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*Full length Research Article*

# Exercise Attenuates the Effect of High Salt Intake on the Cardiovascular Function, Oxygen Saturation, Lung Function and Renal Function of Young Men

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**Summary:** Coronary heart disease (CAD), respiratory disease, and early-onset renal failure, which until recently were only common in high-income countries, are now the dominant source of morbidity and mortality among young Nigerian adults. However, epidemiological studies have suggested the possibility of high dietary salt intake and physical inactivity as behavioural factors that may be responsible for this growing trend. Therefore, the purpose of this study was to determine the influence of elevated salt intake and physical activity and inactivity on the pulmonary function, cardiovascular, and renal function of young Nigerian men. Methods and materials: A total of 20 subjects, comprising 10 non-exercising young men (control) and 10 exercising young men, participated in the study after obtaining an approval from the ethical committee of the animal and human research bioethics department. Lung function, oxygen saturation, blood pressure, urine volume, urine pH, and urine Na+ and K+ concentration were measured under resting conditions before and after five days of 200mmol of salt loads in both groups. The data was analyzed using the SPSS Statistics software package Version 19. The unpaired t-test was used to calculate the P-value across the groups. The paired t-test was used to calculate the p-value within the groups. Statistical significance was reached when $P < 0.05$. Results: Salt loading had no significant effect on the pulmonary function of the control subjects. However, salt loading worsened the pulmonary function values of the exercising subjects, with FEV1, FVC, and PEFR decreased significantly by -0.05 ± 0.05 L, -0.003 ± 0.01 L, and -20.20 ± 7.11 L/min, respectively, without affecting oxygen saturation (SPO2) and FEV1%. Salt loading caused a greater increase in the blood pressure parameters of the non-exercising subjects, with systolic pressure, diastolic pressure, mean arterial pressure, and pulse pressure significantly increased by 18.00 ± 2.04 mmHg, 11.90 ± 1.52 mmHg, 13.97 ± 1.98 mmHg, and 6.20 ± 0.24 mmHg, respectively. In summary, exercising subjects eliminate salt loads more effectively than the non-exercising subjects to reduce salt retention. This might be as a result of the trigger of some pathophysiological mechanisms that alters vital bodily functions such as respiratory function, renal function, and cardiovascular functions.

**Keywords***: Oxygen Saturation, pro-inflammatory cytokines, Hyperinsulinemia, angiotensin-2, and Nitric oxide.*

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## INTRODUCTION

Exercise is a stressful condition that serves as a means of testing the physical capabilities and physiological responses of an individual. It forms the basis for good health and wellbeing with the ability or capacity to endure stress. The burden of unhealthy diets constitutes a major public health concern globally and urgent action is needed to address this growing trends. The over-production and over-consumption of foods and beverages that do not meet healthy nutritional standards, mainly manufactured foods are of great concern due to the high amount of sodium they contain. In most developed and developing countries, a significant proportion of sodium in the diet comes from processed foods, particularly from seasoning and condiments used for cooking to add taste to food or even from fast foods we get from restaurants and canteens. The global mean intake of sodium is about 4310 mg/day (equivalent to 10.78 g/day of salt) which is more than double the World Health Organization's recommendation for salt intake in adults (< 2000 mg of sodium per day, an equivalent to < 5 g of salt per day). This doubling in the consumption of salt in this time and age of modern civilization has really become a public health concern. An estimated 1.89 million deaths each year have been associated with consuming too much sodium, a well-established cause of raised blood pressure and an increased risk of cardiovascular disease (IHME, 2019). Scientists have become aware of the health risks associated with high salt intake, which include hypertension, chronic obstructive pulmonary conditions, renal failures, and other anomalies triggered by high dietary salt intake (AHA, 2009). The ability of regular exercise to augment the bioavailability of Nitric Oxide can have a beneficial influence to counter the effect of this high dietary salt intake and its associated health risk on the respiratory system (Uematsu *et al.*,1995). Several epidemiological

studies have linked dietary salt to the prevalence and severity of obstructive lung conditions particularly in a population on a high salt diet (Burney,1995). Recent studies show dietary salt loading increases serum osmolality by stimulating the systemic increase in pro-inflammatory cytokines (Philipp *et al*., 2006), which has been found to have the potential to damage the lungs and reduce lung function. Another fact points out the influence of dietary salt on circulating blood volume and consequently on hemodynamic functions as another possible link that may relate the effect of dietary salt on lung function and oxygen saturation. The aim of the study is to evaluate the effect of dietary factors such as high salt intake and lifestyle choices of active or sedentary lifestyle on lung function, oxygen saturation, blood pressure, and renal function amongst young men.

## MATERIALS AND METHODS

**Ethical consideration:** An ethical approval with CMUL/ACUREC/05/2012/1635 was obtained from the research and bioethics committee of the animal and research bioethics department before the experiment. An informed consent form was also issued and completed by each participant before the commencement of the experiment.

**Participants:** The experiment was carried out on 20 healthy young male subjects. The subjects were all on a normal diet and none of them were on any type of medication for any disease condition that could affect the result obtained from the experiments. Selection of subjects was based on the selection criteria below. The Selection of the participants was done based on the following criteria: No clinical history of asthma; No clinical history of sickle cells anemia, No history of alcohol or substance abuse. Only healthy male volunteers between the age of 20 – 27 years were eligible for the study.

**Research Design** For the purpose of the study, the participants were all students of the College of Medicine, University of Lagos (CMUL), Idi-araba, Lagos, Nigeria; all living under hygienic condition within the premises of the medical student hostel, CMUL, Idi-araba. A food intake survey was designed to identify sources of dietary salt. Questionnaires were administered to all participants to assess different patterns of meal consumption. The questionnaire was divided into five sections which includes frequency of meal consumption on campus, frequency of meal consumption off campus, frequency of consuming certain food groups that may be a potential source contributing to dietary salt, frequency of consuming certain local dishes that are high in salt, frequency of preparing their own meal. The food group listed in the food intake survey were selected from nutrient composition of Nigerian foods (Tindel, 1985) based on food identified by (Towobola *et al*., 2013) as being high in salt and food group with at least moderate salt content(>100mg sodium /100g).The food intake survey questionnaire (appendices-1) which was administered to all volunteers was aimed at establishing the dietary intake of all participants. The result of the food survey was used in grouping the participants

**Grouping of participants:** The participants were grouped into two groups of ten people each. The groups are; the exercise group, consisting of undergraduate medical students who engaged more frequently in exercises and consumed more frequently food high in salt according to the result of the food intake survey. The second group is the non-exercising group that consisted of participants from among the undergraduate medical students of the university who do not engage more frequently in exercise and consumed food high in salt according to the food intake survey. Participants in each group served as their own control. Anthropometric parameters such as height (metres), weight (Kg), Body Mass (Kg/m2) index, Body Surface area (m2) were measured for each participants. Participants in each group were subjected to a baseline measurement of lung function, oxygen saturation and blood pressure before the consumption of a high salt diet of 200mmol sodium for five days to increase their salt intake. Exercise group engage in an endurance exercise of cycling on a bicycle ergometer to measure the same parameters.

**Salt-loading:** Subjects were loaded with 200mmol sodium chloride solution on a daily basis for a period of 5 days ( Tzemoz *et al*., 2008). This was prepared by dissolving 10.9g scientific graded salt (sigma - Alderich, AHANALA GRADE) in 700mls of water mixed with 300mls of orange squash to make up a whole salt solution. The treatment was well tolerated by all subjects.

