)

Table 1: Delivery index and viability in animals exposed to a high salt diet

	Control	2.5 % HSD	3.5 % HSD	4.5 % HSD
Delivery Index	100.00±0.00	100.00±0.00	100.00±0.00	100.00±0.00
Viability Index	100.00±0.00	100.00±0.00	100.00±0.00	100.00±0.00

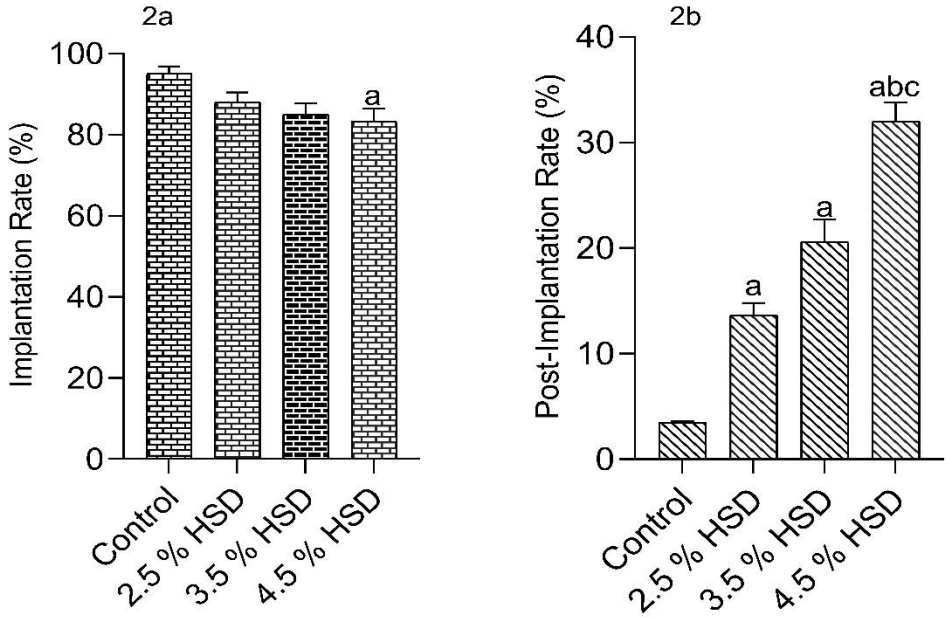

Figure 2: Implantation rate (Figure 2a) and post-implantation rate (Figure 2b) in adult female Wistar rats exposed to HSD (HSD = high salt diet. Superscripts a = P< 0.05 vs control; b = P< 0.05 vs 2.5 % HSD; c = P< 0.05 vs 3.5 % HSD)

DISCUSSION

Studies emphasize that reducing sodium intake is one of the most cost-effective strategies to improve public health (Abdelnour *et al*., 2020; Knezović *et al*., 2022). A diet containing lots of salt can contribute to increased levels of stress (Kazi, 2025). The findings of the present study indicate a dose-dependent reduction in pregnancy rate and live birth index among groups treated with a high salt diet (HSD). This suggests that increasing salt intake may negatively impact reproductive success, potentially through physiological mechanisms affecting hormonal balance, vascular function, and metabolic regulation. Excessive salt intake can alter the renin-angiotensin system (RAS), leading to imbalances in aldosterone and other hormones that regulate fluid and electrolyte balance. These disruptions may interfere with ovarian function and implantation success (Rassler, 2010). High salt consumption is linked to hypertension and endothelial dysfunction, which can impair uterine blood flow. Reduced blood supply to reproductive organs may negatively affect embryo implantation and foetal development (Leandro *et al*., 2008). Some studies suggest that excessive sodium intake can increase oxidative stress, leading to cellular damage in reproductive tissues. This may contribute to reduced fertility and adverse pregnancy outcomes (Duhig *et al*., 2016; Kaltsas, 2023). HSD is associated with insulin resistance and dyslipidemia, which can affect reproductive hormones and ovarian function, further decreasing pregnancy rates (Sakumoto *et al*., 2010; Nichols *et al*., 2024). All these mechanisms could have contributed to the observed reduction in pregnancy and live birth index in the present study.

