

Sodium - A comprehensive Analysis



Researched and Composed by Gabriel "Venom" Wilson, BSc. (Hons), CSCS

Abstract

The macromineral sodium is required for proper levels of osmolarity, electrolyte balance, the thirst mechanism, and much more. Several hormones such as ADH, and the Renin-Angiotensin System were designed to regulate a wide variety of sodium intakes. While your kidneys are highly efficient at excreting this water soluble mineral, excess Na^+ can promote hypertension, and osteoporosis. Proper sodium intakes and athletic considerations will be analyzed.

Minerals

Nearly 4% of our body mass is composed of 21 metallic elements known as minerals. These occur readily in nature, particularly in waters of lakes and oceans, dirt, root systems of plants, and in the structures of those who consume plants and liquids. These inorganic nutrients are constituents of hormones, enzymes, and vitamins. 3 general roles of minerals are regulation, structure, and function: (1) Regulating cellular metabolism by joining with hormones and enzymes that regulate cellular activity. (2) Supplying structure in the formation of teeth and bones. (3) Functioning in the maintenance of muscular contractions, neural activity, heart rhythm, and acid base balance. They may exist free in body fluids, or combine with other chemicals.

The thin line between anabolism and catabolism can collapse if lacking in an essential mineral. For example, several energy-releasing reactions during macronutrient catabolism are activated by minerals. Moreover, the synthesis of the hormones such as thyroxin and insulin, are dependent on zinc and iodine, respectively. Additionally, they are necessary for the manufacturing of nutrients such as glycogen from glucose, proteins from amino acids, etc. Deficiencies can produce a wide range of side effects such as osteoporosis, muscle cramps, anemia, and such like.

Minerals are separated into two classes: macro (also know as major or macronutrient elements) and micro (also referred to as minor, trace, and micro elements) minerals.

Major minerals are required in amounts of more than 100 mg daily, while trace minerals are defined as those required in amounts less than 100 mg daily. There are 7 known major minerals, and 14 trace minerals. Among minor minerals are iron, fluorine, zinc, copper, selenium, iodide, and chromium. Major minerals consist of calcium, phosphorous, potassium, sulfur, chloride, magnesium, and lastly, our topic of discussion, *sodium*.

Sodium

Sodium (NA) has the atomic number (the number of protons in a particular atom. Each proton has a single positive charge) 11. These eleven protons are equally offset by 11 negatively charged electrons (negative charge of one). To fill its valance shelf, however, sodium loses an electron. This gives it a charge of +1, making it an ion (atoms with positive or negative charges), more specifically, a cation (a positively charged ion). A synonym for Sodium is therefore, NA^+ . The most commonly known form of sodium is table salt, or sodium chloride. NA represents 40% of it.



NA^+ is water soluble (can be dissolved/mixed in water, much of this effect is due to its charge, which is attracted to the highly polar H_2O molecule) and highly concentrated in the extra cellular fluids (ECF) of the body. Sodium is loosely bound to macromolecules (large molecules, i.e. proteins); one of its functions is to pass through cell membranes. It operates this way in order to enforce nutrient transport mechanisms and signal nerve impulses.

Sodium is also deemed an electrolyte. That is, a substance which can dissociate into ions in water. Solutions of electrolytes therefore conduct an electric current and can be decomposed in a solution (electrolysis). Electrolytes are both anions (negatively charged ions) and cations (positively charged ions). The body uses them throughout fluid compartments. The goal is to distribute them in such a way that within a given compartment--the blood plasma for example--electrical neutrality is always maintained with the anion concentration exactly balanced by the cation concentration. Major groups of cations include sodium, potassium, calcium, and magnesium. Their negative counterparts consist of chloride, proteins, and bicarbonate, along with low concentrations of organic acids, sulfate and phosphate. The maintenance of pH (level of acidity, the lower the pH, the more acidic), and electrolyte balance is almost always handled by your kidneys.

Approximately 30% of bodily sodium is located on the surface of bone crystals. The remainder is found in the ECF. Lastly, sodium constitutes 93% of the cations in the body, making it by far the most abundant member of this family.

Digestion

Almost 95% of consumed sodium is absorbed by the body, with the remaining 5% being excreted in the feces. Sodium in excess, however, is excreted by the kidneys. Several fundamental pathways are used for sodium absorption across the intestinal mucosa (mucosal cells are any membrane or lining, which contain mucus-secreting glands for lubrication). Among these are:

- The sodium/glucose co transport system, which transpires throughout the small intestine.
- An electrogenic sodium absorption mechanism. This occurs in the colon.
- The sodium/chloride co transport mechanism, active in both the small intestine and colon.
- Electrically silent cotransport on Na^+ , K^+ and 2Cl^- .