**Measurement of lung function (FEV1, FVC, FEV1, %) and oxygen saturation (SPO2):** The lung function and oxygen saturation were measured using the Spirolab III, an open circuit spirometer connected to a pulse oximeter (MIR model). A multi-tasking and a versatile spirometer with a high-performance personal computer software (winspiro - Pro) that automatically generated a participant data card with a preview of the spirometry curve. The spirolab 111 measures the lung function (FVC, FEV, FEV1%) by forcefully breathing in and forcefully breathing out into the turbine of the SPIROLAB via a disposable mouthpiece with the nose clipped. The oxygen saturation was measured by a pulse oximeter connected to the spirolab II1 via a cable. It measures the oxygen saturation by inserting the index finger into the probe of the pulse oximeter, until it picks up the signal which it sends to the spirolab III for processing before displaying on the screen. The lung function and oxygen saturation were measured in the two groups of participants before and after salt loading to have a baseline FVC, FEV1, FEV1% and SPO2 (PRE-TEST) and a test FVC, FVC1,
FEV1% and SPO2 (post-test)

**Collection of 12 hours urine sample:** This was carried out before and after the salt loading protocol. The 12 hours urine sample was collected before and after salt loading into a 2 liters urine collection bottle containing chloroform. The chloroform was used as preservative to prevent bacterial growth in the urine. Subjects were given verbal instructions on how to collect a 12-hour urine sample. They were told to start collection at 9.00pm, urine voided before this time was discarded. Thereafter urine voided as from 9.00pm was collected into the 2 liters urine collection bottle containing chloroform for duration of 12hours i.e. 9.00am of the

following day. Using a 500mls measuring cylinder, the volume of the 12 hours urine sample was accurately measured. The pH of the urine samples were also determined using a pH meter.

**Determination of urine sodium and potassium concentration:** The concentration of sodium and potassium in 12 hours urine samples were analyzed using the ion selective electrode method (ISE method). This involves the use of ISE 600 Analyzer (ISE 600 by SFRI) which analyzes the urine samples after calibration by aspirating sample of urine to determine the concentration of Na+ and K+ in urine. Results are displayed on the screen after analysis. The observed values of the samples were expressed in mmol/L Na+ and k+

**Determination of blood pressure (mmHg):** The blood pressure for each participant was measured in the sitting position with their back rested firmly on the chair and their feet flat on the floor. Subjects were allowed a 5-minute period of rest in this position before commencement of blood pressure measurement.

Subjects were not allowed to wear constrictive clothing on the upper arm in order to avoid tourniquet effect above the Riva – roc-cuff Subjects arm was supported at the level of the heart. The Riva -Rocci cuff of the sphygmomanometer was placed in a way to encircle at least 80% of the arm circumference. Arterial blood pressure measurement was taken from the brachial artery pulsation of the right arm with the arm in the supine position; systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded as the first and fifth korot- koff phases respectively (Kim *et al*.,2023). The cuff was deflated at the rate of 2mmHg per sec. Measurements were taken to the nearest 2mmHg to avoid digit preference. Digit bias or digit prejudice is a situation in which the observer records an inappropriate excess of "Zero" as the last digit in blood pressure recording/ depending on the subjects' circumstance; the observer records the blood pressure just below or just above a cutoff point (Wing field *et al*., 2002). After initial measurement to avoid "White coat" effects, three reading were taken with one minute interval in between for the subjects to relax: the average of the three measurements was recorded.

**Measurement of arterial blood pressure:** Arterial blood pressure measurement was measured using a non- invasive indirect auscultatory method by using the stethoscope and a mercury Sphygmomanometer. The gold standard in the measurement of blood pressure is the use of mercury sphygmomanometer and the Korotkoff sound technique using the auscultatory method (Pickering *et al*., 2005). The auscultatory method is the mainstay of blood pressure methods but is gradually being supplanted by other techniques due to increase in the use of automated blood pressure measuring devices (Pickering *et al*., 2005). The mercury sphygmomanometer consists of a mercury column inside a vertical column to which is attached the riva – rocci inflatable Cuff and an inflator. This device has changed little since it was designed about six decades ago except for the fact that modern versions have very little risk of spilling mercury if dropped. The mercury sphygmomanometer is unique in its simplicity and there is negligible difference in the accuracy of different brands of the instrument (Pickering *et al*., 2005). Baseline blood pressure (mmHg) was determined following the standardized protocol developed by the international collaborative study of hypertension in blacks (ICSHIB) and the American Heart Association (AHA) recommendation for blood pressure measurement in Human (Pickering *et al*., 2005).

**Determination of Pulse pressure and Mean arterial pressure:** Pulse pressure was determined by subtracting the diastolic blood pressure from the systolic blood pressure. Mean arterial blood pressure was determined by adding one third of pulse pressure to the diastolic pressure. PP = Diastolic Blood Pressure (DBP)-Systolic Blood Pressure (SBP)

MAP = 1/3 Pulse Pressure (PP) + Diastolic Blood Pressure (DBP)

**Determination of change and % change of parameters after salt loading:** Changes in the parameters are those changes computed by subtracting an initial value from a final value divided by an initial value. Change (Δ) = final value-initial value

Initial value. % change = (final value-initial value )× 100
Initial value

Data have been summarized and presented as mean ± standard error of mean (S.E.M) and range. A statistical test of significance was prepared at 95% confidence level in order to show how significant the results would deviate from control and thus able to draw conclusion. The student's unpaired t-test was used to calculate the P-value across the group. The students paired t-test was used to calculate the pvalue within the group. Statistical significance was adopted when $P < 0.05$.

## RESULTS

**Results of food questionnaire:** Most of the students had food on campus on a daily basis. Only few of the students had food off campus**.** Most students added soy, salt and sauce to served food on a daily basis. Most of the students consumed more of bakery, salted egg and instant meal than they did consume local foods. Most of the students hardly did their own cooking. Most of the student admitted of adding salt, sauce and fish sauce while cooking. The result of the questionnaire showed that most food consumed more frequently by the students are foods that fail under the category of foods that are high in salt contents as identified by Towobola *et al*.(2013).

**Comparison of baseline anthropometric parameters in non- exercising and exercising young men:** Comparison of baseline anthropometric parameters in both groups of subjects (Table 1) reveal they was within the same age, height, weight, body surface area and body mass index limit. There was no significant difference between the control group and the exercising group.

**Comparison of baseline ventilatory function in non-exercising and exercising young men:** Comparison of the baseline ventilatory function in both groups (Table 2) reveals that oxygen saturation (SPO2), forced expiratory volume in 1 sec (FEV1), forced vital capacity (FVC) and

peak expiratory flow rate (PEFR) differed significantly in both groups (Fig. 1-3) with P < 0.05 and 0.001 respectively. Percentage forced expiratory volume in 1 sec (FEV1%) in both groups were similar.

**Comparison Of Baseline Blood Pressure in Non-Exercising And Exercising Young Men:** Comparison of baseline blood pressure parameters in both groups (Table 3) revealed that only systolic blood pressure differed significantly between the groups. There was no significant difference in the diastolic blood pressure, mean arterial blood pressure and pulse pressure of both groups (Fig. 4).