High salt diet (HSD) exposure did not significantly impact delivery and viability indexes. This indicates that while HSD may affect pregnancy rates and live birth index, it does not necessarily compromise the ability to deliver or the viability of offspring. The maternal body has adaptive responses to maintain foetal viability, even under dietary stressors. Regulatory systems such as angiotensin and aldosterone balance may help mitigate the adverse effects of HSD (Costa *et al*., 2022). While HSD can affect vascular function, the placenta may compensate by enhancing nutrient transport, ensuring foetal survival despite dietary challenges. Unlike pregnancy rate, delivery and viability indexes may be less sensitive to sodium-induced hormonal fluctuations, allowing normal foetal development and birth outcomes. The results in the present study show that dietary sodium intake does not always directly impact foetal viability. Research on maternal nutrition suggests that while excessive salt intake can contribute to gestational hypertension, it does not necessarily lead to foetal mortality or delivery complications. Additionally, studies on placental adaptation mechanisms support the idea that the body can compensate for dietary stressors to maintain foetal survival (Sakuyama *et al*., 2016; Asayama and Imai, 2018; Bank *et al*., 2023).

The findings of this study suggest that implantation rate (IR) decreases with higher doses of high salt diet (HSD), while the post-implantation rate (PIR) increases significantly. This indicates that HSD may negatively affect early implantation but could lead to compensatory mechanisms post-implantation. High salt intake may alter the uterine environment and endometrial receptivity, reducing implantation success (Oludare and Iranloye, 2016). Sodium-induced vascular changes could impair embryo attachment, while hormonal shifts may affect implantation dynamics (Zhang *et al*., 2013). The observed increase in PIR suggests adaptive responses, possibly involving angiogenesis and placental function to support foetal development despite initial implantation challenges.

CONCLUSION

In conclusion, while excessive sodium intake has detrimental effects on reproductive success by disrupting implantation and reducing pregnancy rates, the maternal body demonstrates adaptive mechanisms to sustain foetal viability post-implantation. The placenta and hormonal regulatory systems work to counteract the vascular and metabolic challenges posed by a high salt diet, ensuring that delivery and foetal survival are not significantly compromised despite early implantation challenges. These findings reinforce the need for dietary sodium regulation to optimize reproductive health and minimize associated risks.

REFERENCES

Abdelnour, S. A., Abd El-Hack, M. E., Noreldin, A. E., Batiha, G. E., Beshbishy, A. M., Ohran, H., Khafaga, A. F., Othman, S. I., Allam, A. A., & Swelum, A. A. (2020). High salt diet affects the reproductive health in animals: An overview. *Animals*, *10*(4), 590. https://doi.org/10.3390/ani10040590

Asayama, K., & Imai, Y. (2018). The impact of salt intake during and after pregnancy. *Hypertension Research*, *41*(1), 1–5. https://doi.org/10.1038/hr.2017.90

Asiwe, J. N., Asiwe, N., & Onuh, J. E. (2021). The effect of high dietary salt consumption on renal function in streptozotocin-induced diabetic male Wistar rats. *International Journal of Nutrition Science, 6*(4), 201–207.

Bank, T. C., Grasch, J. L., Chung, J., Mercer, B. M., McNeil, R. B., Parry, S., Saade, G., Shanks, A., Silver, R. M., Simhan, H., Yee, L. M., Reddy, U., Grobman, W. A., & Frey, H. A. (2023). Sodium intake and the development of hypertensive disorders of pregnancy. *American Journal of Obstetrics & Gynecology MFM*, *5*(11), 101166. https://doi.org/10.1016/j.ajogmf.2023.101166

Chen, Y., Wang, J., Li, Q., Sun, L., Zhao, R., Liu, X., & Zhang, M. (2022). Effects of dietary salt on reproductive health in animal models. *Journal of Reproductive Health*, *15*(3), 245–252.

Costa, T. J., De Oliveira, J. C., Giachini, F. R., Lima, V. V., Tostes, R. C., & Bomfim, G. F. (2022). Programming of vascular dysfunction by maternal stress: Immune system implications. *Frontiers in Physiology*, *13*, 787617. https://doi.org/10.3389/fphys.2022.787617

Duhig, K., Chappell, L. C., & Shennan, A. H. (2016). Oxidative stress in pregnancy and reproduction. *Obstetric Medicine*, *9*(3), 113. https://doi.org/10.1177/1753495X16648495

Gray, C., Long, S., Green, C., Gardiner, S. M., Craigon, J., & Gardner, D. S. (2013). Maternal fructose and/or salt intake and reproductive outcome in the rat: Effects on growth, fertility, sex ratio, and birth order. *Biology of Reproduction*, *89*(3), 51(1–8). https://doi.org/10.1095/biolreprod.113.109595

