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# *Original Article*

## High Blood Pressure can be Controlled by Reducing Extra Table Salt Intake

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Abstract

Background: This quasi-experimental community trial was done through door-to-door health education to find out effect of reducing extra table salt intake on patients with high blood pressure.

Material and Method: This study was conducted among 4,930 respondents out of 7,474 population (response rate was 65.96%). Respondents had age 18 years or above living in Mohammadpur area of Dhaka city in Bangladesh. Study period was from August 2005 to February 2009. Intervention was for 18 months on 282 (male 69.5% and female 30.5%) respondents with stage-I hypertension. Respondents on trial had no co-morbidity and they were neither aware about their hypertension nor were ever treated for it. The intervention was given person-to-person to quit extra table salt after signing the informed consent form. Follow-up for selected parameters were done after 6, 12 and 18 month of intervention. Data analysis and interpretation were done through SPSS. Result: Mean Blood Pressure of the respondents was found to be 121/78 mmHg. Overall prevalence of hypertension is 20.1% (JNC-7 criteria). After 18m intervention percent reduction of SBP is -7.0% and DBP is -9.9%. Blood pressure of 14.9% (n=42) went up in spite of behavioural risk reduction. Normal blood pressure was found among 7.8% respondents having stage-I hypertension while 17.7% remained at stage-I but their blood pressure is reduced. Multinominal regression analysis showed chi-square value of 25.8 df 13 p=0.018 between use of extra table salt and systolic blood pressure while the value was 28.684 df 11 p= 0.003 for diastolic blood pressure in a -2 Log Likelihood reduced model. At beginning 44% respondents used extra salt while eating. After 6m, 12m and 18m of intervention, extra salt intake was found among 20.6%, 5.0% and 1.8% respondents respectively. Quantitatively extra salt intake reduced from 63±6.5g per week at beginning to 29±4.6g per week after 18m intervention. Change of salt intake was significantly related to change of both SBP (F= 9.688; p=0.000) and DBP (F=6.544; p=0.002). Quality of life was evaluated for both subjective and objective indices. Conclusion: Reversal of hypertension was 56.7% by lifestyle modification and behavioural changes including salt intake reduction. This study confirmed relation of salt with hypertension and also confirmed reduction of blood pressure after reducing salt intake. This study recommended no extra salt intake for patients at risk or with high blood pressure.

Key words: Salt and Hypertension, Prevalence of Hypertension in Bangladesh.

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# Introduction

Sodium chloride retains water to increase water volume causing increased plasma volume resulting increased cardiac output and increased blood pressure. Increased salt intake causes increased secretion of ouobain by adrenal gland. Ouobain regulates the sodium and calcium present in the smooth muscle cells of the arteries by regulating the proteins.

Excess amount of ouobain secretion disrupts cellular sodium and calcium balance.1 Sodium accumulates in arteries, causing protein regulators to bring in more calcium causing hypertension by constricting artery. Excessive salt consumption can cause narrowing of the renal artery resulting restriction of blood flow through it. This results in secretion of the kidney hormones renin and angiotensin. Renin and angiotensin increase pressure on peripheral arteries and cause hypertension.2 In patients with hypertension a rightward shift in the pressure-natriuresis curve occurs meaning that they need to excrete higher sodium load.3 An study found a new storage area for salt within skin in the body and defected process behind this storage cause hypertension.4Salt is also stored in cells and in the interstitium. Jens Titze and colleagues have shown that a high-salt diet in rats leads to the accumulation of salt in the interstitium of the skin. This process is carefully regulated by the macrophages. The researchers found a gene regulator (transcription factor) called TonEBP (tonicity-responsible enhancer binding protein) in those macrophages. TonEBP is activated in these cells in response to high salt and turns on a gene (VEGF-C - vascular endothelial growth factor C) that controls the production of lymphatic blood vessels. With high-salt diet the lymphatic vessels increase. The researchers found that when these macrophages are depleted or if the receptor for VEGF-C is absent, the animals are not able to store their salt and become hypertensive. Effect of salt on hypertension has been documented in several articles.5,6 This study was designed to find out extra salt intake habit among patients with hypertension and also to analyze effect of reduction of extra table salt intake on high blood pressure.