**Table 1:**
Baseline anthropometric parameters in non-exercising and exercising young men

| Parameters | Non-Exercising Subjects (Mean ± S.E.M) | Range | Exercising Subjects (Mean ± S.E.M) | Range | Remarks |
|---|---|---|---|---|---|
| **Age (yr)** | 23.00 ± 0.88 | 20-28 | 22.90 ± 0.91 | 20-27 | NS |
| **Height (m)** | 1.70 ± 0.02 | 1.60-1.80 | 1.75 ± 2.80 | 1.64-1.91 | NS |
| **Weight (kg)** | 62.00 ± 2.05 | 53-71 | 68.30 ± 2.77 | 53-85 | NS |
| **BMI (kg/m$^2$)** | 21.20 ± 0.46 | 18.83-23.16 | 22.39 ± 1.27 | 17.31-31.21 | NS |
| **BSA (M2)** | 1.72 ± 0.03 | 1.54-1.90 | 1.82 ± 0.04 | 1.64-1.96 | NS |

*Values are expressed as mean ± S.E.M; NS =Not significant*

**Table 2:**
Comparison of baseline ventilatory function in non-exercising and exercising young men

| N= 20 | MEAN ± S.E.M | | |
|---|---|---|---|
| **PARAMETERS** | **Non-Exercising Subjects** | **Exercising Subjects** | |
| FVC (Litre) | 2.73 ± 0.15 | 3.93 ± 0.20*** | SIG |
| FEV1 (Litre) | 2.45 ± 0.13 | 3.58 ± 0.21* | SIG |
| FEV1% | 90.49 ± 2.47 | 90.94 ± 2.17 | NS |
| PEFR (L/MIN) | 411.40 ± 22.70 | 511.30 ± 24.83*** | SIG |
| SPO2 (%) | 95.95 ± 0.55 | 97.40 ± 0.18* | SIG |

*Values are expressed as mean ± S.E.M; SIG =Significant .NS =Not significant*
**= P < 0.05, ***= P < 0.001 respectively*
*FEV1 = forced expiratory volume in 1 sec; FVC = forced vital capacity; PEFR = Peak expiratory flow rate; SPO2 = oxygen saturation*

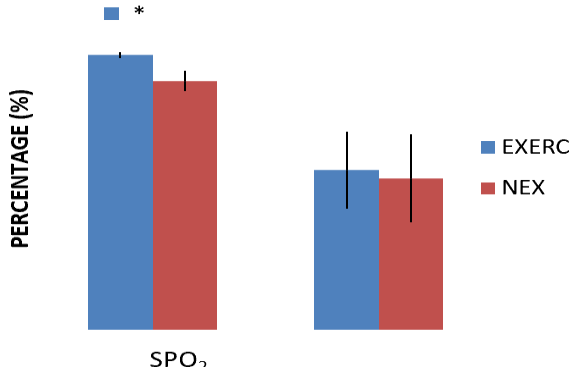



**Figure 1:**
Comparison of oxygen saturation (SPO2) and FEV1% in non-exercising and exercising young men. * = P < 0.05

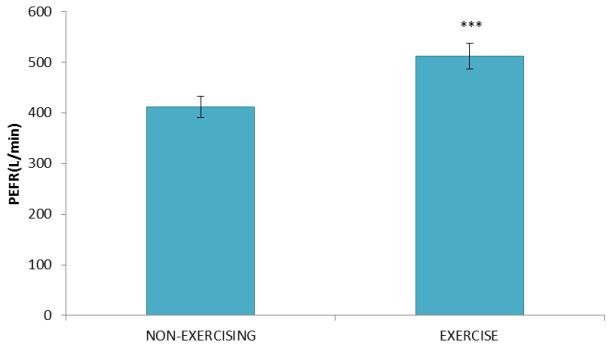



**Figure 3**:
Comparison of peak expiratory flow rate (PEFR) in non-exercising and exercising subjects. *** = P < 0.001

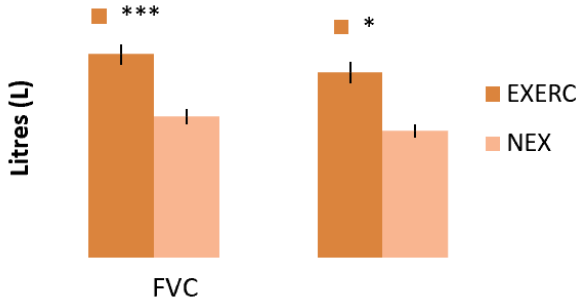



**Figure 2**:
Comparison of forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV1) in non-exercising and exercising subjects. *** = P < 0.001 and * = P < 0.05

**Table 3:**
Comparison of baseline blood pressure in non-exercising and exercising young men

| N= 20 | Mean ± S.E.M | | |
|---|---|---|---|
| **Parameters** | **Non-Exercising Subjects** | **Exercising Subjects** | **Remark** |
| SBP (mmHg) | 107.20 ± 2.06 | 100.50 ± 1.53* | SIG |
| DBP (mmHg) | 70.00 ± 2.84 | 63.50 ± 3.14 | NS |
| PP (mmHg) | 37.20 ± 3.58 | 37.00 ± 2.72 | NS |
| MABP (mmH | 82.40 ± 3.26 | 75.83 ± 2.01 | NS |

*Values are expressed as mean ± S.E.M; SIG =Significant*
*NS =Not significant; *= P < 0.05*
*SBP = systolic blood pressure; DBP = diastolic blood pressure; MABP = mean arterial blood pressure; PP = Pulse pressure*

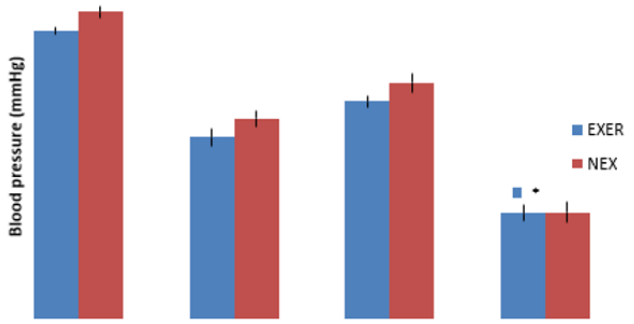



**Figure 4**:
Comparison of blood pressure parameters in non-exercising and exercising subjects; *= P < 0.05

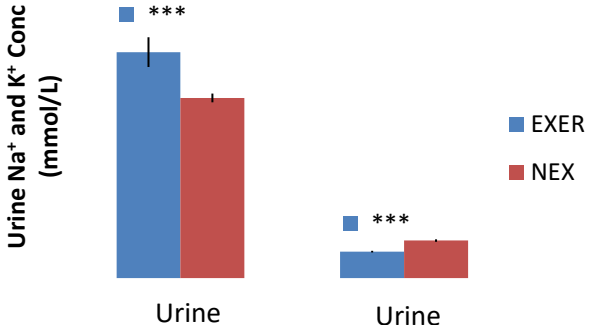



**Figure 5:**
Comparison of urine $Na^+$ and $K^+$ concentration in non-exercising and exercising subjects.
*** = P < 0.001

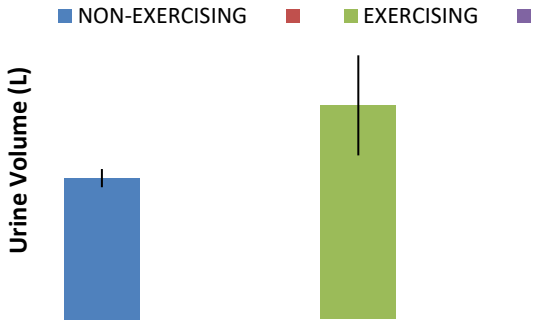



**Figure 6:**
Comparison of urine volume in non-exercising and exercising subjects

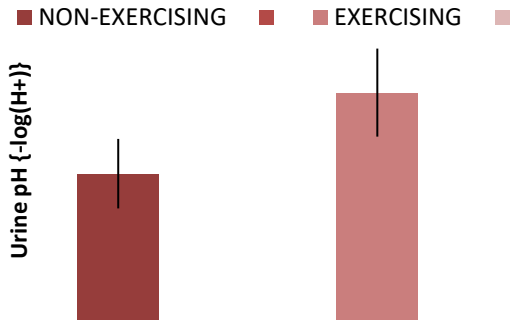



**Figure 7:**
Comparison of urine pH in non –exercising and exercising subjects

**Comparison Of Baseline Renal Function In Non-Exercising And Exercising Subjects:** Urine Na+ and K+ concentration differed significantly in both groups (Fig. 5) with P < 0.001. There was no significant difference in the urine volume and urine pH of both groups (Fig. 6-7).

