# Mineral Logic

Understanding the
Mineral Transport System

**Volume 1 · Overview**

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# FOREWORD

With this new book the Cinderella portion of the nutritional world (minerals) comes out as a beautiful princess on the way to the royal ball.

Through *Mineral Logic* we learn about these fascinating nutrients which the body cannot produce. Absorption and transportation of minerals is fully explained in a technical way but easily understandable.

A text such as this has been sorely needed by the professional, as well as the lay public.

We are introduced to the latest technology in mineral transportation systems; such terms as "picolinate," "ascorbate," and " orotate" are fully explained and each of their benefits is revealed.

With the continuation of this series, the reader will have a complete and comprehensive knowledge of all the essential minerals, how they are absorbed, and most efficiently utilized.

*Earl Mindell, R. Ph., Ph. D.*

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MINERAL LOGIC

*INTRODUCTORY VOLUME*

# *PREFACE*

Through this introductory volume and the ones to follow it will be my intention to present an objective clarification of what is truthfully known about each of the most significant minerals in human nutrition. It was suggested to me that the title of these booklets should be "The Absolutely Final Word About Minerals in Human Nutrition." My overzealous supporter believed such a title would more accurately reflect my thorough resolve to set the record straight. For, indeed, misconceptions abound in the field of human nutrition concerning the absorption, transportation and utilization of minerals. These three concepts combine into an overall perception of true mineral bioavailability, or more accurately, bio-utilization. Just what forms of minerals are best absorbed and carried to the cells? How are they carried? What do they do at the cell site? And ultimately, how much do we really know for sure?

These questions intrigue not only the lay consumer of nutritional supplements seeking optimal health and the most economical way of achieving it, but they also intrigue ardent researchers in the hallowed halls of science. Just how much can they tell us today? What is clearly understood about minerals?

This series of specialized books on the minerals will seek to sweep away myth and misunderstanding - much of which has been perpetrated through self-serving and often inaccurate marketing practices by some of the sellers of branded nutritional supplements. This series will attempt to replace myth with fact, to remove the "mis" from misunderstanding, and to accurately identify the advanced theories of mineral transportation to cells.

In many cases, we may find that what we thought was true may not be; that is, what science established as fact in the recent past may be changed today due to new discoveries, and what some marketers have identified as fact may only be theory.

But - and I am sorry to disappoint my zealous supporter - nothing written here can be considered absolute. The field of nutritional science is too dynamic. New discoveries daily alter our understandings. I could never presume to have absolute knowledge on the subject. What I have learned I will simply pass on to you, all the time wondering what it is I have missed. If the knowledge of any one subject can be likened to a field of grain, once harvested, some grain will always

be left behind, hidden among the furrows, hillocks, and ravaged stalks. We can never hope to get it all -whether it be grain or complete knowledge. And when the following season comes, new grain (new information) grows up adding to what went before. So what is written today may be legitimately revised in the future. Some of what is here may even be obsolete before it is printed.

This necessary qualification of the information offered does not preclude the spirit in which this miniature encyclopedia project is begun. The facts must be set straight and the suppositions clarified. The introductory volume will offer a general and brief background of salient information on the orthodox and avant-garde theories of mineral bio-utilization. Each succeeding booklet will expand on the data presented in volume one, focusing on each mineral or a group of minerals in a single volume. Excerpts from some of these volumes-in-progress are included as an appendix to this volume.

*Mark Timon*
*Ellicottville, NY*
*Spring, 1985*



# MINERAL LOGIC

## *VOLUME 1*

# CHAPTER 1
# ESSENTIALITY

Minerals may be called "the unsung heroes of nutrition." Without enough of the right ones you would simply die. But their importance in the collective mind of the public at large falls far short of that held for the revered vitamins. After all, the name "vitamin" itself directly proclaims the life or death importance of that class of nutrient. Vitamin stands for "vital to life." The name connotes, "Without me, you die."

The less dramatic word "essential" must precede "mineral" before we begin to recognize the vitalness of the minerals which are no less important to life. The present relationship between dominant vitamins and second place minerals in our minds is not without irony; for centuries ago - long before the first vitamin was ever discovered or even conceptualized - minerals were recognized to be vital to life and health. Soaking a ferrous (iron containing) metal in vinegar would yield a draught that could correct anemia. Eating bones was known to build strong bones.

The first half of the twentieth century saw the celebrity of minerals overshadowed by the newly discovered vitamins. Each isolation of a new vitamin focused attention away from minerals and onto the newer nutritional factors which could be implicated in a number of dramatic deficiency diseases. Certainly the curing of mental disorders, rickets, pellagra, beri-beri, and scurvy, for example, contributed to the overpowering chrisma of vitamins.

Osteoporosis, immune deficiency, stunted growth, dental decay, aging, and a host of degenerative diseases are indeed responsive to mineral therapies too. But none, because of their long duration of cure carries the drama associated with vitamin deficiencies, even though many mineral deficiency conditions can become as lethal as any vitamin deficiency state sometimes within an even greater time span.

I believe it is now time to reestablish the notoriety of minerals on a par equal to that of vitamins. Both classes of nutrients are essential to life. Both are

required to make enzymes work and thus to sustain metabolism and life itself. But minerals are also needed as are proteins and fats to maintain the physical structure of the body. This structural role played by minerals is unmatched in the realm of vitamins. We will examine many of the roles of each of the minerals essential to human nutrition and the question of relative bio-utilization of the various forms throughout succeeding volumes of this series. Let us now turn our attention to biochemical and anatomical information that will provide a foundation for understanding the mineral transport system.



# CHAPTER 2

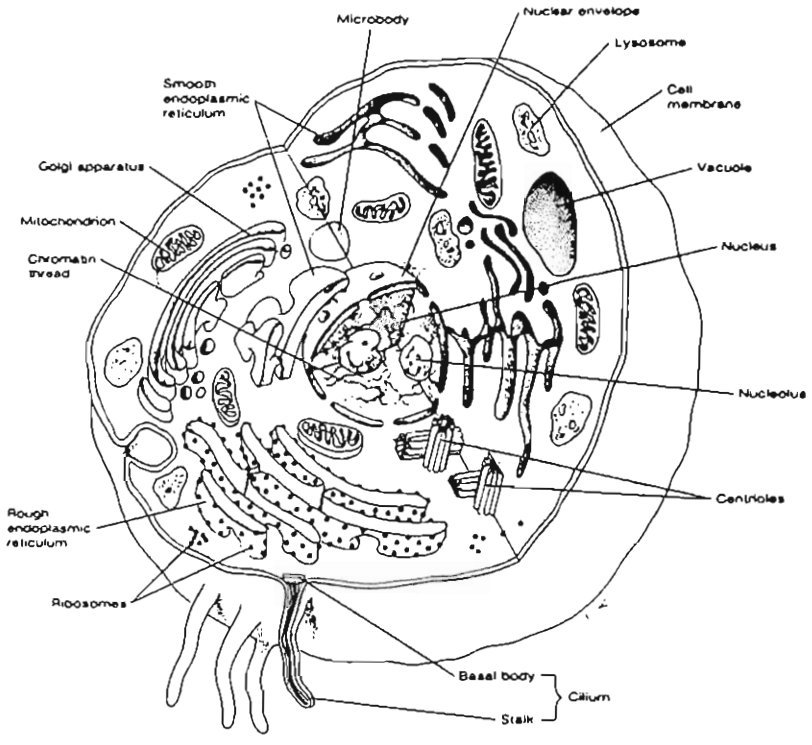
# CELL MEMBRANE TRANSPORT

In 1838 and 1839, two German biologists, Matthias Schleiden and Theodor Schwann, published treatises proposing that all living things were composed of cells. In the years since the formulation of this cell theory, it has been shown that organisms from bacteria to humans indeed consist of cellular units. The basic cellular structure of tissues and organs is actually visible upon microscopic examination. In humans and other higher organisms, cells have become specialized both anatomically and physiologically. Muscle cells, for example, have a well-developed ability to move or contract, whereas nerve cells are specialized to transmit impulses. Other cells may exhibit highly developed properties of metabolism (the ability to process foods, obtain energy, and synthesize products), irritability (the capacity to respond to stimuli), or reproduction (the ability to duplicate themselves). It is important to remember, however, that these properties are present to some degree in all cells and should be regarded as general cell characteristics.

## CELLULAR COMPONENTS

During the early years of cell study, it was believed that cells consisted of a uniform material called protoplasm, and that all of the properties characteristic of life were inherent within this material. As more sophisticated instruments and techniques were developed, it was found that cellular structure was far from uniform and the term "protoplasm" fell into disuse.

A central body called the nucleus was discovered within the cell. The nucleus was found to be enveloped by cytoplasm, a medium in which it was suspended. The cytoplasm was then found to contain a number of other structures called organelles (F.1). "Organelles" stands for "small organs," for indeed the organelles fulfilled functions within the cell similar to those filled by the organs of the human body. As improved biochemical and physiological examination techniques evolved from new technologies, we began to learn more about the functioning of the various cellular structures including the cell membrane. The electron microscope especially made it possible to closely examine the boundary of the cell, the cell membrane.




F.1 A "typical" animal cell showing subcellular organelles. There is probably no actual cell that can be considered "typical" in all respects.

Yet even before the cell membrane was observed microscopically, its existence was inferred from experiments demonstrating that all materials were not able to enter cells with equal ease. Some sort of selective barrier at the cell surface was indicated. This barrier inhibited and controlled free diffusion. The barrier - the cell membrane - was found to be highly selective, affecting the entry of some molecules into the cell much more than others.

At the present time, our knowledge of the exact structure of the cell membrane remains incomplete. For many years, a widely accepted theory envisioned the cell membrane as a relatively rigid structure composed of a phospholipid layer two molecules thick, covered at each surface by a layer of protein (F.2). More recent evidence suggests that the cell membrane is a relatively fluid structure made up of a skin of phospholipid surrounding the cytoplasm. Freckles of protein and mucopolysaccharides are thought to be interspersed throughout the phospholipid skin (F.3). Both views of membrane structure include the existence of pores or channels in the membrane that connect the interior of the cell with the external or extracellular fluid environment. The membrane is also highly

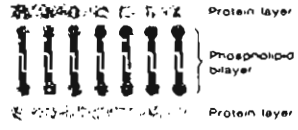




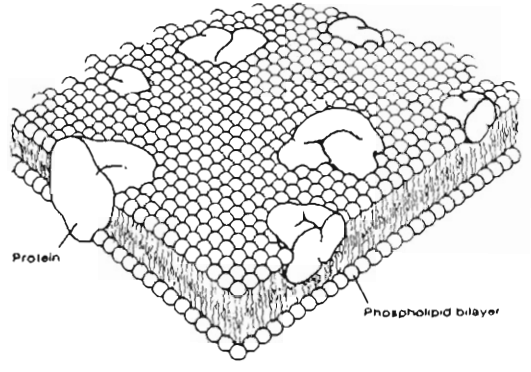
dynamic, changing its shape and structure thousands of times a minute. It is a unit of the cell that is as active as any other organelle.

Basic knowledge of the movement of materials across cell membranes will aid in comprehending the upcoming chapter on mineral bio-utilization. All materials that enter or leave a cell must pass either through this dynamic cell membrane or through pores in the cell membrane. The properties of the membrane as well as the properties of the penetrating molecules or ions are important in determining the ease with which substances will be able to enter or leave cells.