# Materials and Method

This community trial was conducted from August 2005 to February 2009 with an intervention period of 18 months at urban Mohammadpur area of Dhaka City in Bangladesh. Adults with 18 years age or above as recorded in voter list were the population comprising 7,474 persons. Data collection was done among 4,930 respondents comprising a response rate of 65.96%. Cluster randomized sampling7 was done to collect 282 adults, 196 (69.5%) male and 86 (30.5%) female, with stage-I hypertension without any co-morbidity and treatment. Informed consent was taken for intervention adopting WHO-MONICA protocol. One-to-one counselling was done for avoidance of extra table salt in of 3-tier model: home based individual counselling, centre-based individual counselling and center based peer-group interactive group-counselling sessions. Follow-up for selected parameters was done every 6, 12, and 18 month. Quality of life was measured using the GHQ-28, PHQ-9 and SF-36 questionnaire.8,9 Questionnaire was checked for completeness, consistency, mutually exclusiveness, exhaustion, reliability and validity. Equipments for anthropometric measurement or clinical examination were 3M Littmann Classic II SE (USA) Stethoscope, ALPK2 Mercurial Sphygmomanometer, Height-length Measuring stadiometer, Omron Digital Weighing Scale [Model HN-280], Fukuda C 100 ECG machine and Glucometer etc. Measurement of Blood Pressure was done at home-setting as per standard protocol using proper machine with appropriate cuff size.10-14 Salt intake was measured semi quantitatively by using premeasured spoons distributed to respondents to use salt both for eating at table and also for cooking. Seven days recall method was used in this study.

Study or intervention end-points meant death, drop-out or migration of study subject. No incidence of death happened during study period. Evaluation was a factorial design to monitor and test statistically the outcome of interest i.e. change of blood pressure and reduction of salt intake. Data analysis and interpretation were done using SPSS programme version 17.0 for windows.

## Result

Overall prevalence of hypertension was 20.1% (JNC-7 criteria). After 18m intervention, sBP/dBP reduction was -9.1/-8.4 mmHg (Fig-I). Mean systolic blood pressure of the cases with stage-I hypertension was 137.9 ? 11.7 mmHg and became 127.1 ± 9.5mmHg after 18month intervention (Table-I). Mean diastolic blood pressure was 91.2 ? 4.8 mmHg at baseline but became 82.3 ± 4.6 after 18m intervention.

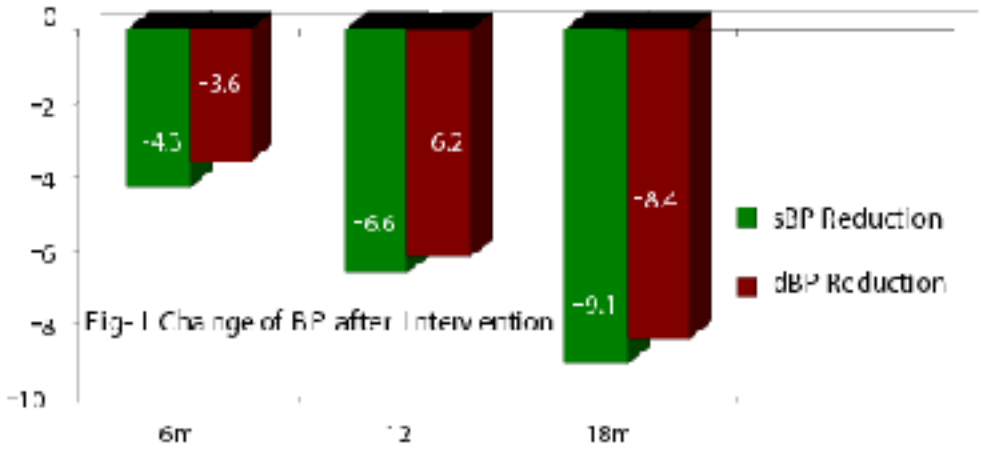


Fig- I Change of BP after Intervention

**Table-I** Mean Blood Pressure during Intervention

| | Systolic | | Diastolic | |
|---|---|---|---|---|
| | Baseline | After 18 m | Baseline | After 18 m |
| **Sample** | 282 | 210 | 282 | 210 |
| **Mean BP (mmHg)** | 137.9 ±11.7 | 127.1 ± 9.5 | 91.2 ± 4.8 | 82.3 ± 4.6 |
| **Percent Reduction** | | 7.0% | | 9.9% |
| **Paired t-test value** | t=22.9 df 209 p<0.001 | | t=26.5 df 209 p<0.001 | |