The mechanisms used for cellular absorption, so that your body can use sodium to properly function, will be discussed subsequently, along with several other fascinating digestive traits.

Sodium/Potassium Pump

Before discussing the 4 aforementioned pathways, it is imperative to understand a transport system, which plays a vital role in their functions. That is, the Sodium-Potassium (K^+) Pump.

There are two forms of transport process, as follows:

1. Active transport- assisted transport through the plasma membrane, requiring metabolic energy to "power" the exchange of materials.
2. Passive transport- the transport of substances through the plasma membrane, requiring no energy.

To clarify, systems have a tendency to go spontaneously from higher energy concentrations, to lower ones (diffusion). For example, if glucose were to move across the membrane of a cell, it would naturally travel from the higher area of concentration (outside the cell) to the lower area of concentration (inside the cell). This is called passive transport because it naturally occurs, and requires no energy. However, if glucose were to be moved in the opposite direction (from lower to higher concentrations), it would be called active transport because it does not occur naturally, but rather, requires energy.



With that said, the $Na^+ K^+$ pump is a form of active transport. Here is how it works: ATP pumps ions uphill against their electrochemical gradients through the membrane by a special protein enzyme known as sodium potassium ATPase (remember, ase at the end of a word refers to an enzyme) that serves as a pumping mechanism. Such processes must occur within living cells for optimal distribution of cellular chemicals. Sodium ions normally stay in the cell, due to its low levels of concentration. As such, sodium outside the cell naturally wants to continue to diffuse into the plasma membrane. Potassium, however, exists in higher levels of concentration and, thus, tends to diffuse into the ECF. To counteract this (that is, counteracting a state of equilibrium which would be reached without such pumping mechanisms) and achieve proper sodium and potassium concentrations surrounding the plasma membrane, for the maintenance of muscular and nerve functions, both cations must move against their normal concentration gradients. This leads to higher levels of Na^+ in the ECF, and higher levels of K^+ in the ICF. From this, muscular contractions and nervous functions, among other vital systems, are able to function at a maximal rate.

For further comprehension, here is a quote from the man himself, President Wilson:

To make a long story short, in the membrane of both muscle cells and neurons are several complex structures. There are voltage gated channels which allow sodium into the cell, and there are voltage gated channels which let potassium out of the cell. Additionally, there are sodium/potassium pumps, which literally pump three sodiums (against their electrical and chemical gradients) out of the cell, while only pumping two potassiums into the cell. Three positives out and two positives in translates to the inside of the cell being more negative than the outside of the cell. In addition to this, there are also channels in the membrane which allow more potassium to escape than sodium to enter, which means that more positive charge leaves the cell than enters. Finally, negatively charged proteins are manufactured inside of the cell. **The point is simple: by complex machinery, both neurons and muscle cells set up what is called a negative membrane potential, which means that**

the inside of the cell is more negative than the outside. Secondly, the cell has driven sodium outside of the cell, against its chemical and electrical gradient; that is, if you could make the membrane more permeable to sodium, it would rush into the cell like a bullet out of a gun!

This is precisely what occurs in an action potential. Without going into horrid detail, there is a certain threshold for each cell to achieve an action potential. This means that enough positive charge flowing toward negative charge has to occur in order for the entire cell to conduct an electrical impulse. Once this is reached, however, the entire cell will go through an action potential (actually its more similar to tiny action potentials propagating themselves across the cell). Finally, once threshold has been reached, there is no stopping the action potential from spreading across the entire cell! This is why it is [the all or none principle](#).

For more, refer to: [Is the All or None Applicable to an Entire Muscle?](#)

Sodium/Glucose transport system

I discussed this mechanism in an earlier issue of JHR, as follows:

[Glucose/Sodium transport system](#)

Earlier in the article I discussed the sodium/glucose co transport mechanism. This concept falls under the heading of secondary active transport. Primary active transport takes place via a pumping system [uses ATP or some other chemical energy source directly to transport substances]. You see, each of your cells contain proteins which break down ATP, into ADP + P + Energy, and use the products to power the pump. The Sodium/Potassium Atpase, pumps three sodiums out of the cell, and only two potassiums into it. This makes sodium's concentration higher on the outside of the cell. Additionally the inside of the cell is more negatively charged than the outside. Sodium is a positively charged ion and is attracted to the negative area. It has been pumped against its electrochemical gradient (concentration is greater outside of the cell and more negative). Thus, Na⁺ (sodium) will now move back into the cell.

