

## Effect of lo-salt as alternative salt on blood pressure, serum glucose and serum cholesterol

Ismael Hasan Mohammed  
Koya university  
Faculty of Science and health  
Department of Medical Microbiology

**Key words:** lo-salt ,blood pressure ,glucose ,cholesterol.

### Abstract

Salt substitutes are low-sodium table salt alternatives marketed to circumvent the risk of high blood pressure and cardiovascular problems .The aim was to compare the effect of a low-salt ( 1gm ) and prepared salt (1gm) on blood pressure and the level of serum glucose and total cholesterol with table-salt (1gm). The study included using of manufactured lo-salt (contains 66% min of potassium chloride, 33.3% max of sodium chloride and magnesium carbonate as anti-caking agent ), Prepared Lo-Salt ( mixture contains same ingredients but recently prepared in laboratory ) and table – salt ( contains sodium chloride 100% ). Results showed that administration of prepared salt and lo-salt did not change significantly the blood pressure and glucose and cholesterol in the blood after one and two hours from salt intake . In contrast the administration of table - salt (sodium chloride) showed significant increasing in blood pressure after one and two hours from salt intake. These findings suggest the benefits of lo-salt (manufactured and prepared) on blood pressure and glucose and cholesterol in the blood giving a green sign for using it safely.

## Introduction

Hypertension is the condition of having high blood pressure. Normal blood pressure is 120/80. The upper limit of normal blood pressure is about 140 over 90, when systolic blood pressure is above 140mm Hg or when diastolic blood pressure is above 90mm Hg, blood pressure is considered high. Hypertension may be caused by a variety of reasons such as: heredity, your genes, high salt in your diet, not being active, obesity, excessive alcohol intake and/or low potassium in your diet (1).

If the blood pressure permanently high, the body's organs and tissues can show greater signs of wear and tear over the years and, for example, give rise to a stroke or heart disease. At 60 years old, three in every ten will have raised blood pressure. At 70 there are more - two in every five. When such a person restricts their salt intake, their blood pressure falls, sometimes enough to avoid the need for blood-pressure-lowering medicines altogether. A high salt intake causes the body to temporarily retain fluid; the problem could be relieved by reducing salt intake. Some research suggests that increasing potassium intake may also help to get rid of some sodium - providing another bonus (2).

Salt (sodium chloride) is a vital component of our diet both for health and for its flavouring effect. In appropriate quantities it is needed for transmission of nerve impulses and contraction of muscles, maintaining water balance, maintaining acid-base balance, absorption and transport of some nutrients. Although in excessive amounts it is associated with heart disease, high blood pressure and strokes. The Dietary Reference Intake (DRI) for sodium is 1,500 mg/day for people 19-50 years of age, 1,300 mg/day for people 51-70 years of age, and 1,200 mg/day for people >70 years of age. The Upper Level for sodium intake for adults is 2,300 mg/day. The Dietary Guidelines for Americans recommends people consume less than 2,300 mg sodium (approximately 1 teaspoon of salt) per day. On average, most adults consume significantly more — 4,000 to 6,000 mg of sodium daily. While the Nutrition Facts Panel on food labels which lists the number of milligrams of sodium per serving of food suggest: 5 mg or less sodium per serving in free Sodium (3). In experimental hypertension, the endothelium-dependent relaxation of vascular smooth muscle is impaired (4,5). Moreover, high sodium chloride diet and severe hypertension in the stroke-prone SHR induce injury to endothelial cells (6), and a high potassium diet protects against the salt-induced endothelial dysfunction (7, 8, 9).

Potassium chloride (KCl) is a metal halide salt composed of potassium and chlorine. In its pure state it is odorless. It has a white or colorless vitreous crystal, with a crystal structure that cleaves easily in three directions (10). It can be used as a salt substitute for food, but due to its weak, bitter, unsalted flavor, it is usually mixed with regular salt (sodium chloride), for this purpose to improve the taste, the addition of 1 PPM of thaumatin considerably reduces this bitterness (11). Medically it is used in the treatment of hypokalemia and associated conditions, for digitalis poisoning, and as an electrolyte replenisher (12). Potassium intake has also been shown to diminish blood pressure in hypertensive humans and rats, thus reducing the incidence of stroke and stroke-related death and preventing cardiac hypertrophy, mesenteric vascular damage, and renal injury (13). Potassium citrate (the potassium naturally found in fruits and vegetables) can be prescribed as an alternative to potassium chloride (14). Magnesium Carbonate is one of lo-salt component serves as an anticaking agent which is an additive placed in powdered or granulated materials, such as table salt, to prevent the formation of lumps, easing packaging, transport, and consumption. Pure bulk magnesium carbonate is a white powder that is odorless, tasteless, freely soluble in water, very slightly soluble in alcohol and insoluble in ether (15).