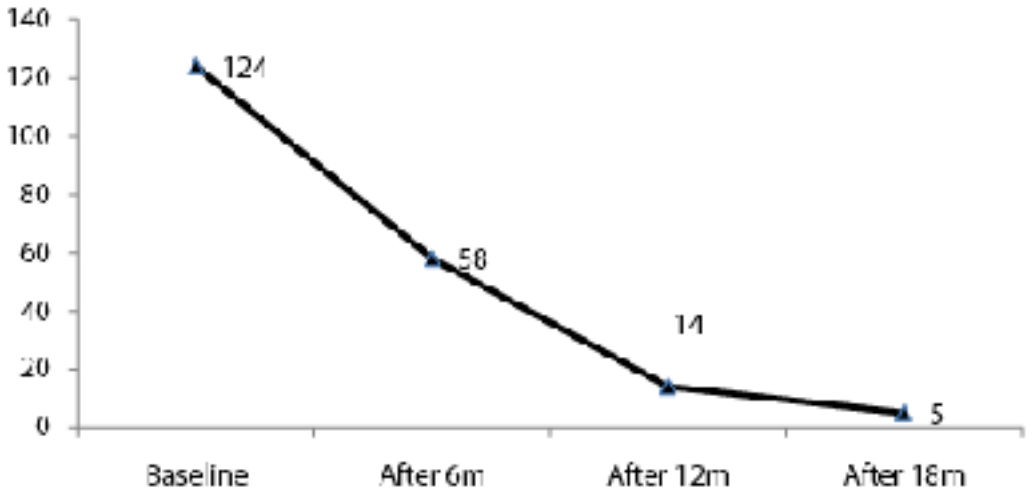
Blood pressure reversed to normal in 7.8% cases and to pre-hypertension level in 48.9% cases. BP remained at stage-I in 17.7% cases but was reduced from the baseline level (Table-II). Blood pressure of 14.9% (n=42) cases went up in spite of behavioural risk reduction and required pharmacological intervention.

**Table-II Outcome of Intervention : Stage-I Hypertension respondents (n=282)**

| Outcome | Reversed to Normal BP | Reversed to Pre-HTN | Remained Stage-I HTN | Drop out | | Total |
|---|---|---|---|---|---|---|
| | | | | Went up Stage II HTN | Other causes | |
| Baseline | | | 282 | | | |
| After 06 months | 14 (5%) | 128 (45.4%) | 116 (41.1%) | 8 (2.8%) | 16 (5.7%) | 282 (100%) |
| After 12 months | 20 (7.7%) | 132 (51.2%) | 72 (27.9%) | 26 (10.1%) | 8 (3.1%) | 258 (100%) |
| After 18 months | 22 (9.8%) | 138 (61.6%) | 50 (22.3%) | 8 (3.6%) | 6 (2.7%) | 224 (100%) |
| End Result | 22 (7.8%) | 138 (48.9%) | 50 (17.7%) | 42 (14.9%) | 30 (10.7%) | 282 (100%) |

Extra table salt was taken by 44% respondents with stage-I hypertension at the beginning of the study amounting on average to about 63±6.5g per week (meaning 7g per day) quantified with pre-measured spoon. Extra table salt intake was reduced to 29±4.6g per week after 18m intervention. Salt intake behaviour was significantly reduced after the intervention. After 6m, 12m and 18m intervention salt intake was found among 58 (20.6%), 14 (5.0%) and 05(1.8%) respondents respectively (Fig-2).

**Fig-2 Change of number of respondents with HTNtaking extra salt (n=282)**




Change of salt intake significantly related to change of both sBP (F=9.688; p=0.000; adjusted r2=0.077) and dBP (F=6.544; p=0.002; r2=0.050) (Table-IV).