There are proteins within a cell membrane which act to transport glucose. However, the binding site for glucose has a low affinity for it, unless sodium is bound to it. Due to the electrochemical gradient, sodium enters a binding site specific for it on the protein, and when it does so, the protein changes its shape (allosteric reaction), so that sodium can now bind and be transported into the cell. This is called co transport because two substances are transported into the cell together, and secondary active transport because it takes advantage of the concentration gradient set up by the primary mechanism. By taking in the proper amount of sodium, you increase the concentration gradient outside of the cell, and therefore increase sodium's ability to bind to transport proteins. In doing so, you not only increase glucose absorption, but as pointed out, you also further increase water uptake across the luminal membrane of the intestine as well.

In summary, glucose and sodium couple with each other to form a co transport system. This process is mediated by the Sodium Potassium pumping system, which provides the proper energy for this to transpire.



For more on this, study, [Dextrose, Maltodextrin, and Sodium an In Depth Analysis.](#)

Electrogenic sodium absorption mechanism

This is called an electrogenic sodium absorption mechanism because the sodium cation is the lone ion moving past cells, and its transport is regulated by several electrical channels. It enters the colon via Na^+ conducting pathways called sodium channels. These channels cause diffusion (passive transport) inwardly by the downhill concentration gradient. This reaction is accompanied by anions and water, causing it to flow down the colon lumen (tube) down to its blood stream. Lastly, the sodium potassium system, pumps it across the membrane, on the blood stream side of the cell.

Sodium/Chloride Co Transport

This theory is still being analyzed. But an electro neutral (sodium is positive, and chloride is negative, so they offset each other, becoming electrically neutral) $Na^+ Cl^-$ system has been suggested. This has been proposed due to the observation that a vast amount of sodium absorption is usually in the presence of Chloride and visa versa [16]. Mechanisms have not yet been established. One proposed system suggests paired ion exchangers, that is, sodium and hydrogen (H^+) exchange with chloride and bicarbonate (HCO_3^-) [4,22]. This theory states that Na^+ and Cl^- are allowed into the cell, in exchange for H^+ and HCO_3^- . The sodium is then pumped across the cell membrane by the sodium potassium pump, followed by chloride, which crosses via diffusion.

To further narrow things down, J. B. Stokes performed a study on the urinary bladder of the winter flounders [38]. He demonstrated that there is a clear

interdependency between Na^+ and Cl^- net absorption of Cl^- and Na^+ , respectively, and that the process did not require K^+ (which has often been proposed).

Electrically silent cotransport of Na^+ , K^+ and 2Cl^- .

Evidence for a tightly coupled $\text{Na}^+ - \text{K}^+ - 2\text{Cl}^-$ transport (NKCC) has been reported in nearly every animal cell. It was first proposed by Geck et al. [17], and has been investigated for over 2 decades, yet it is still in its infancy. Only broad outlines of the structure, function, and regulation of this ion transport mechanism have been suggested.

The NKCC co transport is a secondary active transport (powered by a concentration gradient, or an electrochemical gradient, that was previously created by primary active transport) process, despite its requirement for cellular ATP. It is generally accepted that this system appears to function in cell volume regulation, as it tends to make the cell swell, thus, counteracting cell shrinking. It is electrically silent (like the sodium/chloride pump). [25].

Hypertension

A primary concern to heightened sodium intakes is hypertension. Hypertension refers to an extremely high blood pressure, which is a significant factor in cardiovascular disease, and renal failure. There are several possibilities by which sodium increases hypertension. One is that sodium retention induces water retention (increased bodily fluid volume), which releases a digitalis-like substance that increases the contractile activity of heart and blood vessels. Another is that the sodium itself penetrates the vascular smooth muscle cell, causing it to contract [24]. Sodium induced hypertension occurs in 1/3 of individuals with high blood pressure. Another major factor is weight. It is estimated that being overweight causes to 20-30% of hypertension diseases [27, 35]. The highest incidence of hypertension is found in northern Japan, where the NaCl intake reached 20-30 grams daily [48].