Magnesium is closely involved in maintaining cellular ionic balance through its association with sodium, potassium, and calcium (16). Additionally, Magnesium is also important to heart function. People who do not have enough magnesium manifest noticeable changes in the walls of their arteries and capillaries, which choke and compress in the absence of the mineral. This can result to increased blood pressure which could later develop as a full blown cardiovascular disease. As such, supplements of Magnesium Carbonate can prevent hypertension and heart disease (17, 18). The aim of this research is evaluate the extent of the effect of salt product that called lo-salt on blood pressure as well as glucose and cholesterol in the blood.

## Materials and methods

### Experimental design:

The study included 45 hypertensive female and 45 hypertensive male ,divided into 6 groups; 3 groups were females, each group had 15 patients and 3 groups were males also each group had 15 males. 1gm of each salt had been used ( in each serving for 7 days) by oral administration after dissolving it in 10 ml of distilled water and administrated to each group. Blood pressure had been measured just before administration of salt ( zero time ) , also one and two hours after administration. Serum glucose and serum cholesterol by using the kit. The administration had been done after starving about 12 hours.

The study included using of following salts:

#### 1- Prepared Lo-Salt as a mixture contains:

- Potassium chloride (66% of mixture), (KCl, M.W 74.56) manufactured by Thomas baker (chemicals) PVT.limited 4/86. BHARAT MAHAL.MARINE DRIVE, MUMBAI 400 002, INDIA.
- Sodium chloride (33.3% of mixture), (NaCl, M.W 58.443) manufactured by Thomas baker (chemicals) PVT.limited 4/86. BHARAT MAHAL.MARINE DRIVE, MUMBAI 400 002, INDIA.
- Magnesium carbonate (0.7%of mixture), (MgCO<sub>3</sub>, M.W 84.3139) manufactured by Thomas baker (chemicals) PVT.limited 4/86. BHARAT MAHAL.MARINE DRIVE, MUMBAI 400 002, INDIA. It used as anti-caking agent.

#### 2 – LoSalt :

Manufactured by KLINGE,East Kilbride.Scotland G750GX .Lo- Salt called Reduced Sodium Salt Alternative. It contains four Ingredients: Potassium Chloride (66%min) , Sodium Chloride (33.3%max), anti- caking agent (Magnesium Carbonate).

#### 3 – Table salt ( Sodium chloride ) :

The salt without addition of iodine were used in this study

### Statistical analysis:

T –test at  $p < 0.1$  used for statistical significant differences.

### Results and Discussion

Results in table 1 and figures 1,2,3 that related to the males and table 2 and figures 3,4,5 that related to the females showed there is no significant change in the administration of prepared salt and lo-salt in systolic, diastolic and mean arterial blood pressure after one and two hours from salt intake . In contrast the administration of table salt (sodium chloride) showed significant increase in systolic and diastolic and mean arterial blood pressure after one and two hours from salt intake.

The mechanism by which table salt (Sodium chloride) intake made increasing of blood pressure might be related to that Sodium Chloride is hypertonic & will draw extra fluid into the blood. If there is too much sodium within the blood, the body will retain water (fluids), hence increasing blood pressure, leading to edema, shortness of breath, & potential for heart bomb (6) . In fact, several investigators have noted that high dietary salt raises plasma  $[Na^+]$  by just a few millimolars but has no measureable effect on plasma volume (19, 21), possibly because plasma volume estimation errors are larger than the anticipated volume changes. The rise in plasma  $[Na^+]$  is, however, similar in normotensive and hypertensive, or salt-resistant and salt-sensitive, humans as well as in animals (21).

No changing in blood pressure which showed in prepared salt and lo-salt intake may be related to beneficial effect of potassium which decreases vascular responsiveness to vasopressors, such as norepinephrine (22). It is claimed that the use of LoSalt reduces sodium chloride intake. Also magnesium carbonate which serves as an anticaking agent can prevent hypertension through inhibiting muscle contractions including muscle of blood vessels (18).

Regarding serum glucose (table3) and serum cholesterol (table 4) the results showed no significant changes which agree with multiple reports (17).

**Table 1: Effect of salts (table salt , prepared salt and lo-salt) on the blood pressure in the males**

Type of salt	Time of BP measurement	Systolic Blood Pressure(mmHg)	Diastolic Blood Pressure(mmHg)	Mean Arterial Pressure (MAP) (mmHg)
<b>Table salt</b> average age of males = 47.5 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	149 ± 3.8	87 ± 1.5	107.6 ± 2.0
	BP after 1 hour from salt intake (after 1 hr)	166 ± 3.8 *	100± 1.4 *	122 ± 2.0 *
	BP after 2 hour from salt intake (after 2 hrs)	165± 4.1 *	96± 1.3 *	119 ± 2.03 *
<b>Prepared salt</b> average age of males = 47.4 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	156 ± 3.0	95 ± 2.2	115.3 ± 2.2
	BP after 1 hour from salt intake (after 1 hr)	154 ± 3.0	93± 1.6	113.3 ± 1.8
	BP after 2 hour from salt intake (after 2 hrs)	155± 3.1	93± 5.8	113.6 ± 4.5
<b>Lo-salt</b> average age of males = 48.8 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	151 ± 2.5	94± 2.0	113 ± 1.9
	BP after 1 hour from salt intake (after 1 hr)	150 ± 1.9	92± 1.4	111.4 ± 1.4
	BP after 2 hour from salt intake (after 2 hrs)	150 ± 2.0	91± 1.4	111 ± 1.4