NONMEDIATED TRANSPORT. Many nonpolar compounds (compounds lacking an electrical charge) that are soluble in lipids (fat), move with relative ease through the cell membrane by simple diffusion. Water-soluble or polar (electrically charged) compounds such as mineral ions, on the other hand, are generally believed to move through the membrane by diffusion only with difficulty, if at all. Thus the lipid portion of the membrane is thought to be the primary barrier to substances entering or leaving cells. The membrane has the responsibility of selectively accepting nutrients and rejecting other substances including waste products.




F. 2 A theory of cell membrane structure that was widely accepted for many years maintained that the cell membrane is composed of a phospholipid layer two molecules thick, covered at each surface by a layer of protein.




F.3 A current theory of cell membrane structure proposes that the cell membrane consists of a skin of phospholipid two molecules thick in which freckles of proteins are distributed.

The membrane pores provide alternate routes of entry for polar ions and molecules. This method of entry though is limited to molecules small enough to fit through the pores. In addition, the cell membrane is often thought to carry a charge, probably positive,* that may hinder the movement of ions through pores. Negative ions may be attracted to the surface of the membrane while positive

* One known exception is the red blood cell. Here sialic acids (that make up part of the polysaccharide cell coat) on the surface of the cell membrane are ionized, leaving the red blood cell membrane with a negative charge.

ions are repelled. As a result, neutral particles may pass through membrane pores more easily than charged substances,† and positive mineral ions (potassium, $K^+$, calcium, $Ca^{++}$, magnesium, $Mg^{++}$) may have difficulty approaching and entering the cell. (An ion is an atom or group of atoms that carries a positive or negative electrical charge.)

MEDIATED TRANSPORT. It has been observed that larger polar molecules can still readily enter cells despite the fact that they are not very soluble in lipids nor are they small enough to fit through pores. Various forms of "mediated" transport help to account for the ability of these substances to cross the cell membrane. Mediated transport utilizes intermediary transporting agents in the form of protein carrier molecules that are located within the cell membrane itself or are constituents of the blood. Molecules to be transported across the cell membrane are believed to attach to these carrier molecules. When combined with a carrier, the transported molecule is able to cross the membrane and, depending upon the direction of the transport, either enter or leave the cell (F.4 (a)).

The concept of carrier molecules brings into focus three other considerations, any or all of which may apply to a given carrier molecule. The considerations are of specificity, saturation and competition.

SPECIFICITY. Specificity implies that specialized carrier molecules bind with only one individual, class or group of ion(s) or molecule(s). Thus the substances that may be carried by mediated transport are limited to those that can link with the carrier (F.4 (b)). (Most carrier molecules as yet identified appear to be pure proteins or protein-based.)

SATURATION. Saturation implies that there is a limited number of molecules of a substance to be transported that can cross the membrane by mediated transport in a given span of time. This means that when all of the carrier molecules for a given substance are being utilized, increasing the concentration of that substance will not increase its rate of transport. (There are no more freight cars to carry the coal, you might say.) (F.4 (c) ).

COMPETITION. Competition implies that some carrier molecules can bind with more than one substance to be transported. As a result, these substances can compete for the services of the carrier. Such is believed to be the case for calcium and magnesium, for instance (F.4 (d)).

Suppose both substance A and substance B are transported across the membrane by carrier X. If only substance A is present, then all the carrier X molecules can be utilized in transporting substance A. If substance B is added to the system, it will compete with substance A for the services of the carrier X molecules. Some substance A and some substance B will be transported, and the transport rate of substance A may decrease compared to the rate when

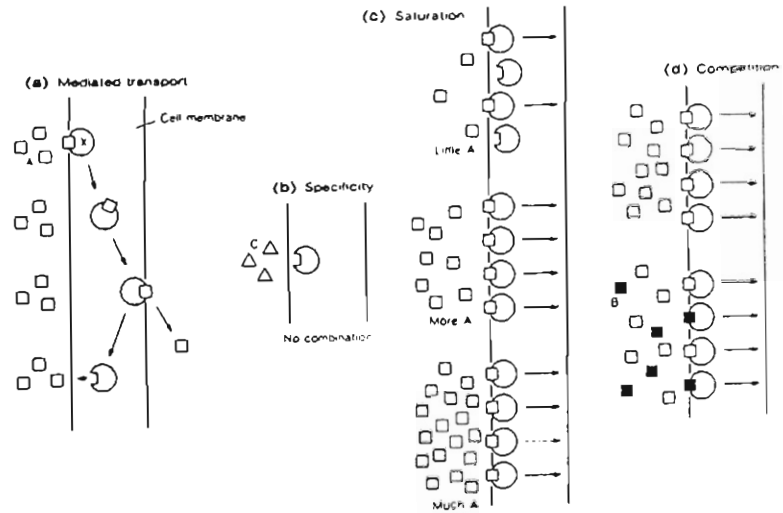
† The assumption that neutral particles may pass through membranes more easily bears directly on our upcoming discussion of bio-utilization. Many of the new sophisticated mineral transport compounds we will examine carry a neutral charge.





substance A was present alone. But maintaining a high concentration in the extracellular fluid can ultimately increase the amount of a substance transported into a cell as the carriers continue to spend 100% of their time shuttling the substance across the cell membrane. Limitations of saturation and competition can thereby be somewhat overcome.

F.4



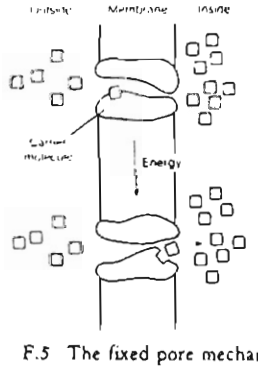

Mediated transport across cell membranes can be either "active" - requiring cellular energy and possibly moving against diffusion gradients - or "passive," requiring no cellular energy and following normal diffusion gradients from an area of high concentration to one of lower concentration.

One type of passive mediated transport is called "facilitated diffusion." In this process, a molecule that by itself may not be able to effectively penetrate the cell membrane is bound to a carrier molecule on one side of the membrane. Once bound, the molecule to be transported is able to be moved across the membrane. Once across the membrane, the bound, transported molecule is then released from the carrier. In this passive method of transport, the carrier is believed to move the substance equally well in either direction, into or out of the cell. However, if more of the substance to be transported is outside the cell than inside, more transfer will occur from outside to inside in accordance with the diffusion gradient resulting in a net movement of the substance inward. Thus the net movement in facilitated diffusion, as with any diffusion, is from a region of high concentration to a region of low concentration. When equal concentrations of the substance on either side of the membrane are reached, there will be equal transport in both directions and no net gain on either side of the membrane. Facilitated diffusion is a passive process in which no cellular energy is used.

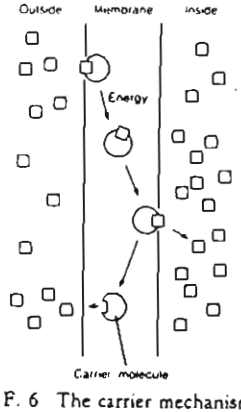
Another type of mediated transport is "active transport." This type of transport is not only able to move materials that might not otherwise pass across

cell membranes, but it is also able to move the materials from regions of low concentration to regions of high concentration against normal diffusion gradients. Such an activity requires cellular energy and depends upon the metabolism of the living cell.

Several theories about how substances are moved by active transport have been proposed. One theory called the "fixed pore mechanism" suggests that a carrier molecule is fixed in place within the cell membrane (F.5). The carrier possesses a binding site that is directed or aimed toward one side of the membrane. A molecule to be transported from that side to the other can attach to the site. Once attached, the carrier molecule lining the pore is motivated either directly or indirectly by the expenditure of energy by the cell. (The energy for this process comes from the breakdown of ATP whose synthesis requires a number of vitamins plus phosphorus. Here is an illustration of how vitamin *and* mineral nutrition work together to promote proper physiological function.) The carrier as a result undergoes a conformational change and translocates the binding site to the other side of the membrane. The translocation of the binding site carries the attached molecule of the substance to be transported across the membrane. Once across the membrane, the bound molecule of the transported substance is released, and the carrier molecule reverts to its original conformation. This returns the binding site to its initial position so that another molecule of the substance can be transported. The fixed pore mechanism then is believed to operate in a fashion similar to an escalator or automated walkway.




F.5 The fixed pore mechanism of active transport.




F. 6 The carrier mechanism of active transport.

An alternate theory of active transport is called the "carrier mechanism." This theory proposes that a carrier molecule within the cell membrane binds on one side of the membrane to a molecule of the substance to be transported (F.6). Again, as in the fixed pore theory above, the expenditure of metabolic energy by the cell causes the entire carrier protein to rotate and move across the membrane





in order to deliver its bound, transported molecule to the other side. There the transported molecule is released. The carrier then rotates back across the membrane and picks up another molecule of the substance to be transported.

Since it would require a considerable expenditure of energy by the cell to support the rotation of an embedded carrier molecule across the span of a membrane, the "carrier mechanism" theory of active transport appears less acceptable than the lower energy cost, "fixed pore mechanism." The fixed pore mechanism also seems to relate more directly to membrane structure as we are coming to understand it than does the carrier mechanism. Although not fully established, available evidence then is beginning to favor the fixed pore mechanism of active transport over the carrier mechanism.

Indeed, Dr. Hans Nieper's theoretical explanations of the mechanisms behind his stunning success in the clinical application of sophisticated mineral transporters rely heavily on the concept of the fixed pore mechanism of active transport. He would not be able to effectively manipulate cellular biochemistry and achieve his positive therapeutic results if he had only to rely on passive transport systems. He could not re-balance the mineral (and hence enzyme) biochemistry of the cell without the aid of an energetic active transport system that could move a substance across a membrane from one side to the other regardless of the concentrations of the substance on either side of the membrane. Thus active transport systems are able to accumulate material on one side of a membrane at concentrations that are many times those of the material on the opposite side of the membrane.

# CHAPTER 3

# A BASIC REVIEW OF INTESTINAL ANATOMY

Although each cell carries on a number of physiological processes, it is more common for groups of cells to cooperate for the benefit of the organism as a whole, rather than simply to function for the satisfaction of their own individual needs. Groups of cells that are similar in structure, function, and origin, and that are bound together with varying amounts of intercellular material, are referred to as tissues. There are four primary tissues in the body: epithelial, connective, muscular, and nervous. It is these four tissues that join together to form the organs of the body. Our focus here will be on epithelial tissues, for it is at the brush border of the epithelial cells lining the intestine that our investigation of mineral bio-utilization will begin.

## EPITHELIAL TISSUES

Epithelial tissues, like those of the gastrointestinal tract, are formed of cells that are very closely joined, with only a minimum of intercellular material between them. Neighboring epithelial cells are attached to a common basement membrane like tract homes that share the same backyard rear fence. In general, epithelia serve as coverings for most of the free surfaces of the body, both internal and external. As such, they form the outer layer of the skin, the lining of the digestive tract, the lining of the body cavities, the lining of the blood vessels, and glandular ducts and tubules. In addition, some epithelial tissues are incorporated into the various glands. With such a variety of locations, it is not surprising that epithelial tissues have diverse functions. For instance, the epidermis, or skin, forms a protective barrier between our internal biochemical events and the external environment. The epithelial linings of the internal body cavities are involved in the absorption of materials into the body, the excretion of waste materials, and the secretion of special products.