Animal studies have testified to the negative results of high sodium intakes. For example, chimpanzees added 5, 10, and then 15 grams of NaCl to their usual diet [11]. Over 20 months blood pressure was significantly increased. These inflictions were completely reversed, however, within 6 months of cessation.

Other animal studies show elevated sodium levels may also increase chances of stroke, arterial disease, left ventricular hypertrophy, and renal (relating to kidney) vascular diseases [28].

A major factor to be considered is salt sensitivity. According to Dr. Tom Brody [48], salt-sensitive people (a higher increase in mean arterial blood pressure, in relation to sodium intake) tend to develop hypertension with an intake of 3-6 grams of Na per day, whereas healthy individuals may not with up to 7.2 grams a day. A very large amount (20 grams) leads to hypertension in both salt-sensitive and insensitive people. People likely to be salt-sensitive are those with already heightened blood pressure levels, such as those with a family history of hypertension, the elderly, and those with kidney impairments, which reduce the ability to readily excrete salt out of your system (as mentioned earlier, kidneys are responsible for emptying excess salt out of the body) [24,36]. Also, studies have been composed concerning a genetic variation in the angiotensinogen gene, which produces the hormone

angiotensinogen. This hormone has been shown to increase chances of hypertension in response to sodium [21]. They constrict small blood vessels, which increases blood pressure. Those with stable kidneys may consume higher amounts of salt than those with physical impairments, and excrete excess sodium, well within 24 hours, resulting in no rise of blood pressure [4, 36].

Results show that those with normal blood pressure and who are not salt-sensitive, will not lower blood pressure with reduction of sodium intake, but as stated above, it can be raised via over-consumption [19]. However, for those who are salt-sensitive, or have heart problems, lowering sodium has been shown to be a promising medicine [39]. Recommendations will be discussed further on.

NA+ related hormones

There are 3 primary hormones to discuss in the regulation of sodium. That is, aldosterone, renin, and vasopressin, also known as, Antidiuretic Hormone (ADH).

Renin/Aldosterone

It is important to understand that, with low amounts of sodium, the hormone aldosterone acts on the kidneys to conserve sodium for proper bodily functions. High sodium intake, however, blunts aldosterone release, allowing excess sodium to be excreted in the urine. This was *designed*, so as to maintain a normal electrolyte balance throughout a wide range of dietary intakes.

Antidiuretic Hormone (ADH)

The term, "Antidiuretic" is self-explanatory. Anti means against, and a diuretic is a substance which causes water loss. So this hormone resists water loss.

Exercise

Loss of water and minerals, such as sodium in sweat, is an important factor during exercise, especially in hot weather. Excess depletion can promote severe dysfunctions in the form of heat exhaustion, heat stroke, and cramps. Sadly, due to improper replenishment of fluids and electrolytes, several athletes have died during sports such as football. These factors will play an important role in future issues of JHR, concerning proper workout nutrition.

Your body does, however, have a mechanism to control rapid mineral and water loss. During intense training sessions, the hormones aldosterone, renin, and ADH (discussed previously) are released. These hormones conserve sodium loss, even under extreme conditions such as running a marathon in humid weather.

During exercise, the pituitary gland releases ADH. This enhances water re absorption from the kidney tubules, causing urine to become more concentrated during heat stress. The adrenal cortex additionally releases the hormone aldosterone (this process, along with renin, was described earlier), which likewise increases the renal tubules re absorption of sodium. Aldosterone also reduces sweat osmolarity. As such, prolonged heat exposure decreases sodium concentration in sweat, which assists in additional prevention of electrolyte depletion [5, 10, 15].

Pre-Contest Training

These hormones play a vital role in the subject of pre-contest preparation. Fortunately, the President of Hyperplasia Journals, Jacob Wilson, tore up the subject in one of his recent journal entries. Within, you will learn how to manipulate sodium for tight skin and low water retention on the day of the show. Refer to: [Pre Contest Week - An In Depth Analysis](#).

Thirst Mechanism

Having an appetite for water and sodium is caused by a complex mechanism.