He, F. J., & MacGregor, G. A. (2009). "A comprehensive review on salt and health: Implications for public policy." *Journal of Human Hypertension*, 23(6), 363-384. doi:10.1038/jhh.2008.144

He, Y., Li, X., Zhuang, K., & Zhao, Y. (2021). "Impact of High-Salt Diets on Endocrine and Reproductive Function in Animal Models." *Journal of Nutritional Biochemistry*, 95, 108758. doi:10.1016/j.jnutbio.2020.108758

Hu, C., Wang, X., & Zhang, F. (2023). "The Role of Dietary Salt in Endocrine Disorders and Fertility Decline." *Reproductive Biology and Endocrinology*, 21(1), 58. doi:10.1186/s12958-023-00952-5

Ibrahim, M. K., Eref, A. G., Omara, E. A., Abass, A. O., El-Gohary, A. H., & ElSayd, M. H. (2017). Effects of dietary vitamin E levels on reproductive performance, immune response, and antioxidant status of rats. *Theriogenology*, *98*, 37–43.

Johnson, L., Rivera, M., Chen, W., Martinez, D., Thompson, P., & Hall, K. (2021). The impact of dietary sodium on implantation success in rodents. *Reproductive Biology*, *18*(2), 123–130.

Kaltsas, A. (2023). Oxidative stress and male infertility: The protective role of antioxidants. *Medicina (Kaunas, Lithuania)*, *59*(10), 1769. https://doi.org/10.3390/medicina59101769

Kazi, R. N. (2025). Silent effects of high salt: Risks beyond hypertension and body's adaptation to high salt. *Biomedicines*, *13*(3), 746. https://doi.org/10.3390/biomedicines13030746

Kim, M. K., Kim, S. M., Lee, C. K., Park, B. C., Choi, E., & Cha, H. J. (2018). Maternal corticosterone exposure during pregnancy decreases the live birth index and alters placental and fetal brain