T-test at  $\rho < 0.1$ 

Each group consists of 15 patients

\* average is significant different from the average in 0.0 time

**Table 2: Effect of salts (table salt, prepared salt and lo-salt) on the blood pressure in the females.**

Type of salt	Time of BP measurement	Systolic Blood Pressure(mmHg)	Diastolic Blood Pressure(mmHg)	Mean Arterial Pressure (MAP) (mmHg)
<b>Table salt</b> average age of males = 47.5 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	155 ± 4.9	92 ± 2.0	113 ± 2.7
	BP after 1 hour from salt intake (after 1 hr)	172 ± 4.3 *	100 ± 1.8 *	124±2.4 *
	BP after 2 hour from salt intake (after 2 hrs)	172 ± 4.6 *	99± 1.5 *	123.3 ±2.3 *
<b>Prepared salt</b> average age of males = 47.4 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	151 ± 4.6	86 ± 1.9	107.6 ± 2.5
	BP after 1 hour from salt intake (after 1 hr)	149 ± 5.0	88± 2.2	108.3 ± 2.8
	BP after 2 hour from salt intake (after 2 hrs)	147 ± 4.4	86± 1.3	106.3 ± 2.1
<b>Lo-salt</b> average age of males = 48.8 average age of females = 49.9	BP directly before salt intake ( 0.0 time)	153 ± 3.2	96 ± 2.2	115 ± 2.3
	BP after 1 hour from salt intake (after 1 hr)	152 ±3.3	95± 1.9	114 ± 2.1
	BP after 2 hour from salt intake (after 2 hrs)	151 ± 3.1	92± 1.3	111.6 ± 1.7

T –test at  $\rho < 0.1$

each group consists of 15 patients

\* average is significant different from the average in 0.0 time

**Table 3: Averages of serum glucose (mg/dl) of experimented groups**

Type of salt	Time of measurement	Male	Female
<b>Table salt</b> average age of males = 47.5 average age of females = 49.9	serum glucose directly before salt intake ( 0.0 time)	105± 4.7	115± 5.5
	serum glucose after 1 hour from salt intake (after 1 hr)	108± 4.9	117± 5.8
	serum glucose after 2 hour from salt intake (after 2 hrs)	103± 4.2	111± 5.1
<b>Prepared salt</b> average age of males = 47.4 average age of females = 49.9	serum glucose directly before salt intake ( 0.0 time)	123± 6.1	118± 4.9
	serum glucose after 1 hour from salt intake (after 1 hr)	127± 6.7	124± 5.5
	serum glucose after 2 hours from salt intake (after 2 hrs)	120± 6.3	120± 5.8
<b>Lo-salt</b> average age of males = 48.8 average age of females = 49.9	serum glucose directly before salt intake ( 0.0 time)	129± 4.9	114± 6.3
	serum glucose after 1 hour from salt intake (after 1 hr)	132± 5.1	118± 6.4
	serum glucose after 2 hours from salt intake (after 2 hrs)	130± 5.0	113± 5.3