Water is the largest constituent in the body. It can represent 55% of the body weight in a healthy adult. About 70% of the water in your body is intracellular. The rest is in the ECF. Maintenance of hydration is of prime importance to proper functions. Therefore, it makes sense that cellular shrinking (dehydration) and diminished ECF/ICF water volume are two primary causes for thirst. Decreased blood volume appears to be detected by cardiac stretch receptors, and enhances thirst. In regulation of osmolarity, your osmoreceptors, which are in the hypothalamus, detect increased osmolarity and induce the thirst mechanism. Now remember, osmolarity refers to concentration of particles in a solution. If you were to increase sodium, your osmolarity would likewise rise, and if you were to decrease water intake, it would likewise rise, and visa versa. Contrary to this, if osmolarity is decreased, thirst is inhibited. Additionally, increased sodium Gastric load, apparently detected by putative sodium receptors in the abdominal viscera, enhances thirst. Again, countering this, increased gastric water load, detected by the same receptors, decreases thirst [45, 46, 47]. The importance of this, in relation to post-workout nutrition, will be discussed in future issues of JHR.



Several hormones also play a role in thirst desire. It appears that the Renin-Angiotensin System (discussed earlier), plays a primary role. Particularly Angiotensin II. For example, several studies have been conducted using rats. ANG II was administered by intracranial injection [12, 40, 41]. It was observed that the animals stopped whatever they were doing, went to the source of water, and started to drink, around 1 min after injection, and consumed significant amounts of water for 10-15 minutes. From an immensely complicated study on the thirst mechanism, DR. J. T. FITZSIMONS has this to say [23]:

The stimulating effect of ANG II on drinking is so powerful and so widespread among the many mammals and birds that have been tested,

and the behavior aroused so apparently normal, it invites speculation on its physiological significance and on how its effects on drinking fit in with the overall fluid and electrolyte economy of the body.

So it is clear that ANG II has a profound effect on thirst appetite. Other factors besides angiotensin hormones include many central neurotransmitters or paracrine agents, including catecholamines, serotonin, amino acids, tachykinins, opioids, and mineralocorticoids [23].

Sodium appetite has similar mechanisms. An increase in sodium hunger is the second behavioral response to hypovolemia (decreased water volume). Many mammals in sodium deficit seek and ingest salt, driven to do so by increased sodium appetite [9, 37, 42]. Through 50 years of intense research, it has been shown definitively that the hormones discussed earlier, such as Renin and ADH, play a primary role in sodium appetite [9, 23]. For example, sodium appetite is increased in rats by inducing the syndrome of apparent mineralocorticoid excess [6, 7]. This mechanism is an essential defense against sodium deficiency, to maintain electrolyte balance, and maximum efficiency [13, 23]. Sodium depletion is of primary concern during hard-core training sessions, where sodium loss is rapidly increased [30].

As an athlete, it is imperative that you listen to your body, and give it the proper nourishment. So if you are thirsty right now, drink up!

Sweat loss

As mentioned previously, sodium is water soluble. This relates to a large amount of sodium loss through sweat during intense training sessions. Sweat is produced by specialized sweat glands beneath the skin. Evaporation of sweat's water components results in a refrigeration mechanism, to cool the body down.

Typically, a well-assimilated athlete will lose .5L - 3L of sweat during each hour of exercise. On average, an athlete loses 1-1.5 liters per hour. Higher intensity results in increased sweat loss. Humidity, heat, and other weather-related factors will result in increased sweat secretion as well. Every liter of sweat contains a whopping .6 g of sodium. This is a vital factor in optimal post-workout nutrition.



It is also important to note that increasing heat and sweat loss before and during training sessions is extremely beneficial to the athlete. For more on this, refer to: [Mobility Training and the Application of Proper Warm-Up for Bodybuilders](#).

Diarrhea

Diarrhea is a serious problem that causes a quarter of the 10 million infant deaths that occur each year.

In adults, it is often caused by food poisoning, overeating, junk food, etc. Moderate cases may result in the loss of 50 mmol of Na, Cl, and K per kilogram of stool. High volume cases may produce 70 mmol losses. The World Health Organization (WHO) has recommended a proper hydration formula consisting of 3.5 g of NaCl, 2.5 g of NaHCO₃ (or 2.9 g of Na citrate), 1.5 g of KCl, and 20 g of glucose or glucose polymer. The glucose assists in intestinal absorption of sodium ions, due to the glucose sodium co transport system discussed earlier. Ample amounts of water should be ingested as well. This is usually suitable for most cases of diarrhea.

Osteoporosis

Sodium has been shown to increase kidney calcium excretion. In the USA, over 90% of ingested sodium is excreted. It was shown that having 2.3 g of sodium a day excreted approximately 40 mg of calcium [31, 34]. Likewise, increased calcium causes natriuresis (increased excretion of sodium). Urinary sodium has been negatively correlated with changes in bone density in the hip of postmenopausal women. Decreasing sodium intake or doubling calcium intake was shown to reduce bone loss equivalently, however [8]. Calcium also has positive effects on hypertension [20, 43]. Recommendations will be discussed further on.