**Table 5:**
Effect of salt loading on the ventilatory function of non-exercising young men

| **N=20** | **Mean ± S.E.M** | | | |
|---|---|---|---|---|
| **Parameters** | **Before loading** | **After loadir** | **Change ± SEM** | **Remarks** |
| FVC (Litre) | 2.73 ± 0.15 | 2.72 ± 0.16 | -0.003 ± 0.01 | NS |
| FEV1 (Litre) | 2.45 ± 0.13 | 2.40 ± 0.18 | -0.05 ± 0.05 | NS |
| FEV1% | 90.49 ± 2.47 | 88.80 ± 2.36 | -1.69 ± 0.11 | NS |
| PEFR (L/MIN) | 411.40 ± 22.7 | 391.20 ± 29.81 | -20.20 ± 7.11 | NS |
| SPO2 | 95.95 ± 0.55 | 95.10 ± 0.71 | -0.85 ± 0.16 | NS |

Values are expressed as mean ± S.E.M
NS=Not significant
FEV1= forced expiratory volume in 1 sec; FVC = forced vital capacity; PEFR = Peak expirat low rate; SPO2 =oxygen saturation.

**Effects Of Salt Loading on The Ventilatory Function Of Non-Exercising Subjects:** Salt loading has no significant effect on the FEV1, FVC and PEFR of non-exercising subjects (Fig. 9-10), slightly reduced by -0.05 ± 0.05 L, -0.003 ± 0.01L and -20.20 ± 7.11 L/min respectively (Table 5). Similarly, they also have no significant effect on their SPO2 and FEV1% (Fig. 8). Which fell slightly by -0.85 ± 0.16% and -1.69 ± 0.11%.

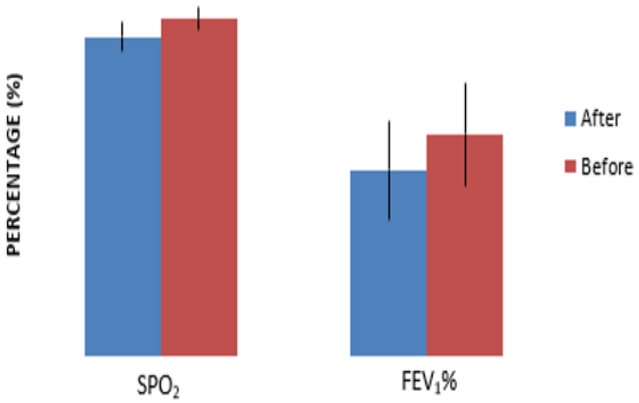



**Figure 8:**
Effect of salt loading on the oxygen saturation (SPO2) and FEV1% in non-exercising subjects

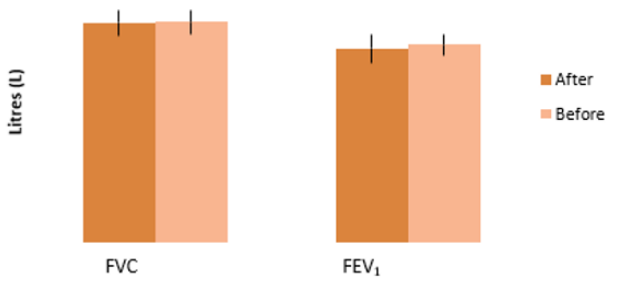



**Figure 9:**
Effect of salt loading on the forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV1) in non-exercising subjects

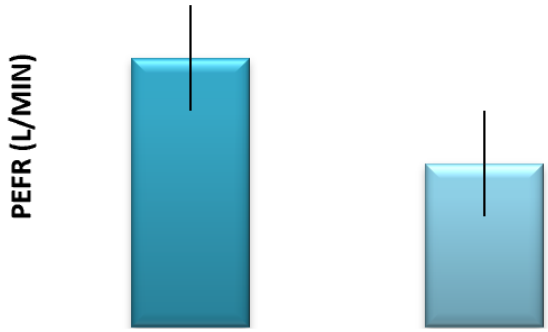



**Figure 10:**
Effect of salt loading on the mean peak expiratory flow rate (PEFR) of non –exercising subjects

**Effect of Salt Loading on the Blood Pressure Parameters of Non-Exercising Subjects:** Salt loading have significant effect on the blood pressure parameters of non-exercising subjects (Fig. 11), causing significant increase ($P < 0.05$ and 0.01 respectively) in the SBP, DBP,MABP and PP of 18.00 ± 2.04 mmHg, 11.90 ±1.52 mmHg, 6.20 ± 0.29 mmHg and 13.97 ± 1.98 mmHg respectively (Table 6) after five days of salt loading.

**Table 6:**
Effect of salt loading on the blood pressure parameters of non-exercising subjects

| Parameters | Before loading | After loading | Change ± SEM | Remark |
|---|---|---|---|---|
| SBP (mmHg) | 107.20 ± 2.06 | 125.30 ± 4.10.* | 18.00 ± 2.04 | SIG |
| DBP (mmHg) | 70.00 ± 2.84 | 81.90 ± 2.66** | 11.90 ± 1.52 | SIG |
| PP (mmHg) | 37.20 ± 3.58 | 43.40 ± 3.87* | 6.20 ± 0.29 | SIG |
| MABP (mmHg) | 82.4 ± 3.26 | 96.37 ± 2.58* | 13.97 ± 1.98 | SIG |

Values are expressed as mean ± S.E.M
SIG=Significant
*= P < 0.05, **= P < 0.01
SBP = systolic blood pressure; DBP = diastolic blood pressure; MABP = mean arterial blood pressure; PP = Pulse pressure

**Effect of Salt Loading on Renal Function Parameters of Non-Exercising Subjects:** Urine K+ concentration increased significantly ($P < 0.05$) by 5.35 ± 0.36 mmol /L, urine Na+ concentration decreased by -18.74 ± 0.28mmol/L after five days of salt loading (Table 7). Salt loading has no significant effect on the urine volume, and urine pH (Fig. 13, 14). This fell slightly by 0.04 ± 0.01L, -18.74 ± 0.28 mmol /L and -0.07 ± 0.06 respectively.

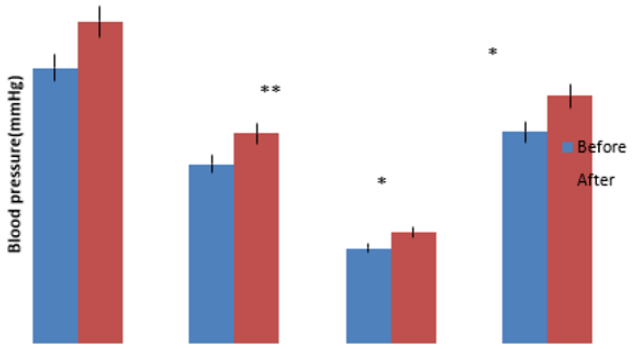



**Figure 11:**
Effect of salt loading on the blood pressure parameters of non-exercising subjects. *= P < 0.05, **= P < 0.01

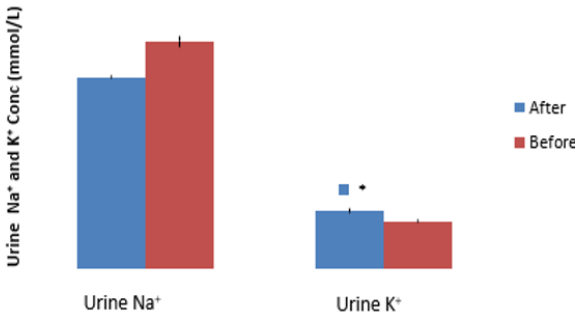



**Figure 12:**
Effect of salt loading on the urine $Na^+$ and $K^+$ Concentration in non-exercising subjects.
*= P < 0.05