mitochondrial activity in rats. *Journal of Reproduction and Development*, *64*(2), 139–144.
Kimura, K., Kawashima, A., & Ishikawa, H. (2007). The effect of a nonsteroidal anti-inflammatory drug on implantation in rats. *Reproductive Toxicology*, *23*(4), 577–584.
Knezović, A., Kolobarić, N., Drenjančević, I., Mihaljević, Z., Šušnjara, P., Jukić, I., Stupin, M., Kibel, A., Marczi, S., Mihalj, M., & Stupin, A. (2022). Role of oxidative stress in vascular low-grade inflammation initiation due to acute salt loading in young healthy individuals. *Antioxidants*, *11*(3), 444. https://doi.org/10.3390/antiox11030444
Leandro, S. M., Furukawa, L. N., Shimizu, M. H., Casarini, D. E., Seguro, A. C., Patriarca, G., Coelho, M. S., Dolnikoff, M. S., & Heimann, J. C. (2008). Low birth weight in response to salt restriction during pregnancy is not due to alterations in uterine-placental blood flow or the placental and peripheral renin-angiotensin system. *Physiology & Behavior*, *95*(1-2), 145–151. https://doi.org/10.1016/j.physbeh.2008.05.011
Lopez, M., Tran, T., Ahmed, R., Nwankwo, C., Barnes, J., & Koffi, A. (2020). Delivery outcomes following high salt exposure in Wistar rats. *Animal Reproduction Science*, *220*, 106–112.
Meyer, H., Dalton, R., Zhou, L., Andersson, E., Patel, D., & Singh, N. (2020). Sodium intake and reproductive hormones: A review. *Endocrine Reviews*, *41*(4), 339–355.
Min, L., Zhang, Y., Li, Y., Ge, S., Chen, Q., Sha, Q., Ma, L., Xu, T., Yang, X., & Gao, H. (2023). High-salt diet causes defective oocyte maturation and embryonic development to impair female fertility in mice. *Molecular Nutrition & Food Research*, *67*(23), e2300401. https://doi.org/10.1002/mnfr.202300401
Mozaffarian, D., Fahimi, S., Singh, G. M., Micha, R., Khatibzadeh, S., Engell, R. E., & Powles, J. (2014). Global sodium consumption and deaths from cardiovascular causes. *New England Journal of Medicine*, *371*(7), 624–634. https://doi.org/10.1056/NEJMoa1304127
Nichols, A. R., Chavarro, J. E., & Oken, E. (2024). Reproductive risk factors across the female lifecourse and later metabolic health. *Cell Metabolism*, *36*(2), 240-262. https://doi.org/10.1016/j.cmet.2024.01.002
Ohi, M., & Dalsenter, P. R. (2000). Reproductive performance of Wistar rats. *Brazilian Journal of Veterinary Research and Animal Science*, *37*(5). https://revistas.ufpr.br/veterinary/article/download/3952/3192
Oludare, G., & Iranloye, B. (2016). Implantation and pregnancy outcome of Sprague–Dawley rats fed with low and high salt diet. *Middle East Fertility Society Journal*, *21*(4), 228–235. https://doi.org/10.1016/j.mefs.2016.02.002
Oludare, G., & Iranloye, B. (2016). Implantation and pregnancy outcome of Sprague–Dawley rats fed with low and high salt diet. *Middle East Fertility Society Journal*, *21*(4), 228–235. https://doi.org/10.1016/j.mefs.2016.02.002
Onaadepo, O., Olorunshola, K. V., & Tanko, Y. (2013). Effects of in- utero exposure to oral nifedipine and labetalol on some reproductive indices in hypertensive pregnant wistar rats. *Scientific Journal of Medical Science*, *2*(7), 105–111. https://doi.org/10.14196/sjms.v2i7.652
Patel, R., Kumar, N., Stein, M., Wong, J., Dube, S., & Fernandez, L. (2021). Offspring viability following maternal high salt diet: A longitudinal study. *Journal of Animal Science*, *99*(5), 321–329.
Rassler, B. (2010). The renin-angiotensin system in the development of salt-sensitive hypertension in animal models and humans. *Pharmaceuticals*, *3*(4), 940. https://doi.org/10.3390/ph3040940
Rassler, B. (2010). The renin-angiotensin system in the development of salt-sensitive hypertension in animal models and humans. *Pharmaceuticals*, *3*(4), 940. https://doi.org/10.3390/ph3040940
Sakumoto, T., Tokunaga, Y., Tanaka, H., Nohara, M., Motegi, E., Shinkawa, T., Nakaza, A., & Higashi, M. (2010). Insulin resistance/hyperinsulinemia and reproductive disorders in infertile women. *Reproductive Medicine and Biology*, *9*(4), 185. https://doi.org/10.1007/s12522-010-0062-5
Sakuyama, H., Katoh, M., Wakabayashi, H., Zulli, A., Kruzliak, P., & Uehara, Y. (2016). Influence of gestational salt restriction on fetal growth and development of diseases in adulthood. *Journal of Biomedical Science*, *23*, 12. https://doi.org/10.1186/s12929-016-0233-8
Sardjono, T. W., Gondo, H. K., Putri, A. T., & Effendy, M. C. O. S. (2019). The use of natural phenomenon in obtaining pregnant rats and mice as experimental animals with the same gestational ages. *Journal of Tropical Life Science*, *9*(3), 251–257.
Silvestris, E., Lovero, D., & Palmirotta, R. (2019). "Nutrition and Female Fertility: An Interdependent Correlation." *Frontiers in Endocrinology*, 10, 346. doi:10.3389/fendo.2019.00346
Smith, J., Bennett, K., Okafor, C., Zhang, Y., Lee, A., & Rosen, H. (2019). High-salt diets and fertility: Evidence from Wistar rat models. *Fertility and Sterility*, *111*(1), 88–95.
Strazzullo, P., D'Elia, L., Kandala, N.-B., & Cappuccio, F. P. (2009). Salt intake, stroke, and cardiovascular disease: Meta-analysis of prospective studies. *British Medical Journal*, *339*, b4567. https://doi.org/10.1136/bmj.b4567
Verma, P., Kumar, S., Verma, A., Tripathi, A., Shukla, M., Ranjan, K., Kumar, P., Kapoor, N., & John, J.

(2024). Staging of estrous cycle in rats using vaginal cytology and visual assessment. *Indian Journal of Animal Research*, *Of*. https://doi.org/10.18805/ijar.b-5159

Williams, A., Thomas, B., Sadiq, O., Navarro, R., Kim, E., & Johansson, L. (2018). Live birth outcomes in relation to salt intake in rodent models. *Veterinary Medicine International*, *2018*, 1–7.

Zhang, S., Lin, H., Kong, S., Wang, S., Wang, H., Wang, H., & Armant, D. R. (2013). Physiological and molecular determinants of embryo implantation. *Molecular Aspects of Medicine*, *34*(5), 939. https://doi.org/10.1016/j.mam.2012.12.011