T-test at  $\rho < 0.1$ 

Each group consists of 15 patients

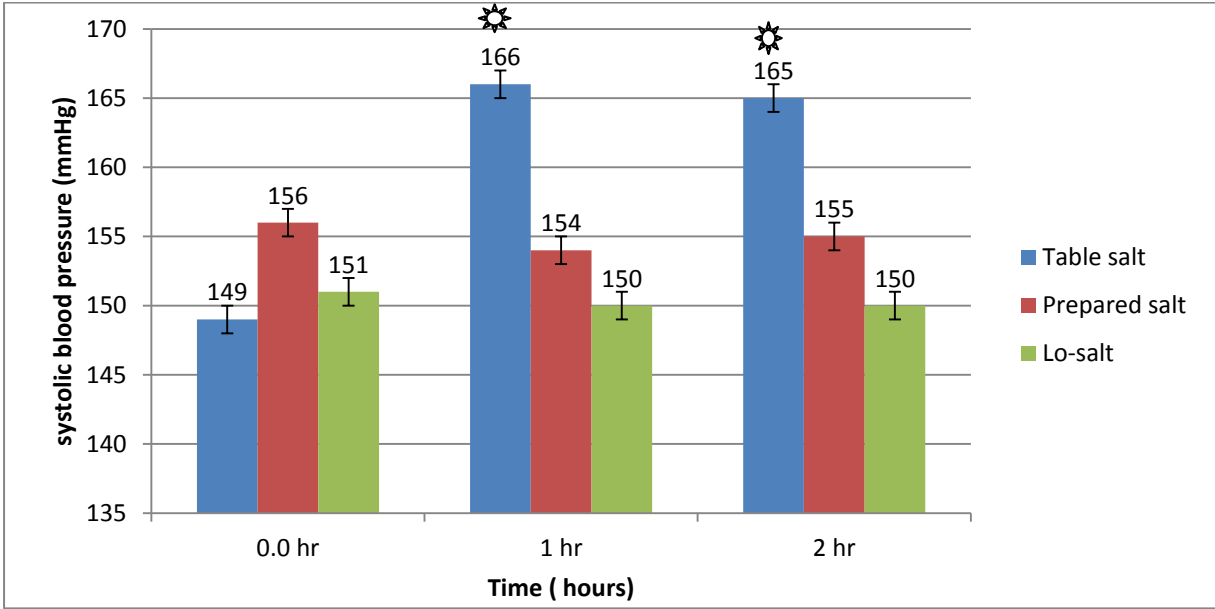


**Table 4: Averages of serum cholesterol (mg/dl) of experimented groups**

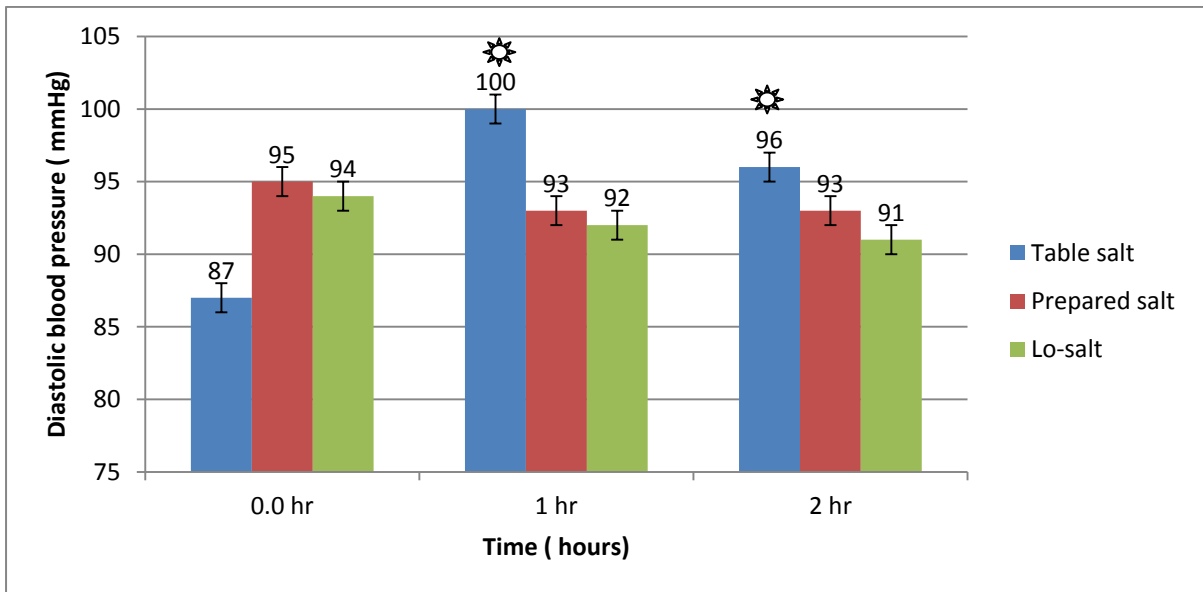
Type of salt	Time of measurement	Male	Female
<b>Table salt</b> average age of males = 47.5 average age of females = 49.9	serum cholesterol directly before salt intake ( 0.0 time)	223± 8.8	234± 7.5
	serum cholesterol after 1 hour from salt intake (after 1 hr)	225± 8.9	235± 7.9
	serum cholesterol after 2 hour from salt intake (after 2 hrs)	221± 8.6	231± 7.3
<b>Prepared salt</b> average age of males = 47.4 average age of females = 49.9	serum cholesterol directly before salt intake ( 0.0 time)	233± 11.4	224± 8.1
	serum cholesterol after 1 hour from salt intake (after 1 hr)	234± 11.2	223± 8.6
	serum cholesterol after 2 hours from salt intake (after 2 hrs)	230± 10.6	220± 8.0
<b>Lo-salt</b> average age of males = 48.8 average age of females = 49.9	serum cholesterol glucose directly before salt intake ( 0.0 time)	227± 9.8	227± 8.9
	serum cholesterol after 1 hour from salt intake (after 1 hr)	225± 9.3	225± 8.5
	serum cholesterol after 2 hours from salt intake (after 2 hrs)	223± 9.1	225± 8.6