**Table 7:**
Effect of salt loading on the renal function of non-exercising young men

| Parameters | Before loading | After loading | Change ± SEM | Remarks |
|---|---|---|---|---|
| Urine $Na^+$ concentration (mmol/L) | 118.71 ± 2.89 | 99.97 ± 3.17* | -18.74 ± 0.28 | SIG |
| Urine $K^+$ concentration (mmol/L) | 24.72 ± 0.83 | 30.07 ± 1.19* | 5.35 ± 0.36 | SIG |
| Urine volume (Litres) | 0.31 ± 0.02 | 0.28 ± 0.01 | -0.04 ± 0.01 | NS |
| Urine pH | 6.23 ± 0.15 | 6.16 ± 0.21 | -0.07 ± 0.06 | NS |

Values are expressed as mean ± S.E.M
SIG =Significant NS =Not significant; *= P < 0.05

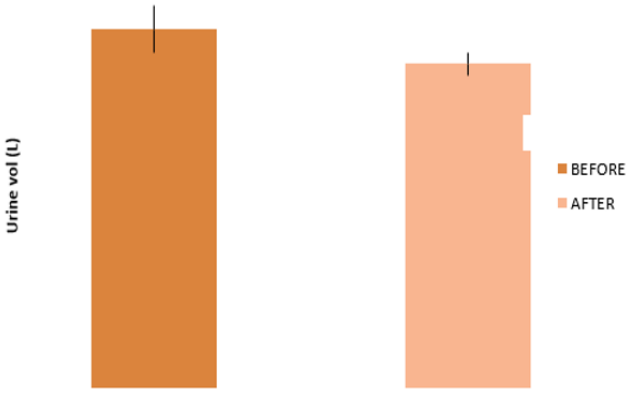



**Figure 13:**
Effect of salt loading on the urine pH of non-exercising subjects

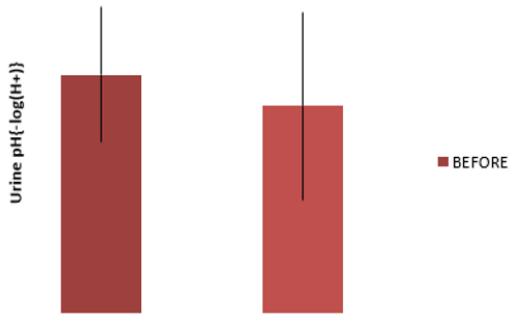



**Figure 14:**
Effect of salt loading on the urine volume of non-exercising subject

**Effects of Salt Loading on The Ventilatory Function of Exercising Subjects:** Exercising subjects experienced significant reduction ($P < 0.05$) in FVC, FEV1 and PEFR (Fig. 16-17) of -0.55 ± 0.04 L, -0.52 ± 0.03 L and -88.58 ± 1.14 L/min respectively (Table 8) after five days of salt loading. Salt loading had no significant effect on the FEV1% and SPO2 (Fig. 15).This fell slightly by -1.17 ± 0.06 % and -1.28 ± 1.06 % respectively.

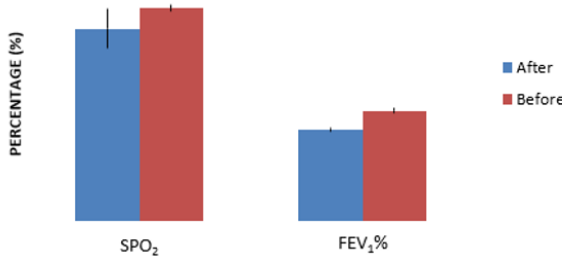



**Figure 15:**
Effect of salt loading on the oxygen saturation and fev1% of the exercising subjects.

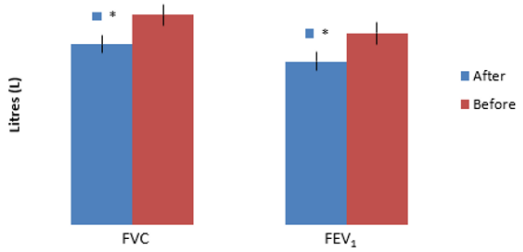



**Figure 16:**
Effect of salt loading on the forced vital capacity (FVC) and forced expiratory volume in 1sec (FEV1) of the exercising subjects
.*= $P < 0.05$.

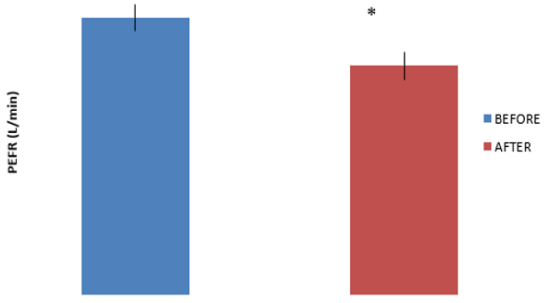



**Figure 17:**
Effect of salt loading on the peak expiratory flow rate (PEFR) of the exercising subjects. *= $P < 0.05$

**Table 8:**
Effect of salt loading on the ventilatory function of exercising subjects

| Parameters | Before loading | After loading | Change ± SEM | Remarks |
|---|---|---|---|---|
| FVC (Litre) | 3.93 ± 0.20 | 3.38 ± 0.16* | -0.55 ± 0.04 | SIG |
| FEV1 (Litre) | 3.58 ± 0.21 | 3.06 ± 0.18* | -0.52 ± 0.03 | SIG |
| FEV1% | 90.94 ± 0.17 | 89.77 ± 0.11 | -1.17 ± 0.06 | NS |
| PEFR (L/MIN) | 511.30 ± 24.83 | 422.72 ± 25.97* | -88.58 ± 1.14 | SIG |
| SPO2 | 97.40 ± 0.19 | 96.12 ± 0.25 | -1.28 ± 0.06 | NS |

Values are expressed as mean ± S.E.M
SIG = Significant .NS =Not significant; *= $P < 0.05$
FEV1= forced expiratory volume in 1 sec; FVC= forced vital capacity; PEFR= Peak expiratory flow rate; SPO2 = oxygen saturation.

**Table 9:**
Effect of salt loading on the blood pressure parameters in exercising subjects.

| Parameters | Before loading | After loading | Change ± S.E.M | Remarks |
|---|---|---|---|---|
| SBP (mmHg) | 100.50 ± 1.53 | 110.00 ± 2.80* | 9.50 ± 0.10 | SIG |
| DBP (mmHg) | 63.50 ± 3.14 | 70.00 ± 3.08 | 6.50 ± 0.06 | NS |
| MABP (mmHg) | 75.83 ± 2.01 | 83.33 ± 3.36 | 7.50 ± 1.35 | NS |
| PP (mmHg) | 37.00 ± 3.33 | 40.00 ± 3.72 | 3.00 ± 0.06 | NS |

Values are expressed as mean ± S.E.M
SIG =Significant NS =Not significant; *= $P < 0.05$
SBP =systolic blood pressure; DBP = diastolic blood pressure; MABP = mean arterial blood pressure; PP = Pulse pressure.