Deficiencies

The range of deficiencies is broad for sodium. These include muscle cramps, mental apathy, reduced appetite, electrolyte imbalances, dehydration, hyponatremia, decreased utilization of nutrients, impaired nervous functions, fainting, imbalanced osmolarity, convulsions, hypovolemia, hypotension, and further deterioration of renal function [43].

Practical Applications

The RDA for sodium ranges from 1100-3300 for both men and women. The American Heart Association recommends 2.4 grams of sodium per day.

Concerning hypertension, patients who have congestive heart failure with edema, as well as salt-sensitive individuals, have responded to a diet restricted to 1.5 to 2.0 g sodium a day [2, 18]. Calcium has also been shown to have positive effects on blood pressure [24]. The National Institute of Health consensus panel has recommended having 1,200 to 1,500 mg of calcium daily, which should be helpful in promoting blood pressure regulation and preventing osteoporosis [20, 43]. For more on calcium, read Seksi's article, [Supplement Review - Calcium](#).

Reducing sodium has been considered for decades an effective way to improve blood pressure, among other diseases. However, recent controversy is prevalent. As stated above, lowering sodium will not decrease blood pressure in already stable individuals. However, it can reduce hypertension in salt-sensitive and disease-inflicted humans. But, these results have had high scrutiny lately; the effectiveness of restricted sodium diets is under serious question [14, 26, 32]. Furthermore, according to Dr. Tom Brody [48], salt-sensitive people (a higher increase in mean arterial blood pressure, in relation to sodium intake) tend to develop hypertension with an intake of 3-6 grams of NA per day, whereas healthy, normal individuals may not with up to 7.2 grams a day. A very large amount (20 grams) leads to hypertension in both salt-sensitive and insensitive people. It should also be noted that hypertension is a complex subject, and several factors must be considered. For example, studies show increasing magnesium, potassium, calcium, and fiber, among other foods, can significantly decrease blood pressure [3].

The normal human body requires 500 mg of NA⁺ per day. Athletic requirements vary, however. As stated above, you can lose more than 500 mg in just an hour of training. You can very easily lose more grams of salt through exercise than what is recommended to be consumed total daily. In fact, many athletes (such as long distance runners) during prolonged exercise in the heat have been shown to lose 13-17 g of salt daily, more than 8 g of what is typically consumed. Long distance runners, who do not properly replace sodium, have indeed suffered fatal consequences. Athletes who do not replace sodium after hard-core training sessions are severely halting their results. In fact, if a post workout solution does not contain a sufficiently high sodium content, excess fluid intake will merely increase urine output, greatly limiting ample rehydration. Results show that drinking of plain water in half amount lost corrects only half water deficit as urinary output remains low for 6 hours of hypohydration. When large quantities are ingested, this induces urination and sweating, which limits retention of expanded plasma volume. Moreover, urine output triples when water intake matches water deficit, and increases 12 fold when intake is twice as large as deficit [33, 44]! Furthermore, pure water absorbed from the gut rapidly dilutes plasma sodium concentrations, decreasing osmolarity, and therefore inhibiting the thirst mechanism. For more on sodium post workout, refer to, [Dextrose, Maltodextrin, and Sodium an In Depth Analysis](#).

Besides post workout nutrition, and in some cases during workout nutrition, athletes generally obtain enough sodium in their regular diets. I would not include exercise sodium supplementation to your daily NA⁺ consumption, however, as you are simply replacing what was lost.

Exercise Nutrition

With the foundation laid, in subsequent issues of JHR, we will be investigating sodium and other factors which coincide with sodium, such as renin, sweat, etc., and applying them to workout nutrition! ***Are You Ready for the Revolution?***

Conclusion

Salt has great spiritual applications [1]:

Colossians 4:6

6 Let your speech be always with grace, seasoned with salt, that ye may know how ye ought to answer every man.

Paul is teaching an important lesson in effectual apologetics (Greek term for giving a defense of the faith). Christians need to season all their conversation with grace, seeking to comfort, edify, instruct, and build up others.

Furthermore, we must, "Be ready always to give an answer to every man who asketh you a reason of the hope that is in you, with meekness and fear (1 Peter 3:15)."