**Table-IV GLM Test between change of salt intake and BP Change at 18 month**

| Statistic | Type 11 1 S urn of Squares | df | Mean Square | F Value | P, |
|---|---|---|---|---|---|
| Change of Salt Use Vs. Change of sBP | 591-175 | 2 | 295,588 | 9.688 | .000 |
| Change of Salt Use Vs. Chan le of dBP | 263-8461 | 2 | 131.930 | 6.544 | .02 |

r`= 0.086 (Adjusted 0-077) for sBP and 0-069 (Adlusted = 0.050) for dBP

**Dependent Variable: Change of BP at 18m**

Multiple regression analysis was done for testing role of change of salt intake, after removing the effect of other variables. For systolic BP reduction of salt intake was found to be the best predictor (Beta =0.273, t= 4.148, p=0.000) (Table-V).

**Table-V Predictor co-efficient and significance for BP at 18m**

| **Systolic Blood Pressure Predictors** | | | | |
|---|---|---|---|---|
| Sl. No. | Description | Beta | t | p |
| 1. | Salt reduction | 0.273 | 4.148 | 0.000 |
| 2. | Activity increment | 0.179 | 2.702 | 0.007 |
| 3. | Weight Reduction | 0.126 | 1.860 | 0.064 |
| 4. | Smoking Reduction | 0.009 | 0.124 | 0.902 |
| Diastolic Blood Pressure Predictors | | | | |
| 1. | Salt reduction | 0.173 | 2.462 | 015 |
| 2. | Weight Reduction | 0.144 | 2.038 | 0.043 |
| 3. | Activity increment | 0.138 | 1.982 | 0.049 |
| 4. | Smoking Reduction | 0.025 | 0.340 | 0.735 |

## Discussion

This was a multi-variable intervention study focusing life style modification and behavioural changes. Impact of other variables was also important confounder for study. Percentage contribution of salt use was very difficult to isolate. Correlation of salt with hypertension was tested at beginning of the study. Relationship between use of extra table salt and blood pressure was tested with multinominal regression analysis showing statistically significant association. The chi-square statistic obtained was the difference in -2 log-likelihoods between the final model and a reduced model. This reduced model was equivalent to the final model because omitting the effect did not increase the degrees of freedom. The reduced model was formed by omitting an effect from the final model. The null hypothesis was that all parameters of that effect were zero. Counselling was done to reduce intake of extra table salt. At the end of 18 month salt intake was found to be reduced both in quantity and also frequency. This reduction in salt intake was again tested for correlation with the change of blood pressure. Positive correlation was observed again. This observation strongly indicated role of salt intake as causation of hypertension and confirms the role for reducing blood pressure when

Similar observation was reported also in other study reports.15,16 This study also statistically proved that reduction of salt intake could significantly reduce both systolic and diastolic blood pressure. Salt was also found to be the best predictor for occurrence and reduction of both systolic and diastolic blood pressure.

Extra salt intake was reduced from 44% at beginning of intervention to 1.8% at the end of 18 month intervention. This change of reduced salt intake significantly influenced change of both systolic and diastolic blood pressure. Multinominal regression analysis showed chi-square value of 25.8 df 13 p=0.018 between use of extra table salt and systolic blood pressure while the value is 28.684 df 11 p= 0.003for diastolic blood pressure (Table-III) in a -2 Log Likelihood reduced model. The chi-square statistic was the difference in -2 log-likelihoods between the final model and a reduced model. This reduced model was equivalent to the final model because omitting the effect did not increase the degrees of freedom.

When tested in reduced model by multiple regression analysis, salt reduction was found to be the best predictor (t=4.148; p=0.000 for sBP and t=2.462; p=0.015 for dBP) for reducing both systolic and diastolic blood pressure compared to other behavioural determinants. Salt reduction contributes more for systolic blood pressure reduction than comparable diastolic blood pressure reduction. In brief, salt intake is found significantly associated with causation of blood pressure and also reduction of salt intake significantly contributes to reversal of blood pressure.

## Conclusion

Reversal of hypertension was 56.7% by combined impact of lifestyle and behavioural changes including salt intake reduction. But individually salt intake reduction was found to be more contributory than any other individual risk factors under study. This study confirms relation of salt with hypertension and also confirms reduction of blood pressure after reducing salt intake. This study recommends no extra salt intake for patients with high blood pressure.

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