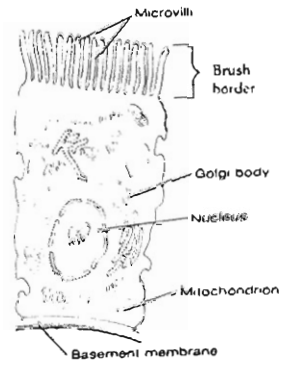
The free surfaces (surfaces not touching against another cell) of the epithelial cells that line the body cavities and the blood vessels are smooth. But the epithelial cells of the intestine have their free surfaces folded into tiny projections called microvilli (F.7). Because microvilli greatly increase the free surface area, they are ideally suited to locations where absorption and secretion are the main activities, such as in the lining of the digestive tract. Before the electron



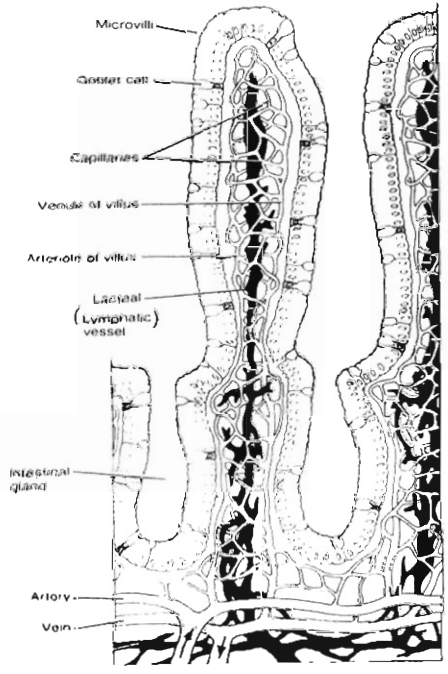


microscope improved our knowledge of their structure, these minute projections were identified as brush borders.

The surface area of the intestine is increased by villi which are finger-like projections of the intestinal lining that reach out into the lumen (space inside the tube) of the intestine. The surface of each villi is formed by a single layer of epithelial cells with microvilli on their free surface (F.8). The villi themselves are so small, so numerous and so close together that the inner surface of the intestine has a velvety appearance (F.9).




F.7 Diagram of an epithelial cell showing structures that are visible with the electron microscope.



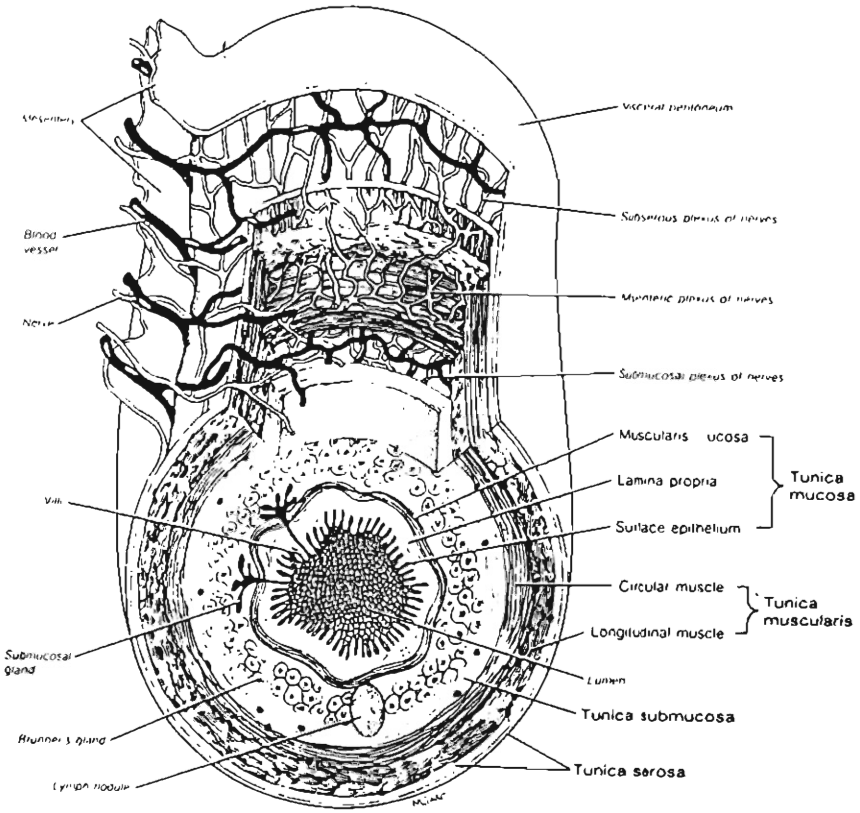

F.8 Section through villi of the small intestines showing blood vessels and lacteals (lymph ducts).

You have also probably heard the velvety lining of the small intestine referred to as the intestinal mucosa. The term derives from the mucus-secreting goblet cells which are interspersed among the epithelial cells covering the villi. The mucus protects the delicate villi from abrasion by passing food material and from chemically induced damage from those same contents. The mucus also keeps the intestinal lining and membranes moist while providing lubrication for the passage of the food.

Nutrients liberated during digestion of food, or delivered from dietary supplements are snagged by the villi and microvilli of the intestinal mucosa and carried (transported) by either passive or active transport across the epithelial cell membrane, through the cell's cytoplasm center, and out

across the basement membrane to be delivered into either a lymphatic vessel or a minute artery at the center of each villi (F.8).

Having a basic understanding of epithelial cells, membrane transport, and delivery of nutrients through the basal membrane into the circulation via blood or lymph will make clear the discussions to follow in chapter 4.






# CHAPTER 4

# BIOLOGICAL UTILIZATION

In the past, the orthodox biochemical viewpoint has equated "biological utilization" of a trace element to its absorption. Yet true biological utilization involves not only absorption, but also transportation and delivery to the tissue in need. Once a mineral is absorbed, it must be carried by the blood to the cell. Once at the site of the cell, the mineral must be transported across the cell membrane in a form which can be utilized by the cell. The three features of absorption, transportation, and utilization make up a more complete concept of biological utilization.

Perhaps because precise technology and testing methods have been lacking, accurate information about transportation and utilization of minerals is also lacking. Most of our understanding of biological utilization is based only on studies of absorption and the appearance of a trace element in the blood after its consumption in the diet — not the relative rate or efficiency of uptake by a specific tissues.

The currently predominant view of biological utilization, focused as it is on absorption, maintains that the most utilizable form of a mineral will be that mineral form which is most soluble. By soluble,what is meant is that the mineral can be easily broken apart from its transporter agents during digestion to release a mineral ion which is dissolved in the water of the intestinal contents.

The most biologically utilizable mineral supplements would then be the ones that released their mineral ions the fastest, according to this orthodox view. If we consider calcium ion as an example, calcium lactate is much more easily dissolved and ionized than would be calcium carbonate, or bone meal. What this would suggest, then, is that calcium lactate would be more biologically utilizable than would bone meal or calcium carbonate. This is because the calcium ion $Ca^{++}$ is more readily transported across the membrane of the intestinal epithelial cells than would be less easily dissolved calcium carbonate or calcium in bone meal forms. The epithelial cells, you will recall, line the intestinal wall. These cells then release the calcium across the basement membrane into the blood once it has been transported in a soluble form across that membrane.

Where does vitamin D fit into this picture of calcium absorption? Vitamin D is changed by the liver to 25-hydroxy vitamin $D_3$. 25-hydroxy vitamin $D_3$ is then

changed in the kidney to 1,25-dihydroxy vitamin $D_3$, the active form of the vitamin. It is believed that 1,25-dihydroxy vitamin $D_3$ then tells the nucleus of the epithelial cells lining the intestine to manufacture the calcium-binding protein that goes into the cell membrane of the intestinal cell to capture the ionic calcium and bring it back. Not only must all the above chemical activity take place, but the parathyroid gland begins the entire process by "keeping tabs" on serum calcium concentration. When it drops too low, the gland secretes parathyroid hormone which tells the liver and kidney to convert vitamin D to its active form.

The above explanation of the first step in biological utilization, how calcium is absorbed, seems neat, orderly, and well-understood. But looking back carefully at the above, you will notice the phrases "would be...according to the orthodox view," "yet tested," "presumably," and "It is believed." These words qualify our understanding of the true mechanism by which calcium is absorbed. We presume that vitamin D causes the epithelial cell to make a carrier or transporting protein. That carrier is called calmodulin but has not yet been definitely characterized. And we lack certainty about how the epithelial cell shuttles the calcium across and into the blood serum. Mystery still surrounds calcium cell membrane transport with many clues unresolved. Similar mysteries surround the other minerals. The mechanisms are not yet completely understood as you will discover in upcoming volumes of this series.

## ALTERNATIVE VIEWPOINTS

The type of absorption described above, where a carrier protein transports the mineral across the intestinal lining and eventually into the blood, may or may not be active transport. Nevertheless, something present in the membrane literally captures the mineral much as a big game hunter may seek out and capture a tiger by throwing a net over the animal. In the case of zinc, the transport substance picolinic acid has recently been suggested. Picolinic acid is currently the focus of spirited scientific debate as to whether it is indeed the primary "big game hunter" for zinc, and possibly for other minerals as well (such as manganese, iron and copper). At this time, however, some investigators only believe that it can serve as an efficient transporter for zinc.

There are also other ways that minerals can be absorbed. Mineral ions are so highly reactive in the chemical sense, that some may actually penetrate the cell membrane of the epithelial cells simply by physically colliding with the cells at the brush border. It is thought that the ion either creates an opening in the cell membrane and passes through, that it is magnetically drawn to a charged mucopolysaccharide component of the cell wall, or that it is attracted and drawn through a pre-existing and electrically charged portal in the membrane (the fixed pore mechanism theory, Ch. 2).

## AMINO ACID CHELATION

There is also evidence that amino acids released from the digestion of proteins may combine with or "chelate" the mineral ions and be transported into the epithelial cells lining the intestine. As the amino acids pass through the membrane, they carry with them the atom of mineral which had chemically bound itself to the amino acid.

Radioactive isotope tracing methods and statistical analyses of relative absorption and inhibition of the different amino acids, has helped establish a case for the belief that there are specific sites or loci on the membranes of epithelial cells lining the intestine which permit the penetration of specific amino acids or types of amino acids. For example, there is a phenylalanine and tyrosine locus. There is a locus for basic amino acids and one for neutral amino acids. Methionine seems to be able to enter all amino acid loci.

Along the intestinal lining, there are regions where cells with a preponderance of loci for a single amino acid or type of amino acid will be grouped together. At that point along the intestinal lining a major absorption site for that amino acid or group of amino acids is said to exist.

The theory behind the classification of amino acid chelated minerals as important supplements for the promotion of mineral absorption and biological utilization is based upon the principle that the mineral chelate of an amino acid will be readily transported across the intestine into the blood through amino-acid-preferring loci on the membranes of the epithelial cells. Animal tissue studies seem to show that these loci for amino acids (with minerals embedded in them to form chelates) exist not only on cells lining the intestine, but also on cells throughout the body.