Christ said to be as wise as serpents and harmless as doves. As ambassadors of our Lord, we must be prepared to answer and defend the faith to all men, always in a loving manner.

Keep it Hardcore,

Venom

Executive of Bioenergetic Research

Venom@abcbodybuilding.com

References:

1. The Holy Bible.
2. American Dietetic Association. Handbook of clinical dietetics. Hanover, MA: Yale University Pres 1981; G5-6
3. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, Bray GA, Vogt TM, Cutler JA, Windhauser MM, Lin PH, Karanja N. A clinical trial of the effects of dietary patterns on blood pressure. DASH Collaborative Research Group. N Engl J Med. 1997 Apr
4. Barrett KE, Dharmasathaphorn K. Transport of water and electrolytes in the gastrointestinal tract: physiological mechanisms, regulation, and methods of study. New York: 1994.
5. Costill DL, Branam G, Fink W, Nelson R. Exercise induced sodium conservation: changes in plasma renin and aldosterone. Med Sci Sports. 1976 Winter
6. COONEY, A. S., AND J. T. FITZSIMONS. Increased sodium appetite and thirst in rat in apparent mineralocorticoid excess induced by glycyrrhizic acid (Abstract). J. Physiol. (Lond.) 487P: 27P, 1995.
7. COONEY, A. S., AND J. T. FITZSIMONS. Increased sodium appetite and thirst induced by the ingredients of liquorice, glycyrrhizic acid and glycyrrhetic acid. Regul. Pept. 66: 127-133, 1996
8. Devine A, Criddle R, Dick I, A longituted study of the effect oof sodium and calcium intakes on regional bone density in postmenopausal women. AM J Clin NUTR 1995.
9. DENTON, D. A. The Hunger for Salt. Berlin: Springer-Verlag, 1982.

10. De Souza MJ, Maresh CM, Maguire MS, Kraemer WJ, Flora-Ginter G, Goetz KL. Menstrual status and plasma vasopressin, renin activity, and aldosterone exercise responses. *J Appl Physiol.* 1989 Aug
11. Denton D, Weisinger R, Mundy N, Wickings EJ, Dixson A, Moisson P, Pingard AM, Shade R, Carey D, Ardailou R, Paillard F, Chapman J, Thillet J, Michel JB. The effect of increased salt intake on blood pressure of chimpanzees. *Nat Med.* 1995;1:1009-1016
12. EPSTEIN, A. N., J. T. FITZSIMONS, AND B. J. ROLLS. Drinking induced by injection of angiotensin into the brain of the rat. *J. Physiol. (Lond.)* 210: 457-474, 1970
13. EPSTEIN, A. N. Prospectus: thirst and salt appetite. In: *Handbook of Behavioral Neurobiology. Neurobiology of Food and Fluid Intake*, edited by E. M. Stricker. New York: Plenum, 1990, vol. 10, p. 489-512.
14. Ely, D.L Overview of dietary sodium effects on the interactions with cardiovascular and neuroendocrine functions. *AM J CLIN NUTR* 1997.
15. Francesconi RP. Endocrinological responses to exercise in stressful environments. *Exerc Sport Sci Rev.* 1988
16. Frizzell RA, Field M, Schultz SG. Sodium-coupled chloride transport by epithelial tissues. *Am J Physiol.* 1979 Jan;236
17. GECK, P., C. PIETRZYK, B.-C. BURCKHARDT, B. PFEIFFER, AND E. HEINZ. Electrically silent cotransport of Na⁺, K⁺ and Clin Ehrlich cells. *Biochim. Biophys. Acta* 600: 432-447, 1980
18. Goodhart R, Shils M. *Modern nutrition in health and disease* 5th ed. Philadelphia: Lea & Febiger 1978.
19. Gradual NA, et al. Effects of sodium restriction on blood pressure, renin, aldosterone, catecholamines, cholesterol, and triglyceride: meta-analysis *JAMA* 1998.
20. Gruchow H, Sobociniski K, Barboriak J. Calcium intake and the relationship of dietary sodium and potassium to blood pressure. *AM J CLIN NUTR* 1988.
21. Hunt SC, Cook NR, Oberman A, Cutler JA, Hennekens CH, Allender PS, Walker WG, Whelton PK, Williams RR. Angiotensinogen genotype, sodium reduction, weight loss, and prevention of hypertension: trials of hypertension prevention, phase II. *Hypertension.* 1998 Sep;32(3):393-401.
22. Independent Na⁺ and Cl⁻ active transport by urinary bladder epithelium of brook trout
23. J. T. FITZSIMONS. Angiotensin, Thirst, and Sodium Appetite. *PHYSIOLOGICAL REVIEWS* Vol. 78 No. 3 July 1998
24. *J Am Coll Nutr.* 1995 Haddy FJ, Pamnani MB. Role of dietary salt in hypertension.
25. John M. Russell. Sodium-Potassium-Chloride Cotransport. *Physiological Reviews*, Vol. 80, No. 1, January 2000
26. Luft G.S and Weinberger, M.H Heterogeneous responses to changes in dietary salt intake: the salt-sensitivity paradigm. 1997.
27. MacMahon SW, Cutler J, Brittan E, et al. *Eur Heart J* 1987; 8 (Suppl B):57-70.
28. MacGregor GA. Salt—more adverse effects. *Am J Hypertens.* 1997;10:37S-41S.
29. MONDER, C., AND P. C. WHITE. 11-Hydroxysteroid dehydrogenase. *Vitam. Horm.* 47: 187-127, 1993
30. MCCANCE, R. A.. Experimental sodium chloride deficiency in man. *Proc. R. Soc. Lond. B Biol. Sci.* 119: 245-268, 1936.