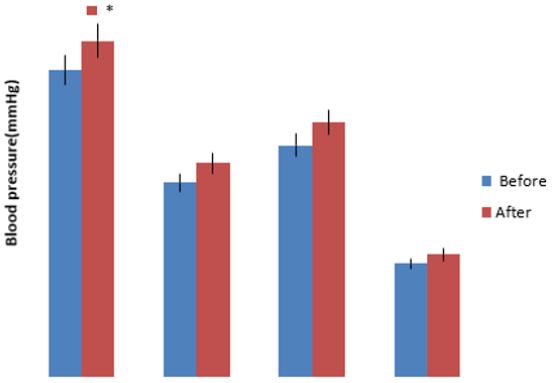



**Figure 18:**
Effect of salt loading on the blood pressure parameters in exercising subjects.
.*= $P < 0.05$.

**Effect of Salt Loading on Blood Pressure Parameters of Exercising Subjects:** The Systolic blood pressure increased significantly ($P < 0.05$) from 100.50 ± 1.53 mmHg to 110.00 ± 2.80 mmHg. (Table 9) after salt loading. This is within the normal range in young adults. There was no significant increase in the Diastolic blood pressure, Mean arterial pressure and Pulse pressure after salt loading in the exercising subjects (Fig. 18). Which decreases slightly by

6.50 ± 0.06 mmHg, 7.50 ± 1.35 mmHg and 3.00 ± 0.06 mmHg respectively.

**Effect of Salt Loading on Renal Function in Exercising Subjects:** Urine Na+ concentration increased significantly ($P < 0.05$) from 148.96 ± 9.80 mmol/L to 160.23 ± 10.97 mmol/L after five days of salt loading (Table 10). Similarly, there was also a decrease in the Urine K+ Concentration, this fell significantly ($P < 0.05$) by -3.73 ± 0.21 mmol/L after five days of salt loading. Salt loading has no significant effect on their urine volume and urine pH. (Fig. 23-24).

**Table 10:**
Effect of salt loading on the renal function in exercising subjects

| Parameters | Before loading | After loading | Change ±S.E.M | Remarks |
|---|---|---|---|---|
| Urine $Na^+$ concentration (mmol/L) | 148.96 ± 9.80 | 160.23 ±10.97* | 11.27 ± 1.17 | SIG |
| Urine $k^+$ concentration (mmol/L) | 17.44 ± 0.58 | 13.71 ± 0.37* | -3.73 ± 0.21 | SIG |
| Urine volume (Litres) | 0.47 ± 0.11 | 0.51 ± 0.10 | 0.01 ± 0.01 | NS |
| Urine pH | 6.58 ± 0.19 | 6.44 ± 0.17 | -0.14 ± 0.03 | NS |

Values are expressed as mean ± S.E.M
SIG =Significant NS =Not significant; *= $P < 0.05$

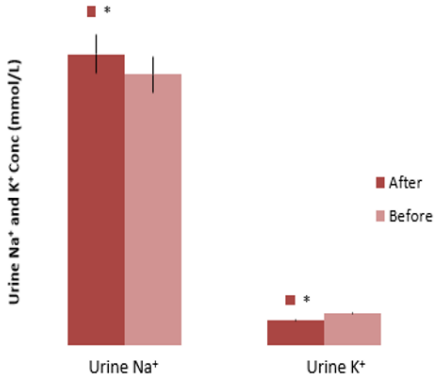



**Figure 19:**
Effect of salt loading on the urine $Na^+$ concentration in exercising subjects; *= $P < 0.05$

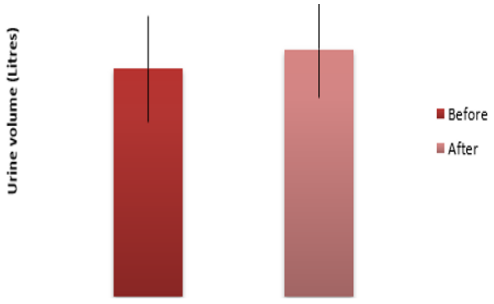



**Figure 20:**
Effect of salt loading on the urine volume of exercising subjects

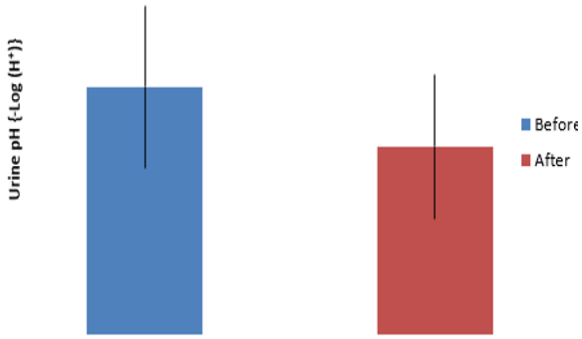



**Figure 21**:
Effect of salt loading on the urine pH of exercising subjects

**Comparison of the Change (Δ) in Ventilatory Function in Non-Exercising and Exercising Subjects after Salt Loading:** Salt loading caused a greater change in FVC ($P < 0.05$), FEV1 ($P < 0.05$) and PEFR ($P < 0.05$) in exercising subjects than the non- exercising subjects (Fig. 24-26) There was no significant change in the FEV1% and SPO2 of both groups.

**Table 11:**
Comparison of the change (Δ) in ventilatory function of non-exercising (control) and exercising subjects after salt loading

| Parameters | Non-Exercising Subjects | Exercising Subjects | Remark |
|---|---|---|---|
| FVC (Litre) | -0.003 ± 0.01 | -0.55 ± 0.04* | SIG |
| FEV1 (Litre) | -0.05 ± 0.05 | -0.52 ± 0.03* | SIG |
| FEV1% | -1.69 ± 0.11 | -1.17 ± 0.06 | NS |
| PEFR (L/MIN) | -20.20 ± 7.11 | -88.58 ± 1.14* | SIG |
| SPO2 (%) | -0.85 ± 0.16 | -1.28 ± 0.06 | NS |

Values are expressed as mean ± S.E.M
SIG =Significant NS =Not significant; *= $P < 0.05$
FEV1= forced expiratory volume in 1 sec; FVC = forced vital capacity; PEFR= Peak expiratory flow rate; SPO2 = oxygen saturation.

**Comparison of The Change (Δ) in Blood Pressure Parameters in Non-Exercising and Exercising Subjects After Salt Loading:** Five days of salt loading caused a greater change in the systolic blood pressure ($P < 0.01$), diastolic blood pressure ($P < 0.01$), mean arterial blood pressure ($P < 0.01$) and the pulse pressure ($P < 0.001$) in the non-exercising subjects than the exercising subjects (Fig. 27, 28). This explains the powerful effect salt loading has on the blood pressure parameters of non-exercising subjects over their exercising counterpart.

**Comparision Of The Change (Δ) In Renal Function In Non- Exercising And Exercising Subjects After Salt Loading:** Salt loading caused a greater change in urine Na+ concentration and volume ($P < 0.001$and 0.05) in exercising subjects than the non- exercising subjects. Similarly, they also caused a greater change in the urine K+ concentration ($P < 0.01$) in non- exercising subject than the exercising subjects (Fig. 29).There was no significant change in the urine pH of both groups (Table 13).