Amino acid chelates of specific minerals are useful in neutralizing the ionic charge of the mineral. Once this electrical charge is neutralizer-balanced, it allows for more rapid absorption of the mineral across the intestinal epithelial cells. The actual charge (polarity) of a chelate depends upon the type of substance that is used in the formation of the chelate. For instance, lysine will make a chelate with a positive charge whereas glutamic acid will form one with a negative charge. Other chelates are neutral in charge; therefore, chelates can be positive, negative or neutral. Nevertheless, the amino acid joins with the ion to form a new stable compound, and has different transport properties than the mineral ion by itself. Whereas mineral ions on their own that are positively charged may actually be repelled or resisted by positively charged cell membranes, chelates with neutral or negative charges may approach new sites for absorption on the intestinal surface. In effect, the cell perceives the compound as an amino acid which has absorbed a mineral rather than just as the mineral itself.

This different acceptance of an amino acid chelate by cell membranes implies that mineral absorption may be enhanced through the use of such chelates. Furthermore, the existence of specific amino acid loci on the membranes of cells throughout the body carries the additional potential that chelates (or aminoates, as I prefer to call them) may be more readily accepted by cells in body tissues

Animal and human tests have, as yet, been inconclusive in demonstrating unequivocally the enhanced biological utilization of amino acid chelates over

other forms of trace elements. Results from one group of researchers has too often differed from those of another group. For example, testing conducted in the laboratory facilities of a major international manufacturer of amino acid chelates for animal supplementation shows greatly enhanced uptake in the organs and tissues of the bodies of the animals tested. Chelates consistently boosted tissue levels of the mineral examined. Percentage of increase in tissue mineral stores would range from a few percent to several thousand percent improvement over levels produced by the conventional oxide, gluconate, or lactate forms of the mineral.

Other researchers, however, have been unable to unanimously duplicate the findings of the chelate manufacturer. Some indeed find improved absorption such as with iron EDTA versus ferrous gluconate, but are unable to confirm changed tissue levels. Others show such minerals as magnesium to be better absorbed than conventional forms when chelated and to produce higher tissue levels. When evaluated as a whole, the existing research on the absorption and biological utilization of amino acid chelates is, at best, inconclusive.

It is reasonable to assume that the inconsistencies in the results of the research can be traced to a number of variables that have not been adequately controlled. Although the methods of conducting radioactive isotope testing (of the type used to trace the mineral nutrients) are well established and can be precisely controlled by trained laboratory personnel, the quality of the amino acid chelates being tested may be questionable and nearly impossible to control.

The manufacture of amino acid chelates is difficult. Temperature, acidity of the reactive solution, time permitted for the reaction to take place, the method of blending ingredients, the sources of the ingredients, the skill of the manufacturing chemist, and even the "wetness" and trace mineral content of the water used to dissolve the ingredients will all affect the quality and purity of the finished product. Highly mineralized "hard" water can interfere with the chelation process, blocking the efficient combination of mineral with amino acids to some degree, and thereby resulting in a finished product with an elemental chelated mineral content lower than desired. Artifacts of trace mineral chelates would be present that could interfere with the results of any laboratory testing.

Chelates that were produced too quickly may present so little of the mineral as an actual chelate that they will perform in testing as if they were never chelated at all. For example, calcium gluconate or lactate may be employed as the starting material. These forms of calcium would then be added to the acidic chelation solution. If the reaction were terminated too quickly, and the material removed, washed and dried, only a small portion of the calcium in the finished product would be chelated. The remainder would be present in its original gluconate or lactate form. If material of such low quality were then to be used in an absorption or biological utilization study, the results would probably mirror those of simple calcium gluconate or lactate. Thus we can see that a valid piece of scientific research must be conducted using the highest quality amino acid chelates produced by skilled professionals.





Unfortunately, researchers have no technique to determine the absolute portion of their test material which exists as a true chelate. Chemical analysis of the material can only measure the total amount of the mineral and protein in it, but cannot prove that the mineral is actually bound to the protein. Advances in laboratory technology are being made, however. We can expect a definitive laboratory analysis technique to be developed in the near future. Until then, our belief in the quality of any amino acid chelate must be based on the integrity and skill of the manufacturing chemist. The rudimentary laboratory analyses available today can only uncover grossly inferior amino acid chelates.

Even the best chemists, too, believe they can deliver a finished chelate which will supply only up to 60% of its mineral in chelated form. The manufacturing technique has simply not yet been developed which can overcome the minerals' and amino acids' resistance to complete bonding of 100% of the reagents. The best technique of the day, skillfully orchestrated by the most conscientious chemist of the day, may still only deliver 60% of the mineral content in true chelated form. The remaining 40% or more will remain in its original form. Referring back to our example above, that 40% would be as calcium gluconate or lactate.

To move one step further toward understanding chelates, it is important to realize that in any quantity of amino acid chelated material, most of the weight or, more accurately, mass of the substance, will be provided by the protein content. Typically, only 2%, 6%, 15%, or possibly 20% of the mass will come from mineral in the material. The percentage mineral in the total weight of the material depends on the atomic weight of the mineral, the ionic valence (electrical charge) of the mineral, the production techniques and source materials used.

**TYPICAL PERCENTAGE OF RAW MATERIAL MASS PRESENT AS ELEMENTAL MINERAL IN HIGH QUALITY AMINO ACID CHELATES**

| | |
|---|---|
| Calcium Aminoate (20%) | Manganese Aminoate (12%) |
| Chromium Aminoate (2%) | Molybdenum Aminoate (0.1%) |
| Copper Aminoate (6%) | Potassium Complex (20%) |
| Germanium Aminoate (0.01%) | Selenium Complex (0.1%) |
| Iron Aminoate (10%) | Vanadium Aminoate (0.1%) |
| Lithium Complex (0.1%) | Zinc Aminoate (15%) |
| Magnesium Aminoate (20%) | |

To summarize, in a given amount of amino acid chelated mineral powder, not more than 20% of the material by weight can be mineral — often referred to as "elemental mineral." Of that maximum 20% mineral, up to 60% of it may be amino acid chelated. Commerical supplies of amino acid chelates widely used in nutritional supplements today typically range from 0% to 30% of their elemental mineral in true chelated form. (Yes, 0%. Unfortunately, there are still quite a few bogus chelates sold in the marketplace.)

The inability to produce a finished material with 100% of its elemental mineral as a true amino acid chelate or "aminoate" undoubtedly compromises any opportunity to achieve consistency in bio-utilization studies. Simply too much of the mineral is available in its original, non-chelated form. The presence of a relative abundance of the original mineral form perverts the findings of the research.

Researchers are also inconsistent in their selection of material suppliers. One research team may test an iron amino acid chelate from supplier X, believeing it to be representative of all iron chelates, and another team may select their iron aminoate from supplier Y, but only 35% of the elemental iron in X's aminoate may be chelated while 60% of the elemental iron in Y's aminoate may be chelated. Obviously, Y's aminoate is of higher quality. It would not be surprising to find that the biological utilization or absorption as measured by the two research teams would be quite different. In addition, the actual amino acids used, their ratios, sources, and the methods by which they were liberated from their food source can also bear on the ultimate absorption and bio-utilization of the finished product.

It is no wonder, then, that world leaders in preventive and wholistic medicine who employ various aminoates or other more specialized mineral transporters in their therapeutic programs tend to remain loyal to nutritional products from manufacturers who are able to consistently produce stable, high quality mineral transport compounds.

Dr. Hans Nieper of the Silbersee Hospital in Hannover, West Germany, went so far as to patent a specific production method for creating a type of chelate called orotic acid chelates, or orotates. He helped finance and establish a quality manufacturing laboratory in Germany so that he would have continued access to mineral orotates of consistent quality. Nieper's production methods are now being duplicated by manufacturing plants in Japan, the United States, and even in Germany and other European countries. Dr. Nieper has chosen not to enforce his patent because he has little interest in the commercial aspects of the business, and wishes to devote his time and energy to his patients, writing, and research.

It has been Dr. Nieper who has spent a considerable amount of time developing theories related to mineral transportation and the important role that chelates play as more biologically utilizable forms of minerals. Dr. Nieper has not been reticent to educate his fellow physicians about his work. He has spent many months travelling internationally to lecture to professionals and the lay





public alike, concerning the medical successes that he has enjoyed with his patients, utilizing his orotate and other mineral chelate therapies. He has developed a very intricate explanation of the way he believes the orotates, as partates, amino-ethanol phosphates, and other chelates work in improving cellular absorption and utilization of trace elements. It is his adherence to these theories that allows him to effectively manipulate cellular biochemistry.

Backed by his own clinical successes with patients, his training in cellular biology, and the information from a group of European research centers in Zurich, Switzerland; Dresden, East Germany; Hamburg, West Germany, and Paris, France; Dr. Nieper has formulated concepts explaining how his mineral transport therapies may work. The modern hypotheses of cell-mediated "fixed pore mechanisms" of active transport discussed earlier, and his belief that there exist loci on cell membranes (both the outer and inner membranes) for the acceptance and transport of specific amino acid chelates, has been used to explain the mode of selectivity of orotate and other chelates developed by Dr. Nieper.

In his clinical work, Dr. Nieper and his staff have employed several different organic mineral compounds which are specialized chelates and are made up of not only orotates, but also aspartates and aminoethanol phosphate. Dr. Nieper has postulated that these sophisticated mineral chelates have selective abilities to deliver mineral ions to specific areas of the cell which have absorptive loci that respond only to selective chelates or proteinaceous compounds.

His theory is a concept of biological utilization built upon a model of mineral absorption and transport. It implies that the biological utilization is dependent upon the composition and form of the mineral compound delivered to the cell. This theory brings to the concept of bio-utilization the added dimensions of transport across the cell membrane and specific delivery of minerals to cells in forms that can be used. In short, the theory suggests that the mineral, when chelated by an appropriate amino acid substance, is delivered to the intestinal epithelium where it is transported either by passive or active transport into the blood as an intact unit. That unit then is delivered to the liver where it is either reformulated into a new mineral chelate substance or is released directly to the blood. From the blood it is taken up by cells in tissues via their own specific transport mechanisms at the cell membrane level. This selective uptake and delivery of trace elements, then, can enhance entry of minerals into specific locations in cells and tissues.

The theory is very glamorous and is built upon reasonably contemporary concepts of cell membrane physiology and biochemistry. It, however, yet lacks definitive verification. The theories require exquisitely detailed biochemical proofs which are in the throes of being developed. As is often the case with new theories, Nieper's are born out of clinical observation.

Dr. Neiper and his colleagues report magnificent achievements in the clinical management of patients with a wide variety of disorders by utilizing specialized chelate substances. They were not able to realize similar successes when they were using other mineral forms such as gluconates, lactates, or standard in-

organic forms of the element. Only when they switched to the selective transport agents did they begin to witness remarkable improvements in recovery of their patients.

From these observations Dr. Nieper then spent much time developing the theories of bio-utilization for his mineral substances, showing how they related to contemporary membrane physiology. It is postulated by Dr. Nieper that the minerals are released at the tissue site of need by interacting with a specific receptor site in the cell membrane or organelle membrane. The membrane then transports that mineral selectively, thereby improving cellular function. The ions released at or in the membrane (and then transported) are available to partake in and stimulate any number of important biochemical activities in the specific regions of the cell most receptive to a particular, specialized mineral substance.