T –test at  $\rho < 0.1$ 

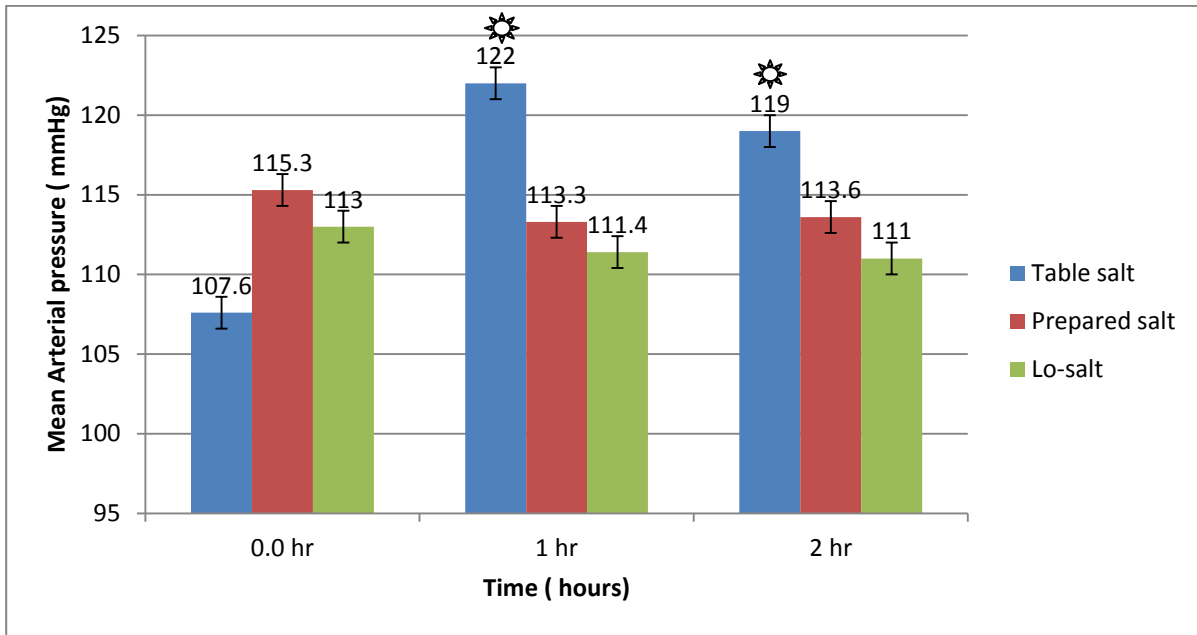
Each group consists of 15 patients



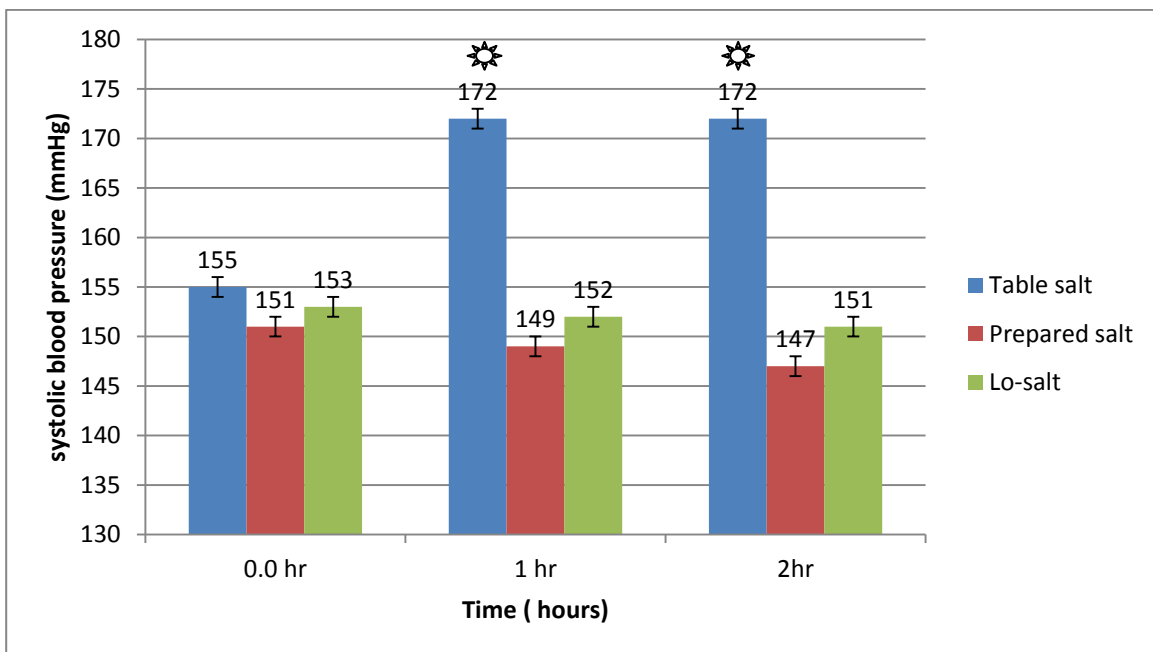
**Figure 1:**  
Effect of three types of salts on the systolic blood pressure in the males