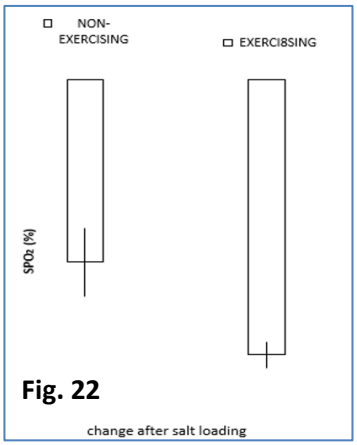


Fig. 22

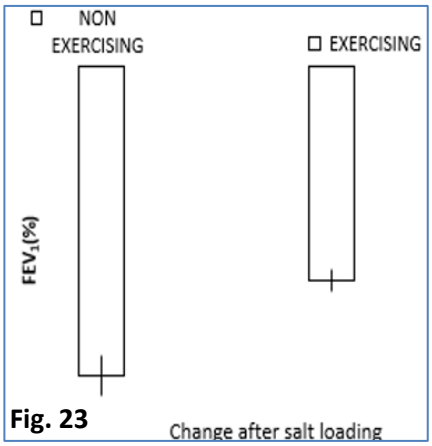


Fig. 23

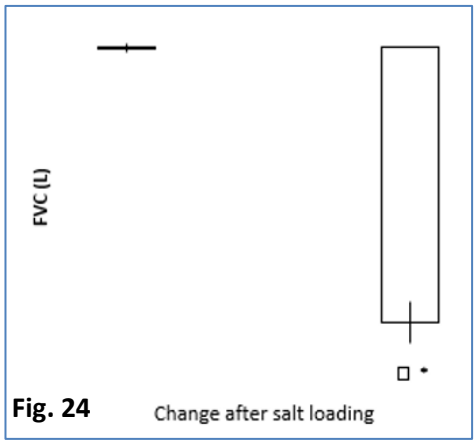


Fig. 24

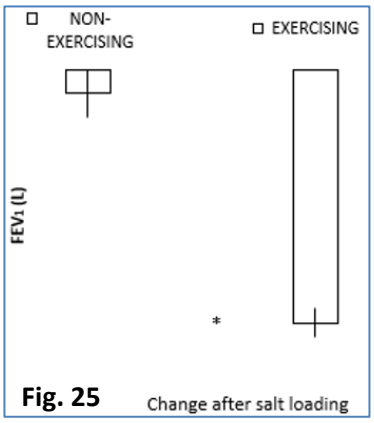


Fig. 25

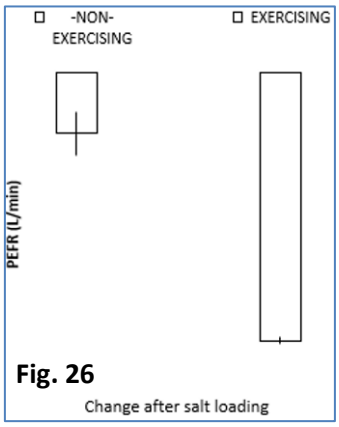


Fig. 26

**Figures 22 – 26**
Changes in oxygen saturation (SPO $_2$, **Figure 22);** FEV1%**, Figure 23),** FVC **(Figure 24),** (FEV1, **Figure 25) and** peak expiratory flow rate, PEFR, **Figure 26**) in non-exercising and exercising young men. *= P < 0.05

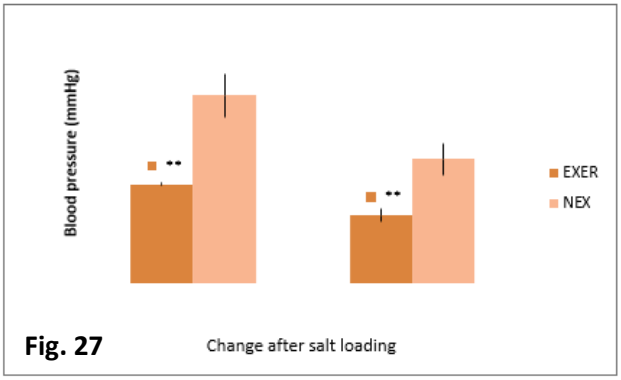


Fig. 27

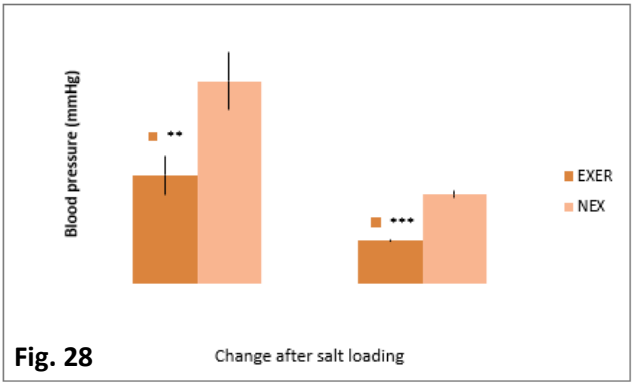


Fig. 28

**Figures 27 and 28**
Changes in systolic and diastolic blood pressure, **Figure 27)** and changes in pulse pressure (PP) and mean arterial pressure (MAP) **Figure 28**) in non-exercising and exercising young men. *= P < 0.05

**Table 12:**
Comparison of the change (Δ) in blood pressure parameters in non-exercising and exercising subjects after salt loading

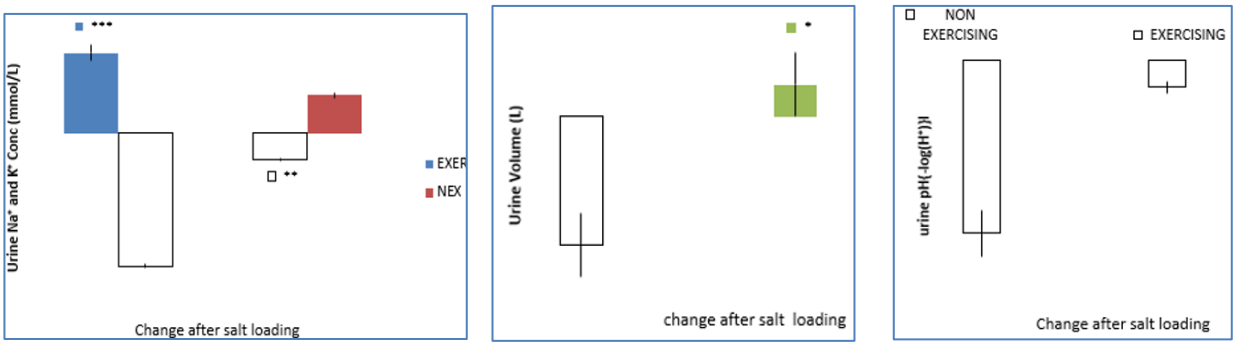
| Parameters | Non-Exercising Subjects | Exercising Subjects | Remarks |
|---|---|---|---|
| SBP (mmHg) | 18.0 ± 2.04 | 9.50 ± 0.10** | SIG |
| DBP (mmHg) | 11.90 ± 1.52 | 6.50 ± 0.60** | SIG |
| MABP (mmHg) | 13.97 ± 1.98 | 7.50 ± 1.35** | SIG |
| PP (mmHg) | 6.20 ± 0.29 | 3.00 ± 0.06*** | SIG |

Values are expressed as mean ± S.E.M
SIG =Significant; **= P < 0.01, *** = P < 0.001 respectively
SBP = systolic blood pressure; DBP = diastolic blood pressure; MABP = mean arterial blood pressure; PP = Pulse pressure

**Table 13:**
Comparison of the change (Δ) in renal function in non-exercising and exercising subjects after salt loading

| Parameters | Non-Exercising Subjects | Exercising Subjects | Remarks |
|---|---|---|---|
| Urine $Na^+$ conc. (mmol/L) | -18.74 ± 0.28 | 11.27 ± 1.17*** | SIG |
| Urine $K^+$ conc. (mmol/L) | 5.35 ± 0.36 | -3.73 ± 0.21** | SIG |
| Urine volume (litre) | -0.04 ± 0.02 | 0.01 ± 0.01* | SIG |
| Urine pH | -0.07 ± 0.06 | -0.14 ± 0.02 | SIG |

Values are expressed in mean ± S.E.M
SIG =Significant
**= P < 0.01, ***= P < 0.001 respectively




**Figures 29 - 31:**
Changes in urine $Na^+$ and $K^+$ concentration (Figure 29), urine volume (Figure 30) and urine pH (Figure 31) in non-exercising and exercising subjects.; ** = P < 0.01, *** = P < 0.001