By carefully selecting the mineral and its organic transporter, Dr. Nieper has suggested that he can manipulate the biochemistry of the cell to return it to a more normal state from an unbalanced or diseased state (where the cell may be overly acidic). This exciting theory deserves extensive research. It might pull together a wide variety of observations made by investigators the world over. Their observations concerning the specific role of various sophisticated trace element compounds and the improvement of human health has previously gone unexplained from lack of a central theory.

Neiper's assumptions are not his alone; it is known, in the food processing industry, that amino acid chelates of a variety of minerals are more stable and more biologically utilizable than unchelated forms of the element. For instance, iron, when chelated to an amino acid, can resist conversion to ferric iron which is both unabsorbable and would form in the gut if not bound to a transport agent.

Dr. Nieper has found that this concept of specific amino acid chelation and delivery of the trace elements seems applicable not only to the heavy elements such as manganese, chromium, and copper, but also to the lighter alkaline minerals such as calcium and magnesium. Large doses of conventional forms of calcium such as the lactate and gluconate, he observed, only succeeded in creating high blood calcium levels (hypercalcemia), whereas utilizing equal amounts of calcium orotate returned blood calcium levels to normal and alleviated symptoms in patients suffering from low tissue calcium.

Since the time that Dr. Nieper has advanced his theories, many other medical investigators and the sanctioning bodies in the Federal Republic of Germany have come to acknowledge that magnesium, potassium, and calcium aspartates; calcium and magnesium aminoethanol phosphates; and the mineral ascorbates and other mineral orotates all are able to participate in unique delivery mechanisms of trace elements to specific tissues in human subjects. Nieper and other research physicians such as Drs. Blumberger and Köhler believe that they have shown that aspartates have an affinity for and travel to the inner layer of the outer cell membrane, whereas the aminoethanol phosphates deliver their ions at the outer layer of the outer cell membrane, and the orotates pass right through the outer cell membrane and travel to the





cytoplasm of the cell and into cellular organelles. Other clinicians have suggested that mineral ascorbates are readily accepted by all outer and inner membranes and the cytoplasm of cells. They are suggesting that there is a differential absorption and transport of various mineral chelates which can effect different clinical outcomes in patients.

A recent theory also proposes that the mineral lysinates, which are mineral ions chelated to the amino acid lysine, will be most effective at delivering the mineral inside the cell mitochondria, the tiny energy centers of the cells. Lysine is known to be the precursor of the amino acid carnitine which is responsible for carrying fatty acids into the mitochondria to be used as a source of energy. Since 70% of our energy — under aerobic conditions — is derived from the burning of fatty acids, then it is quite possible that the stimulation of carnitine synthesis in the body from lysine supplementation may improve energy function — and this may explain why mineral lysinates seem to enhance cellular activity. The minerals may even "tag along" with the lysine component in the newly synthesized carnitine, thereby being efficiently delivered into the mitochondria.

It should be stressed that all of these theories are still considered hypotheses, and have not been proved by controlled studies to be unequivocally correct; nor are they accepted by all researchers in the field of mineral metabolism. These theories, taken as a central theme however, allow explanation of many clinical observations that have been made by hundreds of investigators over the past several decades. They are an attractive new way of explaining specific mineral activities in human physiology. As with all theories, their value is only as good as their clinical applications — applications which must be successfully and consistently reproduced.

The proof through reproducibility awaits further study; however, the study of the activity of these amino acid chelates is dependent upon the quality and purity of the products that are produced and evaluated. Many studies that have been done in the past were done on material of high impurity or very low content of true chelate. The negative results from these studies may have been more a result of poor quality product than of a lack of efficacy inherent in the amino acid chelate. This exciting area of investigation is now challenging contemporary research investigators in the biochemistry and nutrition fields, and is promoting the development of new research methods and exploratory tools. The emerging understanding of how cell membranes work in the uptake of nutrients and the release of waste products is allowing better understanding also of how specific mineral transporters may operate in cellular systems. No one knows, with absolute certainty, whether mineral transport compounds such as orotates, aspartates, lysinates, picolinates, or aminoates are absorbed intact or are broken down in the gut, only to recombine in the bloodstream like two lovers walking hand-in-hand in the woods who must let go of one another to climb over a fallen tree blocking their path, only to rejoin hands on the other side.

Whatever is truly happening at the cellular level, however, it appears as though there is a difference in absorption and biological utilization of various

mineral chelate substances. Dr. Nieper points out that atherosclerotic arteries have become more elastic when magnesium orotate is a part of the therapy, and bone recalcification seems to be enhanced by calcium orotate, presumably because of its superior ability to penetrate cell membranes. Nieper has also used zinc aspartate or orotate in his clinics to help boost "immune defense".

For athletes and sports-minded individuals, one of the most interesting observations is that muscular fatigue and the lack of oxygen in tissues after exercise may be partly overcome by the administration of potassium-magnesium aspartate. This particular material seems to enhance the production of ATP which is the energy-giving substance in all muscle cells. The interpretation of these findings is that potassium-magnesium aspartate is transported to the inner layer of the outer cell membrane and there activates enzymes which are responsible for the formation of ATP. Again, evidence is in conflict over this issue. Different teams of investigators are both proving and disproving that supplementation in submaximal exercise with aspartates improves exercise tolerance. The challenge is certainly before us to critically evaluate this hypothesis.

Continuing to list applications for mineral transporters could fill many more books. Similar applications of more conventional nutrients are also enumerated in the scientific literature. What the research and clinical results strongly suggest is that the mineral transporters are either more effective or the only effective forms of the nutrients in a number of both preventive and crisis intervention applications.

Work is underway now at a number of leading research institutes, not the least of which are the Linus Pauling Institute and Utah State University, to try to identify with precision which forms of minerals are most bio-utilizable. We hope healthy as well as diseased subjects will be tested, for a disease state may alter the manner in which an animal or human absorbs, transports, and utilizes a nutrient. Equal bio-utilization of a conventional mineral versus a mineral transporter may change dramatically under conditions of disease, yet it seems unlikely that superior utilization of the transporter forms would only become obvious and measurable when "the going gets tough" and normal absorption and metabolism is impaired by disease. Our understanding of human biochemistry would imply that a nutrient more easily utilized under conditions of disease would also be more readily accepted in states of health.

If the evidence that minerals can be targeted to specific parts of the cell can be proved for the different transporters, then a special combination of mineral transport compounds could be formulated which would deliver thorough mineral nutrition to all parts of the cell. The implications for health and longevity would be enormous. A properly nourished cell would not undergo the debilitating stresses associated with momentary or chronic mineral imbalances. The cell would continue to live out its life in metabolic harmony with youthful vigor. Since our human life and vitality is the sum of the lives and vitality of our trillions of cells, we could expect to express that cellular vitality through the whole of our beings. Mineral transporters as part of a non-therapeutic, nutri-





tional maintenance program of supplementation may be one more key to unlock that vitality.

# GLOSSARY

basement membrane - a thin layer of biological material to which epithelial cells are attached in mucous membranes

cytoplasm - the colloidal complex of protein, water, organic and inorganic substances within a cell which surrounds and suspends the nucleus and organelles, filling the space bounded by the outer cell membrane

homeostasis - the state that exists when body organs function together harmoniously to create and sustain a stable internal environment for the well-being of the body; a state of equilibrium

ion - an atom or group of atoms that carries a positive or negative electric charge as a result of having lost or gained one or more electrons

ischemia - localized tissue anemia and usually hypoxia (oxygen starvation) due to obstruction of the delivery of nutrient-rich arterial blood

jejunem - the part of the small intestive approximately 1.5 meters long, coming after the duodenum and before the ileum; the upper third of the small intestine

ligand - a substance such as an atom or group of atoms (molecule) that bonds itself to another

lipid - any of various substances including fats, waxes, phosphatides and related compounds that are virtually insoluble in water but highly soluble in fat and fat solvents; fats and fatty acids

metabolism - the sum of the processes involved in the building up and breaking down of cells and tissues, i.e. of the chemical changes in living cells by which energy is provided, new material is produced, repair is completed and waste is excreted; the total of the chemical reactions in the cell or body

molecule - a very small mass of matter made up of two or more atoms bound together as a unit

neurological - of or relating to the nervous system

phospholipids - phosphorus-containing lipids which are characteristically major components of cell membranes

plasma - the fluid portion of blood or lymph

rectum - the short, downward portion of the large intestine leading to the anus

renal - pertaining to the kidney

serum - the amber, watery liquid which remains after coagulation





# APPENDIX

*Previews of*

*Upcoming Volumes*

The following copy has been selected from forthcoming volumes of *Mineral Logic* now in progress.

CALCIUM: Ca: common ion $Ca^{++}$

| Vital Statistics: | 1980 U.S. RDA | 1973 U.S. RDA |
|---|---|---|
| Children | 800 mg | 800 mg |
| Males 4 to adult | 1200 mg | 1000 mg |
| Females 4 to adult | 1200 mg | 1000 mg |
| Pregnant and Lactating | 1600 mg | 1300 mg |

*Note:* The latest (1980) Recommended Daily Allowances put forth by the National Academy of Sciences have never been adopted by the Food & Drug Administration for use in the labeling of nutritional supplements. For labeling purposes, the 1973 U.S. RDA figures still apply.

Normal (Average) Daily Intake: 600 mg
Absorption: Children - up to 75% of ingested Ca
Adults - 10% - 30% of that ingested

*Note:* Given the statistics above, it is no wonder that the USDA Household Food Consumption Survey, Ten State Nutrition Survey, Health and Nutrition Examination Survey and the USDA Nationwide Food Consumption Survey all identified calcium as a nutrient at risk. An absorption of 60 to 180 mg per day falls far short of the 1000 mg U.S. RDA. A good deal of the latest research into calcium nutriture is beginning to call for an even higher RDA of something closer to 1500 mg per day.

Typical Daily Excretion: In feces 250 mg; in sweat 20 mg to 350 mg to 1000 mg in hot climates under heavy labor; in urine 80 to 250 mg.

*Note:* With possible daily calcium losses ranging from 350 to 1500 mg per day, even an excellent absorption percentage of 30% would mean that 1167 to 5000 mg of calcium must be ingested to balance daily losses.

Food Sources: Dairy products - milk, cheese, yogurt, buttermilk, sardines, eggs, green vegetables, beans, peanuts, sunflower seeds, walnuts

Protagonists (helpers for absorption or utilization): Lactose (milk sugar), moderate exercise, Vitamin D, hydrochloric acid (maybe), magnesium, phosphorus, exposure to ultraviolet light, lysine, arginine

Antagonists: Too much magnesium; too much phosphorus; too little Vitamin D; excessive, vigorous exercise; phytic acid (from wheat and other grains); high fat intake; high sugar intake (maybe); cocoa, soybeans, kale; spinach and other oxalate rich foods; hexaphosphoinositol from brown rice; rich polish; bran and whole wheat; sedentary lifestyle; stress; thyroid hormone and medication; cortisol medication





## DISCUSSION

The importance of calcium cannot be overstated. It is the most abundant positively charged ion (cation) in the human body and the fifth most common inorganic element. The average adult human holds 1000 to 1200 grams (1.2 kilograms) of the mineral in his or her fat-free tissues. 99% of calcium is retained in bone tissue while the other 1% circulates in blood plasma and soft tissues. The hormones calcitonin from the thyroid gland and parathyroid hormone from the parathyroid gland help maintain plasma calcium levels at ±3 percent throughout the day. Calcitonin causes the deposition of calcium in bone while parathyroid hormone liberates it from bone.