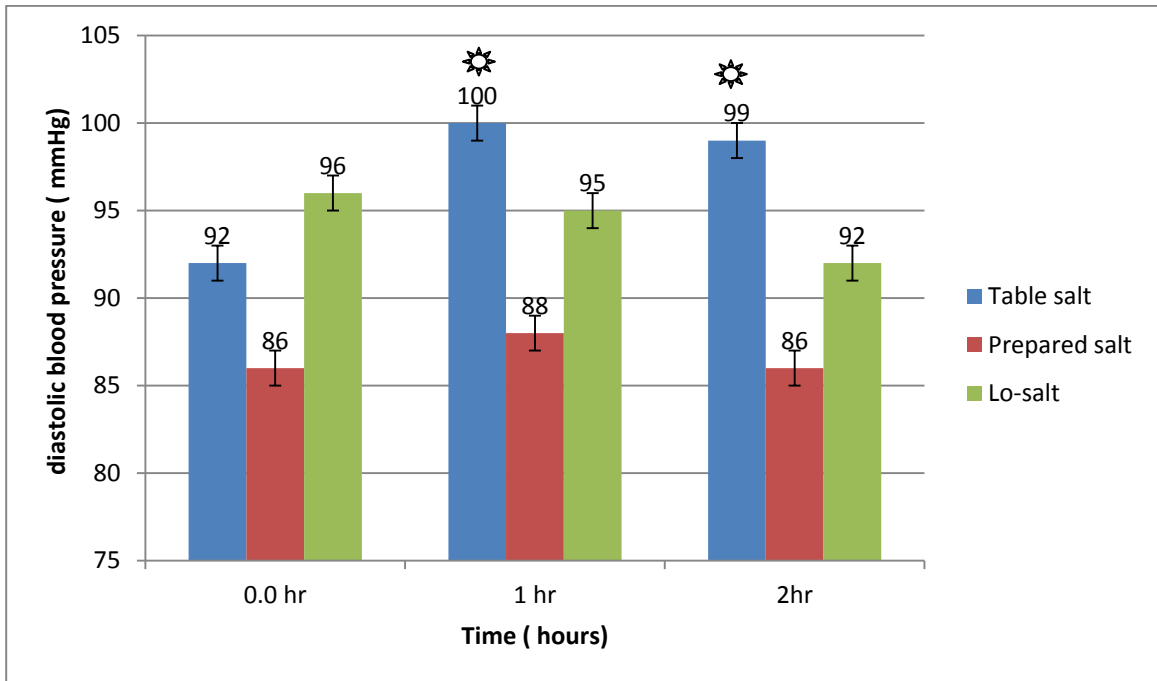
**Figure 2:**  
Effect of three types of salts on the diastolic blood pressure in the males



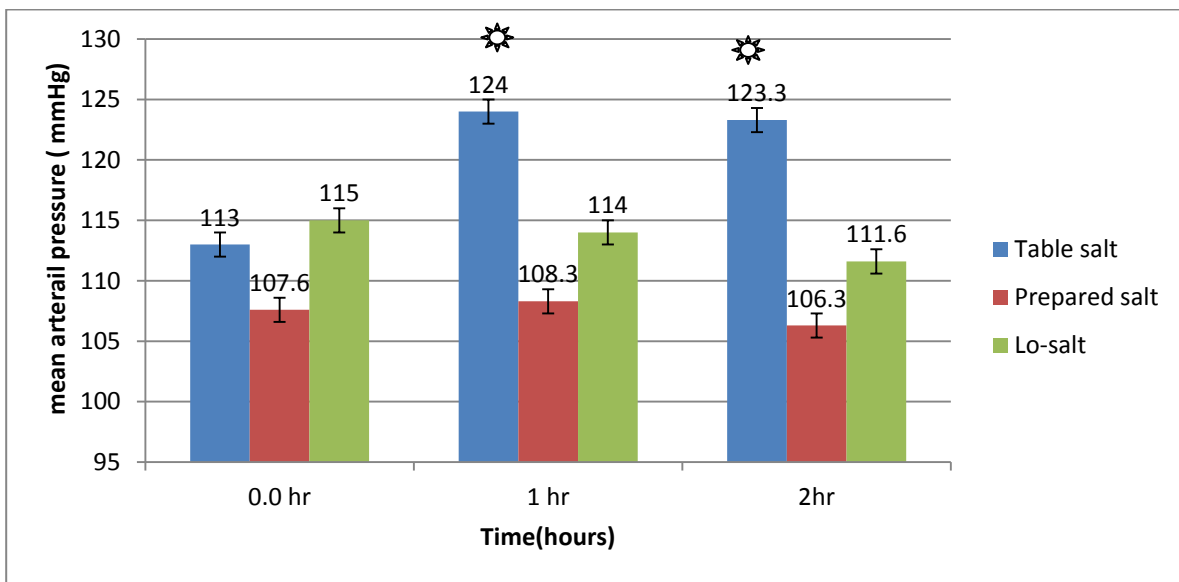
**Figure 3:**  
Effect of three types of salts on the mean arterial pressure in the males



**Figure 4:**  
Effect of three types of salts on the systolic blood pressure in the females



**Figure 5:**  
Effect of three types of salts on the diastolic blood pressure in the females



**Figure 6:**  
Effect of three types of salts on the mean arterial pressure in the females

A clinical trial on 150 Chinese men and women with borderline to mild hypertension found that moderate supplementation with 500 mg/day of potassium chloride for 12 weeks resulted in a significant 5 mm Hg reduction in systolic BP compared to placebo; no changes in diastolic BP were observed in this study (23). In contrast, observation verified that KCl supplementation did not alter the systolic blood pressure (24). Although that low potassium intake may play an important role in the genesis of high blood pressure while increased potassium intake should be considered as a recommendation for prevention and treatment of hypertension, especially in those who are unable to reduce their intake of sodium (23).