## DISCUSSION

Several investigators have demonstrated the possible influence of high dietary salt intake on the severity of asthma in asthmatics (Stephanie *et al.,2024)*. Large amount of experimental and clinical studies have also emphasized the critical role of sodium in the regulation of blood pressure and the implication of an abnormal sodium balance in the development of hypertension in humans (Lucia *et al.,2019)*. The aim of this study was to ascertain the effect of high salt intake on the blood pressure, Oxygen saturation, Lung function and renal function of young men who engage more often in exercising activities

In this study, the effect of high salt intake on the blood pressure, lung function, and renal function of young men who regularly exercise was investigated. The results showed that before salt loading, exercising subjects had better lung function and lower blood pressure compared to nonexercising subjects. However, after salt loading, lung function and blood pressure worsened in exercising subjects while remaining unchanged in non-exercising subjects. This suggests that exercise may protect against the adverse effects of high salt intake on lung function and blood pressure. Additionally, salt loading affected renal function differently in exercising and non-exercising subjects, with exercising subjects showing increased urine sodium and potassium excretion compared to non-exercising subjects. The study highlights the complex interplay between salt intake, exercise, and various physiological parameters. Exercising subjects had better FVC values than the nonexercising subjects before a high salt intake. This observation is in line with the work of Prakash *et al*., (2007) who made a similar observation in men engaging in physical exercise. A study by Douglas *et al*., (1959) also made similar findings in men who engage in contact sports like basketball showing a significantly higher forced vital capacity compared to men that don't engage in such sport .Such observation was also reported in Athletes by Onadeko *et al*., (1976), reporting a significantly higher FVC in athletes compared to non-athletes. Also, when nonexercising and exercising groups were compared, result showed higher FEV1 in exercising subjects as opposed to the report of Ayesha *et al*., (2007) that did not observe any significant change in FEV1.

PEFR values were much higher in the exercising subjects than the non-exercising group. This observation is supported by the report of Nagarathna and Nagendia (1985) reporting an improvement in PEFR after two weeks of yoga training . Likewise, FEV1 % and SPO2 were lower in nonexercising subjects than the exercising subjects as supported by the reports of previous studies observing significant difference in FEV1 % and SPO2 in actively exercising men. This might be due to the difference in their pulmonary capacity (Adegoke and arogundade, 2002).

Evidence that increased extracellular osmolality is associated with pro-inflammatory response is provided by studies manipulating serum osmolality using salt loading in rats, in which an increased serum osmolality resulted in an increase in the level of pro-inflammatory cytokines such as interleukin -1beta after rehydration (Summy-long *et al*., 2006). This makes changes in osmolality after salt loading a modifier of the immune system. The possibility of exercise to intensify this effect by stimulating the release of more inflammatory mediators might be the mechanism via which exercise and high salt intake reduces lung function. Five days of salt loading decrease the value of FVC, FEV1 and PEFR more in the exercising subject than the nonexercising subjects, in the absence of any significant change in the SPO2 and FEV1% in both groups, owing to the augmentation of the pro-inflammatory effect of salt loading on ventilatory function by exercise training (Robert *et al*., 2000).

Result obtained from this present study shows the value of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP) were lower in the exercising subjects compared to the higher values in the non-exercising subjects. This may be due to the low fat deposition, elastic arterioles, increased shear stress, increased flow mediated release of nitric oxide (NO) and robust cardiovascular system in the exercising subjects (Hutcheson and Griffith, 1991).

In the exercising subjects five days of salt loading caused significant increase in systolic blood pressure (SBP) with no significant increase on their diastolic blood pressure (DBP), mean arterial pressure (MAP) and pulse pressure (PP) compared to the significant increase in the blood pressure parameters of the non-exercising subjects. This agrees with the confirmation that salt loading elicits a sustained elevation in blood pressure i.e hypertension in humans (Fujita *et al*.,1980) and experimental animals (Elias *et al*.,2004).It also agree with the work of He and Macgregor (2002), who established a dose–response relationship between blood pressure and sodium intake between 50– 200mmol.The possibility of physical activity or exercise to prevent the significant increase in some of

the blood pressure parameters could be as a result of their protective effect to decrease the sensitivity of blood pressure to salt. This occurs through several potential mechanisms, such as reduction of insulin resistance, improvement of endothelial function and inhibition of sympathetic nervous system activity. Physical activity has been shown to be associated with reduced insulin resistance (Gill,2007), and insulin resistance decreases renal sodium excretion, leading to extracellular fluid volume expansion and salt sensitive hypertension (Chen *et al*., 2009). The mechanism by which physical exercise decrease insulin resistance are not yet fully explored (Gill and Malkova, 2006). It has been shown that vigorous physical activity result in physiologic adaptations of the skeletal muscle cells in adults. Some of the physiologic adaptations include increase capillary supply to the skeletal muscles (Hawley,2002), an increase in the activity of enzymes of the mitochondrial electron transport chain, and a concomitant increase in mitochondrial volume and density (Hoppeler and Fluck, 2003). In addition, an increased substrate use (Holloszy and Coyle, 1984) with a decrease of carbohydrate oxidation and an increased muscle glucose transport might play a role (Holloszy, 2005). Indirectly, regular physical exercise may act by increasing lean body mass and concomitantly reducing body fat (Ruiz *et al*,2006).There is substantial evidence that resistance to insulin -mediated glucose disposal in non-diabetic individual is strongly associated with hyperinsulinemia (Hollenbeck and Reaven, 1987;Sun *et al*.,2008).It has also been shown that the ability of insulin given acutely to enhance renal Na+ retention is maintained in individuals who are insulin resistant (Shimamoto *et al*,1994;facchini *et al*.,1999). Consistence with this considerations is the current findings that the natriuretic response to Nacl loading was decreased in proportion to the degree of insulin resistance and presumably to the associated hyperinsulinemia .However, I believe this represents the demonstration that delayed Na+ balance and enhanced Na+ retention occurs in healthy, non-obese, insulin – resistant individuals to cause the change in blood pressure associated with sodium chloride loading. Before salt loading, exercising subjects had a higher urine Na+ concentration and volume than the nonexercising subjects. This could be as a result of their augmented basal and agonist induced nitric oxide release (Pritchard *et al*, 1994) to modulate the excretory response to changes in arterial pressure via the pressure natriuresis and diuresis mechanism. A mechanism that have being demonstrated by separate studies to be reduced by acute (Majid *et al*,1993),short term (guarasci and kline,1996) or long term (Atucha *et al*,1996) inhibition of nitric oxide synthesis .Another possible mechanism that could explain this difference is the inhibition of tubular reabsorption of sodium (Na+) and water by nitric oxide in several segments of the nephron (Garvin and Oritz, 2002),which may be induced by changes in renal interstitial pressure (Garcia and roman, 1989), and intra -renal medullary hemodynamics (Cowley,1992).

Urine Na+ concentration and volume increased significantly in exercising subjects. In non-exercising subjects, salt loading significantly decreased urine Na+ concentration, causing a slight reduction in urine volume. Similar observation was found in Dahl- sensitive rats (DS) and Dahl resistant rats (DR) by Hustel *et al*. (1996), who reported greater Na+ reabsorption by the inner medullary collecting duct cell from Dahl salt sensitive rat (DS) than those from Dahl salt resistant rats (DR).

In conclusion, five days of salt loading in non-exercising and exercising young men. Worsens pulmonary function more in exercising subjects than the non-exercising subjects in the absence of any effect on FEV1% and SPO2. Increase blood pressure significantly in the non-exercising subjects than the exercising subjects. Increase urine Na+ concentration and 12 hours urine volume in exercising subjects compared to the decrease in the non-exercising subjects. Decrease urine k+ and pH in the exercising subjects compared to the increase in the non-exercising subjects.

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