Calcium absorption, deposition, and utilization is not solely controlled by hormonal factors, however. Dietary intake of phosphorus, exposure to sunlight, protein, fat and sugar intake along with exercise all affect calcium status. (The role of these factors and others will be examined more fully in the forthcoming volume of this series devoted to calcium.)

The question of providing adequate calcium nutriture and utilization is made difficult by the control mechanisms of the body. For instance, starvation or calcium deprivation causes uptake to become more efficient! The less you take in, the higher the *percentage* of absorption. Conversely, the more you take in, especially in a single dose, the smaller the percentage of absorption. Calcium appears to be a supplement then that is less effective if taken in single mega-dose amounts. Total daily absorption might then be enhanced by taking smaller doses spread throughout the day.

Disguising the calcium ion through binding to aspartic acid, ascorbic acid, orotic acid, lysine or some other organic ligand that by itself is readily accepted into the epithelial cells lining the intestine may further improve absorption. This basic concept lies behind the therapeutic use of aspartates, orotates, ascorbates and lysinates in Europe, and behind the applications of those same compounds as nutritional dietary supplements in the United States. The goal in both cases has been to improve absorption and bio-utilization by the cells.

Bio-utilization of calcium, which encompasses absorption, transportation into and utilization by the cells, can be enhanced through moderate exercise. In response to the stresses of exercise, calcitonin is secreted by the thyroid gland and stimulates the deposition of calcium in bone. Heavy, vigorous, sustained exercise as might be seen in a world class athlete, on the other hand, can cause a net loss of calcium from the body and a weakening of bone and connective tissue. This problem is most evident in female athletes whose intense training schedules may create hormonal imbalances characterized by amenorrhea and lowered estrogen levels. Since estrogen is a skeleto-protective hormone stimulating deposition of calcium in bone, lowered levels may lead to a net loss of calcium from the bones of female athletes. Careful diet management and supplementation

would be required under such conditions. We will look more closely at how exercise affects calcium metabolism in the upcoming volume on calcium, and we will also evaluate current evidence which seems to suggest that a strong acid medium in the stomach may be unnecessary for the efficient absorption of the mineral.

The long-standing belief that an acidic environment enhances calcium ionization and, therefore, absorption has not been borne out in human studies. Volunteers who had their digestive tracts purged and alkalinized through special medication, demonstrated a nearly uncompromised ability to continue absorbing calcium at normal levels.

From the brief discussion in this overview volume of the series on minerals, we can identify the salient points on calcium as follows.

Calcium is a mineral that is generally undersupplied in the American diet. The daily absorption and delivery to the cells is not enough for most persons to compensate for the daily loss of calcium in feces and perspiration. Regular exercise may either aid or aggravate the situation depending upon the intensity of the exercise program and the type of diet and supplementation, if any, styled around the exercise.

Vegetarian diets rich in grains and soybeans can inhibit absorption several ways. Phytic acid from grains, oxalic acid from spinach and inositol hexaphosphate from brown rice and whole wheat can bind with calcium ions in the gut to form unabsorbable compounds. Vegetarian diets tend to be so heavy in fiber that transit times of food through the G.I. tract are relatively brief compared to the optimal residency period for maximum calcium absorption. Food rushes through too fast to permit thorough absorption of calcium. And if acidity in the stomach and of the contents leaving the stomach is important, then vegetarians tend to be hypochlorhydric (low in acid production). The low protein vegetarian diet does not stimulate copious acid production by the parietal cells of the stomach.

One must be careful though to not assume that if low protein vegetarian diets inhibit absorption, then high protein diets should be ideal. On the contrary, an excessively high protein diet can actually cause a net loss of calcium from the body. The abundant phosphorus and sulfur released from the protein acidify the blood through the delivery of phosphate and sulfate ions to the serum. The parathyroid gland responds to the acid challenge by sending parathyroid hormone into circulation. The hormone stimulates osteolysis - the breakdown of bone tissue - to release calcium into the blood. The calcium combines with the acidic phosphate and sulfate ions, neutralizing them, and returning the blood pH to normal. Most of the calcium phosphate and calcium sulfate is then excreted in the urine.

A moderate diet, free of extremist ideological or philosophical influences appears best then to promote not only adequate calcium uptake, but also to promote optimum health in general. Complex carbohydrate in the form of fresh





vegetables with some whole grains should supply most of the calories, probably between 50% and 60% of them. 20% to 25% of the remaining calories would then come from animal proteins with cold ocean fish earmarked as the preferred form. Some cultured dairy products including cheeses would help fulfill the protein calorie requirements. Lastly, fat should be held to approximately 20% of dietary calories.

Though lipids are essential to life itself, the American habit of taking in more than 40% of dietary calories as fat has not only unbalanced the diet, but unbalanced the health of the country in general. In very broad terms, excessive fat intake is implicated in a host of degenerative diseases including (but not limited to) cancer, heart disease, diabetes, autoimmune diseases, arthritis and other inflammatory disorders.

More narrowly, and focusing on our topic of calcium, excessive fat in the diet can impair calcium absorption by coating the intestine and physically blocking the mineral's ability to penetrate epithelial cell membranes. Fat can also join with calcium ions in the gut to form new compounds which are, technically speaking, soap. The resulting bubbling fatty diarrhea is called steatorrhea, where minerals are simply bound in soap-like compounds and flushed from the body. The advantage of a lower fat diet as it relates to calcium absorption and the absorption of other minerals is evident. Let us now look at some of those other minerals.

PHOSPHORUS: P: common ions $H PO_4^{-2}$, $H_2PO_4^{-}$

| Vital Statistics: | 1980 U.S. RDA | 1973 U.S. RDA |
|---|---|---|
| Children 1 - 10 | 800 mg | 800 mg under 4 yrs |
| Males 11 - 18 | 1200 mg | 1000 mg, 4 to adult |
| 19 and older | 800 mg | |
| Females 11 - 18 | 1200 mg | 1000 mg, 4 to adult |
| 19 and older | 800 mg | |
| Pregnant and Lactating 11 - 18 | 1600 mg | 1300 mg |
| 19 and older | 1200 mg | |

Normal (Average) Daily Intake: 1000 to 1500 mg (1 - 1.5 grams)

Absorption: 60% to 70% at normal levels of intake
up to 90% at very low levels of intake

Typical Daily Excretion: 200 mg in feces
667 to 1000 mg in urine

Food Sources: Milk, poultry, fish, meats, soft drinks (phosphoric acid)

Protagonists: Vitamin D (maybe)

Antagonists: Unsaturated fatty acids, iron, aluminum, calcium

## DISCUSSION

The relationship of calcium to phosphorus is a crucially important one. For that reason, phosphorus is our next mineral worthy of mention. Analyses show there is from 11 to 14 grams of phosphorus per kilogram of fat-free tissue in the "normal" adult. This compares to 20.7 to 24.8 grams per kilo of calcium. Therefore, "normal" tissues, taciturn though they may be, seem to be telling us that they need more calcium, approximately 1.7 to 1.9 times the amount of phosphorus.

But if, in the adult, calcium absorption is excellent at 30% absorption, then how much must be ingested to balance the more easily absorbed phosphorus? Examining the question from a numerical point of view only, it would appear that one would need to take in 3.9 times more calcium than phosphorus in order to supply bodily tissues with calcium and phosphorus in a ratio of 1.8 to 1, if 30% of the ingested calcium and 65% of the ingested phosphorus were absorbed.

Since phosphorus is ubiquitous relative to calcium in the diet of western man, an expected intake of 1500 mg. phosphorus in a day would imply an absorption of 975 mg. To achieve the 1.8 to 1 ratio, the phosphorus intake would have to be balanced with an intake of 5, 850 mg. of calcium, 30% of which would be absorbed to deliver 1,755 mg. of calcium. The 1.8 to 1 ratio would then be achieved. But, of course, it would be nearly impossible to eat enough cheese, drink enough milk or eat enough greens to deliver the nearly six grams of calcium. And since the calcium/phosphorus ratio in food is not 3.9 to 1, the ratio





could never be reached through normal dietary means. Supplements of calcium alone could be used, but that would be unwise. (Toxicity levels could be reached.)

The calcium/phosphorus ratios in vegetable foods are different than those in human and animal tissues. This fact points up the enormous complexity of metabolism with its checks and balances and neatly orchestrated relationships that allow for parsimonious retention of scarce and valuable nutrients and the quick expulsion of abundant or superfluous materials. The complex network of interrelationships that is biochemistry defies simply numerical analysis. Thus we find that in spite of phosphorus being relatively ubiquitous, the human body will retain more calcium. An example of the body's miserly attitude is seen in the way it stores 99% of its calcium but only 85% of its phosphorus in bone.

Phosphorus is not only more easily absorbed than calcium, it is also more easily excreted. Most, if not all, dietary phosphorus is absorbed as free phosphate ions. About 70% will remain as hydrogen phosphate or dihydrogen phosphate as it circulates in the blood. As yet there is no known physiologic mechanism regulating intestinal absorption of phosphate in man. The control and balancing of this "free agent" phosphorus is largely achieved through manipulation of dietary intake and excretion through the kidneys.

The vital statistics that introduced this section show that approximately two-thirds (67%) of dietary phosphorus is usually eliminated in the urine. Only 13% to 25% of dietary calcium is lost in a similar fashion. Yet the high protein diets of western man, with the concomitant use of a plethora of phosphorus-rich processed foods including the replacement of innumerable glasses of milk with cans and bottles of soft drinks, have overpowered the natural homeostatic (balancing, compensating) mechanisms operating within the subtle confines of human biochemistry. The abnormally high phosphorus content of the western diet has resulted in a calcium/phosphorus ratio that is too low to maintain the integrity of skeletal tissue in spite of the body's ability to compensate.

The abundance of phosphorus stimulates the parathyroid gland to secrete parathyroid hormone, a hormone that liberates calcium and other minerals from bone. Over many years of a high phosphorus diet, the deterioration of bone will become evident as osteoporosis.

From animal studies we know that ratios of calcium to phosphorus that are less than 1 to 1 will lead to bone wasting. When returned to diets supplying more calcium than phosphorus, the animals showed restoration of bone tissue. Although it is recognized that the ratio for humans should also be 1 to 1 or slightly greater, epidemiological studies estimate that the true ratio of calcium to phosphorus is 0.75 to 1. In short, chronic calcium undernutrition afflicts the western societies.

We do not begin life with the low calcium burden, however. Mothers' breast milk is 2/1, calcium/phosphorus. Cow's milk is 1.3/1. It appears then that our enjoyment of processed foods for pleasure combined with high protein intakes upset the crucial balance. Less risk would be inherent in a diet of fresh whole foods as described in the foregoing section on calcium.

This discussion is not meant to be a diatribe against phosphorus. The mineral is essential to life, but must be balanced with other nutrients such as calcium. Without phosphorus for instance, there would be no bone, teeth or connective tissue. It joins with calcium and other minerals (in smaller amounts) to form the hydroxyapatite crystal that is the rigid component of bone, and readily combines with collagen.

Phosphate ions are required for the metabolism of carbohydrate, fats and protein through their role as a part of many enzymes employed by cells to release energy from those major food components. In fact, the actual "molecule of energy," ATP, is adenosine *triphosphate*, a molecule containing *three* phosphate ions. By repeatedly giving up and regaining one of its phosphate groups, ATP completes the energy cycles inside each cell. Without phosphorus, there is no ATP. Without ATP, there is no biochemical energy, no cells, and no human embodiment. We would be left with only some plane of existence other than the physical one with which we are familiar.