Experimental studies also demonstrated an important role of magnesium in endothelial function. Magnesium produces endothelium dependent and endothelium-independent relaxations in rat aorta in a concentration-dependent manner. These relaxant actions of magnesium on intact aortic rings, but not on denuded rings, were suppressed by nitric oxide synthase inhibitors, indicating the important role of nitric oxide in magnesium-mediated dilation (25).

### **Conclusion:**

Unlike table salt, that raises blood pressure in hypertensive patients, the lo- salt does not lead to raise blood pressure, allowing them to use it safely.

## References

- 1-Kaplan , N. (2007) . Systemic Hypertension: Therapy. In: Libby, P. ,Bonow , R., Mann , D. and Zipes , D. , eds. Braunwald's Heart Disease : A Textbook of Cardiovascular Medicine. 8th ed. Philadelphia , Pa : Saunders Elsevier : chap 41.
- 2-Kevin ,M. and Fiona, E.(2006). Salt Handling and Hypertension. Annual Review of Nutrition. 26: 343-365.
- 3-Whitney, E. and Rolfes , S. (2005) . Understanding Nutrition, 10th ed. Thomson/ Wadsworth Publishing Co., Belmont, CA.
- 4- Konishi , M . and SU , C. (1983) . Role of endothelium in dilator responses of spontaneously hypertensive rat arteries. Hypertension. 5: 881-886.
- 5- Tesfamarim, B. and Halperin, W. (1988). Endothelium-dependent and endothelium - independent vasodilatation in resistance arteries from hypertensive rats. Hypertension.11:440-444.
- 6- Tobian, L. (1988). Potassium and sodium in hypertension: The Volhard Lecture. J. Hypertens.6: 12-24.
- 7- Sugimoto, T., Tobian , L. and Ganguli, M. (1988). High potassium diets protect against dysfunction of endothelial cells in stroke - prone spontaneously hypertensive rats. Hypertension. 11: 579-585.
- 8- Clozel , M . , Kuhn, H . and Hefti , F. (1990). Effects of angiotensin converting enzyme inhibitors and of hydralazine on endothelial function in hypertensive rats.Hypertension.16: 532-540.
- 9-Wiemer, G., Scholkens, B., Becker, R. and Busse , R. (1991) . Ramiprilat enhances endothelial autacoid formation by inhibiting breakdown of endothelium - derived bradykinin. Hypertension. 18: 558-563.
- 10- Sheng , H . (2000). Sodium, chloride and potassium. In: Stipanuk M , ed . Biochemical and Physiological Aspects of Human Nutrition. Philadelphia: W.B. Saunders Company. 686 - 710.
- 11- Lorient, D. and Linden ,G . (1999) . New ingredients in food processing :biochemistry and agriculture. Boca Raton: CRC Press. p. 357.
- 12- Gennari, F. (1998) .Hypokalemia. N. Engl. J .Med. 339(7):451-458.
- 13- Adroque, H.and Madias, N. (2007). Sodium and potassium in the pathogenesis of N. Engl. J. Med. 356:1966.

- 14- He, F. ,Markandu, N., Coltart, R., Barron, J.and MacGregor, G. (2005)."Effect of Short-Term Supplementation of Potassium Chloride and Potassium Citrate on Blood Pressure in Hypertensives". *Hypertension*. 45 (4): 571.
- 15- Patnaik, P. (2002). *Handbook of Inorganic Chemicals*. McGraw-Hill, ISBN 0070494398.
- 16- British Pharmacopoeia Commission Secretariat . (2009) ." Index , BP2009 " . [http://www.pharmacopoeia.co.uk/pdf/2009\\_index.pdf](http://www.pharmacopoeia.co.uk/pdf/2009_index.pdf).
- 17-Abraham, A.S., Rosenmann, D., Kramer, M., Balkin, J., Zion, M.M. Farbstien, H. and Eylath,U. (1987) . Magnesium in the Prevention of Lethal Arrhythmias in Acute Myocardial Infarction. *Arch. Intern. Med.* 147 (4): 753- 755. View abstract.
- 18- Pokan, R., Hofmann, P., Von Duvillard, S., Smekal, G., Wonisch, M., Lettner , K .,Schmid, P., Shechter, M., Silver, B. and Bachi , N. (2006) . Oral magnesium therapy, exercise heart rate, exercise tolerance, and myocardial function in coronary artery disease patients .*Br. J. Sports. Med.* 40: 773 – 778.View abstract.
- 19- Cowley AW, Jr, Skelton MM, Merrill DC, Quillen EW, Jr, Switzer SJ. Influence of daily sodium intake on vasopressin secretion and drinking in dogs. *Am J Physiol Regul Integr Comp Physiol* 245: R860–R872, 1983 .
- 20- Fang Z, Carlson SH, Peng N, Wyss JM. Circadian rhythm of plasma sodium is disrupted in spontaneously hypertensive rats fed a high-NaCl diet. *Am J Physiol Regul Integr Comp Physiol* 278: R1490–R1495, 2000 .
- 21- de Wardener HE, He FJ, MacGregor GA. Plasma sodium and hypertension. *Kidney Int* 66: 2454–2466, 2004.
- 22- Ascherio, A., Rimm, E. and Hernan ,M. (1998). Intake of potassium, magnesium, calcium, and fiber and risk of stroke among US men.*Circulation*. 98(12):1198-1204.
- 23- Whelton , P . , He , J . and Cutler , J . (1997) . Effects of oral potassium on blood pressure .Meta - analysis of randomized controlled clinical trials. *JAMA*. 277(20):1624-1632.
- 24- Giovane, S ., Airton, M., Valeria, M., Franca, I.and Andrea, C. (2010) .Evaluation of exercise and potassium chloride supplementation on blood pressure and nociceptive threshold in hypertension rats. *Appl . Physiol. Nutr .Metab .* 35:184-187.
- 25- Yang, Z.W., Gebrewold, A., Nowakowski, M., Altura, B.T., Altura, B.M., 2000. Mg<sup>2+</sup>-induced endothelium-dependent relaxation of blood vessels and blood pressure lowering: role of NO. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 278 (3), R628–639.