31. Massey LK. Dietary factors influencing calcium and bone metabolism: introduction J nutr 1993.
32. Midgley J.P et al Effect of reduced dietary sodium on blood pressure: A meta analysis of randomized controlled trials JAMA 1996.
33. Maughan RJ, Leiper JB. Sodium intake and post-exercise rehydration in man. Eur J Appl Physiol Occup Physiol. 1995
34. Nordin B, Need A, Morris H, Horwitz M. The nature and significance of the relationship between urinary sodium and urinary calcium in women. J nutr 1993.
35. National High Blood Pressure Education Program Working Group. Arch Intern Med 1993
36. Overlack A, Ruppert M, Kolloch R, Gobel B, Kraft K, Diehl J, Schmitt W, Stumpe KO. Divergent hemodynamic and hormonal responses to varying salt intake in normotensive subjects. Hypertension.. 1993;22:331-338
37. ROWLAND, N. E., AND M. J. FREGLY. Sodium appetite: species and strain differences and role of renin-angiotensin-aldosterone system. Appetite 11: 143-178, 1988
38. STOKES, J. B., I. LEE, AND M. D'AMICO. Sodium chloride absorption by the urinary bladder of the winter flounder. A thiazide-sensitive, electrically neutral transport system. J. Clin. Invest. 74: 7-16, 1984
39. Stamler J. The INTERSALT study: background, methods, findings, and implications, AM J Clin Nutr 1997.
40. SEVERS, W. B., AND A. E. DANIELS-SEVERS. Effects of angiotensin on the central nervous system. Pharmacol. Rev. 25: 415-449, 1973
41. SIMPSON, J. B., A. N. EPSTEIN, AND J. S. CAMARDO. Localization of receptors for dipsogenic action of angiotensin II in the subfornical organ of rat. J. Comp. Physiol. Psychol. 92: 581-608, 1978
42. SCHULKIN, J. Sodium Hunger: the Search for a Salty Taste. Cambridge, UK: Cambridge Univ. Press, 1991.
43. Schneider H, Anderson C, Coursin D. Nutritional support of medical practice. Hagerstown, MD: Harper & Row, 1977.
44. Shirreffs, S.M et al. Post exercise rehydration in man: effects of volume consumed and drink sodium content. Med Sci. 1996
45. Stricker EM, Sved AF. Thirst. 2000
46. Stricker EM, Callahan JB, Huang W, and Sved AF. Early osmoregulatory stimulation of neurohypophysial hormone secretion and thirst after gastric NaCl loads. Am J Physiol Regul Integr Comp Physiol 282: R1710-R1717, 2002.
47. Starbuck EM and Fitts DA. Influence of the subfornical organ on meal-associated drinking in rats. Am J Physiol Regul Integr Comp Physiol 280: R669-R677, 2001.
48. Tom Brody. Nutritional Biochemistry. Academic Press. 1998.
49. William D. Mcardle, Frank I. Katch, Victor L Katch.
50. Witterman J, Willet W, Stampfer M. Dietary calcium and magnesium and hypertension: a perspective study. Circulation 1987.