As mentioned earlier, phosphorus modifies the acid-base balance of blood plasma, and affects bone remodeling both as a structural component and a stimulator of hormonal activity. It works with B-vitamins in enzyme systems, and helps secrete hydrogen ions through the kidneys. We cannot live without it, but simply need to manage phosphorus responsibility through a whole foods diet and a greater level of understanding.





MAGNESIUM: Mg: common ion $Mg^{++}$

| Vital Statistics: | 1980 U.S. RDA | 1973 U.S. RDA |
|---|---|---|
| Children 1 - 3 | 150 mg | 200 mg, under 4 yrs |
| 4 - 6 | 200 mg | |
| 7 - 10 | 250 mg | |
| Males 15 - 18 | 400 mg | 400 mg, 4 to adult |
| All other ages | 350 mg | |
| Females all ages over 11 | 300 mg | 400 mg, 4 to adult |
| Pregnant and Lactating | 450 mg | 650 mg |

Normal (Average) Daily Intake: 180 to 480 mg.

Absorption: 23% to 76% depending upon daily intake. For example, 564 mg. intake has yielded 23.7% absorption (133.7 mg. absorbed); 240 mg. intake has yielded 44.3% (106 mg.) absorption; 23 mg. intake has yielded 75.8% (17 mg.) absorption.

Typical Daily Excretion: in feces 100 to 336 mg
in urine 35 to 224 mg
in perspiration 7 mg to 8 mg

Food Sources: Nuts,. whole grains, green vegetables, cauliflower

Protagonists: Triamterene (diuretic, conserves Mg); normal transit time; lactose; whole food diet.

Antagonists: Aluminum; diuretics (furosemide, ethacrynic acid - strong antagonists; thiazides - less antagonistic); rapid transit time; high fat diet

## DISCUSSION

At the close of the discussion on phosphorus, you read how that mineral was absolutely essential to the production of energy in each cell. But the mysterious intrigue of biochemistry can't allow any single nutrient to bear entire responsibility. For as we look closely at the metabolic activity of ATP, we find magnesium nestled in the enzymes ATP must use to give up and then regain its phosphate group in the production of energy.

So we are left with a "chicken-and-the-egg" story. Which came first, or rather, which mineral is most important to energy production, phosphorus or magnesium - or are the protein and vitamin components of the enzymes most significant? The answer is: none and all. None is more important than the other and all are needed, equal in importance. We find this same thematic fact of vital interrelationships running throughout all investigations in biochemistry.

We find magnesium not only vital to energy production, but also needed for protein synthesis through its action on ribosomal aggregation and the binding of RNA to them. It is also necessary for the degradation of DNA, and the burning of glucose for energy under anaerobic conditions. Magnesium is also critical to

the activity of alkaline phosphatase, the enzyme required for the construction of the hydroxyapatite (calcium phosphate) crystal which provides rigidity to bone. Magnesium is either a synergist with or an antagonist against calcium in the regulation of muscular activity and the nerve impulses that control the activity.

In fact, magnesium is a synergistic mineral that resembles a number of others. Like potassium, it is an important intracellular (inside the cell) constituent. Like sodium, kidneys will respond quickly at times of deprivation to retain body stores of magnesium. But under normal dietary intake, magnesium, sodium, potassium and phosphorus are all readily excreted through the kidneys in urine. And dietary intake varies greatly. Normal intakes range from 150 to 400 mg. per day, with 60% to 70% excreted in the stool.

From these statistics we can see that magnesium is also like calcium in many ways; 30% to 40% will be absorbed, a percentage only slightly higher than calcium, and we find magnesium working in consort with calcium to regulate heart function, hormone secretion and bone metabolism. Even 55% of the body's magnesium is held in bone though not as part of the rigid hydroxyapatite crystal. Yet next to calcium and phosphorus, it is a major component of bone. Soft tissues retain another 27% of the magnesium with the remaining 18% circulating in free ionic form (10%), complexed form (bound to other biochemicals, 2.3%) or protein-bound form (chelated, 5.7%) in the fluids of the body.

Absorption of magnesium follows the now familiar pattern for other minerals of increasing percentage absorbed as daily intake diminishes (see vital statistics above). However, even though efficiency may decrease, total milligrams absorbed is greater as dosage increases. Water intake, that is, extra amounts of water, appear to enhance magnesium ion absorption. This effect has been termed the solvent drag, as magnesium ionized in the water seems to follow the water along the latter's absorptive pathway. The control of magnesium absorption and excretion, like phosphorus, seems to be largely reliant upon intestinal uptake and kidney conservation mechanisms respectively. Responses by the epithelial cells in each of these organs maintain homeostatic magnesium levels over a broad range of dietary intake. Such control has obviously evolved as an adaptation to a food supply where magnesium is not ubiquitous but rather concentrated in some foods and nearly absent in others.

We can only speculate on why magnesium homeostasis operates free of hormonal control. In laboratory studies, calcitonin administered to normal human test subjects caused the expected drop in serum calcium levels as more calcium was delivered to bone. But there was no significant change in magnesium levels.

When parathyroid hormone was given with the expectation that magnesium, calcium and phosphate salts and ions would be liberated from the bone, little or no rise in serum magnesium was observed. But in spite of magnesium's relative freedom from hormonal control, it is closely tied, metabolically speaking, to a mineral that is subject to hormonal control, calcium.

Magnesium is required for the secretion of parathyroid hormone. When magnesium is low, hormone secretion is diminished. Evidence also exists that





magnesium deficiency allows calcium to be somewhat resistant to the effects of parathyroid hormone. So as magnesium becomes deficient, circulating parathyroid hormone may diminish, calcium may not be liberated from bone, and hypocalcemia (low serum calcium) may result. Potential problems with the neurological regulation of heartbeat and the flexion and relaxation of muscle may occur. Muscle spasms, tremor, tetany, heart arrhythmia, anorexia, nausea and apathy are also symptoms of magnesium deficiency. But potential problems do not relate solely to magnesium depletion.

Both calcium status and potassium status are affected by fluctuations in magnesium. As magnesium becomes deficient, potassium is spilled through the urine. Hypokalemia (low potassium) occurs. The loss of these two electrolytes, magnesium and potassium, to heart tissue can dramatically upset the nerve impulses controlling rhythm. The problem can be further complicated if calcium intake remains adequate. When calcium intake is adequate in the face of dietary magnesium deficiency, hypocalcemia can occur. This is not to say that absorption is impaired. Calcium and magnesium *absorption* occur independently. One can be taken up without the other being present. So when magnesium is undersupplied and calcium intake remains normal, a positive calcium balance can persist, i.e. more calcium is coming in than is being excreted from the body. Yet low serum calcium levels will remain as long as magnesium is deficient.

What is happening? The calcium is being stored somewhat indiscriminately in body tissues. The low level of parathyroid hormone due to magnesium deficiency is unable to balance calcitonin and maintain adequate serum levels of calcium. Even in those individuals whose parathyroid hormone levels remain normal, hypocalcemia is found. It is believed the lack of magnesium has made the calcium in the bone resistant to liberation by the hormone. Calcium absorbed from the gut is then too efficiently stored in bone tissue *and* in soft tissue.

In the heart and other muscles, a delicate electrolyte balance is upset with more calcium ions present relative to magnesium and potassium. In neurological tissue, potassium is low relative to sodium, and nerve impulses are disturbed. And don't forget that energy production also relies on magnesium. As magnesium is drained from the muscles, lethargy, apathy and confusion may result.

Magnesium is truly a "tie that binds." Energy, nerves, bone, muscle are all reliant upon its presence. Most identifiable deficiency states occur in relationship to disease. Obvious manifestations are seen in chronic alcoholism, severe malabsorption syndromes, burns, kidney disease, parathyroid gland disorders, and in uncompensated use of diuretics in congestive heart failure. Malnutrition cannot be ruled out, however.

And what of subclinical states of deprivation? If a major contributor to the arteriosclerotic hardening of arteries in heart disease is calcium, could that calcium have been deposited there, in the soft tissue of the arterial wall, at least partly in response to a relative magnesium deficiency? Magnesium supplements from magnesium chloride to magnesium orotate have demonstrated an ability to

restore a degree of elasticity to hardening arteries. Their effectiveness is not entirely without precedent. Bodily responses to magnesium supplementation are quite rapid. Barring a predisposing malabsorption syndrome, serum calcium will usually return to normal within a week of repletion therapy with magnesium, the first few days requiring doses of 2 to 4 grams, followed by more moderate levels nearer the U.S. RDA.

It has been suggested in the recent scientific literature that the ratio of calcium to magnesium in the diet would more completely support optimum health if it were a 1 to 1 or 1 to 2 ratio. Biochemical orthodoxy has for decades held to a ratio of 2 to 1, calcium to magnesium as the ideal. A ratio close to 2 to 1 is reflected in the U.S. RDA's. With the continued prevalence of degenerative diseases where mineral imbalances can always be identified in the patients, perhaps it is time to rethink the historical dogma attached to mineral nutriture.





SODIUM: Na: common ion $Na^+$

Vital Statistics:

| | 1980 Estimated Safe and Adequate Daily Dietary Intake (ESADDI) |
|---|---|
| Children 1 - 3 | 325 - 975 mg |
| 4 - 6 | 450 - 1350 mg |
| 7 - 10 | 600 - 1800 mg |
| Adolescents 11 and older | 900 - 2700 mg |
| Adults | 1100 - 3300 mg |

*Note:* In 1979 a number of minerals long known to play a role in human nutrition were for the first time evaluated for daily intake. Though no official U.S. RDA's were established for them, an Estimated Safe and Adequate Daily Dietary Intake (ESADDI) was developed, and expressed as an acceptable range, by the Food and Nutrition Board of the National Academy of Sciences -National Research Council, the same organization that determines the U.S. RDA's. No ESADDI's were set prior to 1979-80.

Normal (Average) Daily Intake: 2,000 to 10,000 mg (2 - 10 grams)

Absorption: Rapid and efficient throughout nearly the entire gastrointestinal tract. Rectum only does not absorb sodium; jejunem with the aid of sugars and amino acids.

Typical Daily Excretion: 92% of daily intake.

Food Sources: Table salt, seafood, root vegetables, pork, cured meats, junk foods, processed foods (cereals, canned meats, pastries, condiments), cheese

Protagonists: Sucrose, glucose, lactose, galactose, amino acids

Antagonists: diarrhea, steatorrhea

## DISCUSSION

If ever there were a mineral that was easy to absorb, sodium is it. If ever there were a mineral the popular media detested, sodium is it. If ever there were a mineral people loved, sodium is it! How good so many foods taste with it added as sodium chloride, and how bland they taste without it. Our appreciation of food has been trained by sodium. And now we are badgered by news reports and magazine articles decrying the results of our over-consumption. The surreptitious addition of sugar and salt to the burgeoning class of processed foods has certainly upset the normal dietary balance of sodium to other minerals.

Our bodies evolved on a wild diet rich in potassium and relatively deficient in sodium. Since the two minerals balance each other at the membrane of nerve cells, dancing quickly back and forth across the border to create the electric impulse of a firing nerve, it was obvious that the body must evolve a method of conserving

sodium, the *scarce* member of the team. It did. The kidneys took on the task.