## پوختنه

گۆرینی خویی چیشته به خویکی تر به ناوی لو-سولت دیاری کراوه بۆ لابردنی کاریگه‌ری خراپی له سه‌ر په‌ستانی خوین ، کیشه‌ی دل و سووری خوین. ئامانجمان به‌راوردکردنی کاریگه‌ری یه‌ک گرام لو-سولت و یه‌ک گرام خویی ئاماده‌کراوه له سه‌ر په‌ستانی خوین له‌گه‌ل کاریگه‌ری یه‌ک گرام خویی چیشته . لیکۆلینه‌وه‌که ئه‌و خوییانه ده‌گریته خۆی وه‌ک لو-سولت که پیکهاتوه له %٦٦ کلوریدی بوتاسیوم %٣٣.٣ کلوریدی صودیوم و کاربوناتی مغنیسیوم که ئه‌مه کار ده‌که‌ی وه‌ک دژه تۆپه‌ل بوون. من تیکه‌له‌ی لو-سولت ئاماده‌م کردوه وه‌ک تاکیگه . ئه‌ نجامه‌کان نیشته‌نی داوه که خویی چیشته هیچ گۆرانکاریه‌کی به رچاوی نه‌کردوه له سه‌ر په‌ستانی خوین له ماوه‌ی ١-٢ کاتژمیر له وه‌رگرتنی ، له به‌رامبه‌ردا ئه‌نجامه‌کانی خویی چیشته کاریگه‌ری زیاد بوونی په‌ستانی خوینمان پی نیشان ده‌دات دوا‌ی ١-٢ کاتژمیر له وه‌رگرتنی خویی چیشته‌که . ده‌رکه‌وتنی کاریگه‌ری - و خویی ئاماده‌کراوه له سه‌ر په‌ستانی خوین. ئه‌م دۆزینه‌وه‌یه‌ش زیاتر ده‌رخه‌ری سویدی خویی لۆ-سۆلته (دروستکراوه و ئاماده‌کراوه) له سه‌ر په‌ستانی خوین ، شه‌کری خوین و کۆلیسترۆلی خوین، وه ئامازه‌یه بۆ سه‌لامه‌تی به‌کاره‌ینانی ئه‌م خویییه .

## الخلاصة

الاملاح البديلة هي املاح المائدة ذات المحتوى القليل للصوديوم جعلت من اجل تقليل خطر ارتفاع ضغط الدم ومشاكل الاوعية الدموية والقلب . كان الهدف من البحث مقارنة تأثير ملح المصنع lo - salt (١غم) و الملح المحضر (١غم) على ضغط الدم ومستوى الكولوكوز والكوليستيرول في الدم مع ملح المائدة (١غم) . شملت الدراسة استعمال الملح المصنع lo-salt ( يحتوي على الاقل ٦٦ %/ كلوريد البوتاسيوم ، و على الاكثر ٣٣,٣ %/ كلوريد الصوديوم و كاربونات المغنيسيوم كمانع تكتل ) ، الملح المحضر Lo-Salt ( خليط يحتوي على نفس المكونات ولكن تم تحضيره مؤخرا في المختبر و ملح المائدة ( كلوريد الصوديوم ) . اظهرت النتائج ان إعطاء الملح المحضر و الملح المصنع lo-salt لم يحدث تغيرا معنويا لضغط الدم وكولوكوز وكوليستيرول الدم بعد ساعة و ساعتين من تناول الملح . على العكس من ذلك اظهر اعطاء ملح المائدة زيادة معنوية في ضغط الدم بعد ساعة وساعتين من تناوله . النتائج هذه اعطت ايضاحات اكثر حول منافع الملح المصنع والمحضر فيما يتعلق بتاثيرهما على ضغط الدم وكولوكوز وكوليستيرول الدم معطية اشارة خضراء حول سلامة استعمالهما .