Sodium (and other materials) are filtered out of the blood through clusters of capillaries called glomerular bundles. The sodium is caught by a cup (Bowman's capsule) at the end of a renal (kidney) tubule, a tiny tube designed to carry away waste materials. If needed, and under the influence of the hormone aldosterone, the sodium can be reabsorbed from the renal tubule and sent back into circulation. This same mechanism is employed to conserve other precious minerals such as magnesium. It is less effective with potassium. Whereas the kidneys can slow the loss of sodium through the urine to only 10 milligrams a day, it will still allow a minimum of 240 milligrams of potassium to escape. Why? Because the system developed in an environment that provided an abundance of potassium and a scarcity of sodium. Were we still eating natural whole foods exclusively instead of a diet laced with salted butter, corn chips and an occasional restaurant meal, we would be at risk of sodium deficiency.

Confusion, heart fibrilations, dehydration, weakness, lethargy, low blood pressure and neurological disturbances are evidence of sodium deficiency. In modern times, only physical laborers in warm climates, athletes exercising in hot sun and workers in hot mines, steel mills and furnace rooms should pay extra attention to their sodium needs. 10 grams or more, equivalent to 25 grams of table salt (sodium chloride, NaCl) can be lost in a day of heavy perspiration.

But for most of western man, the body burden is one of sodium. Extra amounts of water are required to flush the extra dietary sodium and chloride ion out through the kidneys. That is why salty foods induce thirst. It is the body's safeguard against the impending loss of water.

Water retention is the modern concern attached commonly to "salt" and "sodium." For many persons, a meal of salty foods leaves them feeling bloated and heavy the day after. For congestive heart patients, the concerns over salt and sodium are more seriously tied to fears of heart attack and death and not just to a swollen appearance. The use of salt can cause excess water retention in the heart patient, creating a dangerously elevated back-pressure against the weakened heart as it struggles to force blood through constricted capillaries in swollen tissues. Blood pressure increases. The poor blood flow also delays elimination of the sodium and chloride through the kidneys.

The most recent investigations into the salt/sodium/water/high blood pressure relationships are causing a stir. Sodium alone does not appear to be responsible for the water retention and elevated blood pressure experienced when salt is eaten. Instead the chloride ion (chlorine with a negative charge, $Cl^-$) has been identified as the offender. Sodium in combination with other ligands caused no rise in blood pressure. Sodium chloride did. These findings could imply that quality miso and tamari soy sauce, where much of the sodium is bound to protein and not to chloride, may be suitable alternatives to the use of table salt. At the very least, the new findings of sodium's innocence could lead to a reevaluation and reformulation of "low sodium" diets for application in degenerative disease.





**CHLORIDE:** $Cl^-$: common form as $Cl^-$

**Vital Statistics:**

| | 1980 ESADDI |
|---|---|
| Children 1 - 3 | 500 - 1500 mg |
| 4 - 6 | 700 - 2100 mg |
| 7 - 10 | 925 - 2775 mg |
| Adolescents 11 and older | 1400 - 4200 mg |
| Adults | 1700 - 5100 mg |

**Normal (Average) Daily Intake:** 3000 mg to 15,000 mg

**Absorption:** 90+ percent

**Typical Daily Excretion:** Virtually all that is ingested, through the urine

**Food Sources:** Table salt; processed foods, chlorinated water

## DISCUSSION

A clarification must be drawn at the outset. Chloride is quite different than chlorine. Chlorine is of course a deadly, poisonous gas. For that matter, sodium is also a poisonous metal. But each are almost magically converted to non-toxic, gentle substances in their ionic states (sodium = $Na^+$, chloride = $Cl^-$). The sodium ion ($Na^+$), which doesn't seem to have a shorter name and the chlorine ion which does - chloride ($Cl^-$) - combine to form table salt, a substance certainly not considered a toxin.

Chloride need not enter the body as part of table salt. In seafoods, for instance, from fish through sea vegetables, chloride is delivered in combination with both sodium and potassium. Once absorbed across the intestinal lining, a process of high efficiency, the chloride ion is maintained in blood plasma at a ratio just slightly less than 3-to-2, sodium-to-chloride.

The main role of chloride is to maintain the electrical neutrality of tissues. Sodium and chloride ions ($Na^+$ and $Cl^-$) predominate in the fluids outside cells, the *extra*cellular fluids. Inside the cells, intracellularly, potassium ions ($K^+$) and phosphate ions ($HPO_4^{-2}$ and $H_2PO_4^-$) are the majority. All these ions balance each other to maintain an overall neutral electrical charge in the tissues.

But of course chloride must do more. The parietal cells in the lining of the stomach incorporate chloride into the hydrochloric acid secreted by them to digest protein. By aiding in the breakdown of protein, chloride indirectly influences the acid-base balance of blood plasma. Amino acids, phosphate and sulfate released by the digestion of proteins are absorbed and acidify the blood.

By stimulating retention of water in the tissues, chloride can also indirectly work to alkalinize the blood plasma by simply causing the medium to become more diluted. The mineral is innately benign; but dietary abuse as part of our modern fast food/processed food - indulgent culture has overpowered the

body's marvelous excretory control of chloride. The system rapidly sheds the ion and the water it holds with it, usually beginning the process less than two hours after ingestion. But for many the system cannot work fast enough. An unnecessary rise in blood pressure may follow.





ZINC:

| Vital Statistics: | 1980 U.S. RDA | 1973 U.S. RDA |
|---|---|---|
| Children 1 - 10 | 10 mg | 8 mg, under 4 yrs |
| Males 11 and older | 15 mg | 15 mg, 4 to adult |
| Females 11 and older | 15 mg | 15 mg, 4 to adult |
| Pregnant | 20 mg | 15 mg |
| Lactating | 25 mg | 15 mg |

Normal (Average) Daily Intake: 10 - 15 mg

Absorption: 33% to 50%

Typical Daily Excretion: 0.5 mg in urine

Food Sources: Shellfish, fish, poultry, eggs, beef, lamb, pork, milk, yeast, wheat germ

Protagonists: Whole food diet, milk (maybe)

Antagonists: Cadmium, lead, mercury, grains (phytic acid)

## DISCUSSION

If you commute on the highways, you would be well advised to take supplemental zinc. Cadmium, a toxic heavy metal, is used in the manufacture of the synthetic rubber in automobile tires. As tires wear down from friction on the road, they release cadmium into the air - air other drivers breath. So the person who commutes by car is exposed to extra amounts of cadmium. The cadmium, once absorbed through the lungs, has an affinity for kidney tissue and will collect there.

The toxic effect of cadmium on the kidneys is to force a rise in blood pressure. Elevated blood pressure is an infamous risk factor for heart disease. The chain reaction has been initiated. But natural chelating agents can dislodge and extract the cadmium. Vitamin C, the amino acid cysteine and especially zinc are effective at replacing cadmium in and removing zinc from kidney tissue.

Zinc is also known to counterbalance and remove another toxic heavy metal that threatens not only commuters but also all urbanites, and that is lead. Lead has become ubiquitous in our environment since the Greeks and Romans first began mining it for use in plumbing fixtures. A quiet theory among some historians suggest that the fall of the Roman Empire may have been due at least in part to the exposure of the ruling class to lead in their water. Excessive intake of lead impairs the immune response, making one more susceptible to disease. As lead accumulates, mental functioning is compromised. Memory, judgement and emotional stability suffer, and there is a drop in I.Q. Sterility is also a symptom of lead toxicity.

The Roman ruling class may have been functioning under a terrible biochemical burden. Their lives may have been shortened or their effectiveness compromised so from illness that their rule was weak. Or perhaps judgement became so poor that followers could find no reason to remain loyal. Or perhaps the ruling class simply could not reproduce itself. But our concern today is not with the Roman past, but with our own future.

Lead from gasoline ("unleaded" fuel still contains some lead) contaminates the air of all leading metropolitan centers in the United States except Honolulu. European, Australian and other industrialized cities suffer the same plight. Lead is now present in the bones and tissues of all animals and humans, even before they are born!

So that we can be assured that the fate of Rome's ruling class will not affect us in like fashion, it is wise to make a conscientious attempt to maintain our daily zinc intake at a safe and adequate level. Foods should provide the zinc under normal conditions - but conditions are no longer normal.

Yes, there is a strong case for zinc supplementation. Our exposure *and* absorption of toxic metals is greater now than at any time is history. Our soils are also more depleted of zinc today than at any time in history. The foods grown on those soils are therefore "out of synch" with the zinc levels around which our human biochemistry evolved. And the biochemical engines that "run" us today are still the same biochemical engines that ran us millenia ago when we were still nomadic members of hunter-gatherer societies. Then, we ate wild, trace nutrient-rich foods. Today our enzyme systems still require the same nutrient density per calorie from our foods that our ancestors required, but in almost all cases the density is missing. Zinc is one nutrient slipping from foods.

Modern food processing again is a culprit; it worsens an already critical situation by removing zinc through washing, freezing, grinding, cooking and storing of foodstuffs. Foods that were *not* ideally rich in zinc are further depleted. Our unfortunate annihilation of zinc comes at a time when we need it most to combat the growing burden of toxic heavy metals. Nearly universal supplementation may be in order.

The latest laboratory research on absorption indicates that zinc picolinate may be the most easily absorbed of all zinc compounds. Picolinic acid itself has been suggested as an efficient transporting ligand for zinc (and possibly copper, iron and manganese) across epithelial cell membranes. Depending on bioindividuality - each person's own unique biochemistry - one could expect to absorb from 2-1/2 to 7 times more zinc in picolinate form than in a more conventional gluconate or oxide form. For this reason, zinc picolinate is of special value in nutritional repletion therapies for persons with any of a variety of malabsorption snydromes.

But absorption is only one aspect of bio-utilization. Once the zinc is released in ionic form through the base membrane and enters the bloodstream, it is no more bioavailable than any other form. It may even be less available to the cell as an ion than some of the sophisticated mineral transport compounds such as zinc





orotate or zinc aspartate. Since these latter forms may be readily transported intact through cell membranes, a complete and effective zinc supplement that tries to achieve superior bio-utilization should include them all. Absorption would be assured as would final transport into the tissue cells of zinc in forms that could be utilized by the cell. All three aspects of bio-utilization - absorption, transportation, and utilization - may possibly be met by a zinc supplement that complexed the best zinc transporters together.

Supplemental daily dosages will vary with environment, diet, age and other physical determinants. Research now implies that daily dosages approaching and above 100 mg. can depress the immune response whereas dosages up to 50 mg. or slightly higher can boost immunity. 15 mg. to 50 mg. per day appear to provide optimal maintenance of body zinc status. RNA and DNA synthesis is supported, growth and tissue repair continue.

Zinc also is essential for bone health as a major component of alkaline phosphatase, the enzyme required for the production of the hydroxyapatite crystal in bone. It is likewise an essential component of pancreatic proteolytic (protein - digesting) enzymes, and the potent cellular antioxidant (anti-aging) enzyme, superoxide dismutase (S.O.D.). And the list goes on. There are more than 80 zinc-containing enzymes and proteins that have been identified, many with fascinating biochemical roles to play.....but then, you may read about those and many more facts about zinc in the *Mineral Logic* volume devoted entirely to zinc.

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