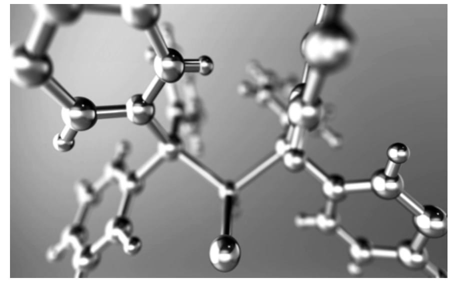
INTERNATIONAL
IV NUTRITIONAL THERAPY
GLOBAL PHYSICIAN EDUCATION

IV Nutrients and NAD

Advanced NAD IV
Dr. Brenden Cochran, Dr. Virginia Osborne

NAD

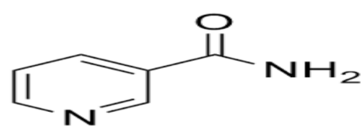


NAD

- Originally discovered in 1905 in yeast, NADH is also known as the reduced form of coenzyme 1, a complementary enzyme utilized in the production and regulation of energy via oxidative phosphorylation.
- **NADH is the reduced** coenzyme form of vitamin B3
- **NAD is the oxidized** coenzyme form of vitamin B3.
- NAD and NADH are converted into each other in numerous different metabolic activities.

Parenteral "B-3"

Normally in the Niacinamide form:



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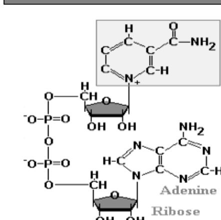
NAD versus B3



Nicotinamide Adenine Dinucleotide (NAD^+)

- Holds two "B-3" molecules

AND

- Two Ribose Molecules

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NAD

- NAD and NADH also serve to activate various enzymes. **NADH is the first of five** enzyme complexes of the electron transport chain where much of the ATP bioenergy that runs every biological process of the body is formed.

NAD

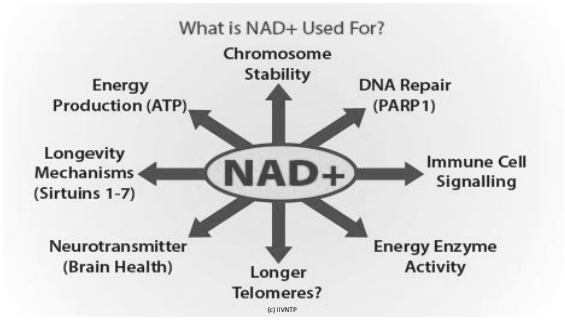
- In some metabolic reactions:
- NAD which is the needed catalyst
- NADH a useful by-product
- In other reactions the situation is reversed.

NAD

- NAD(H) is necessary to oxidize all macronutrients into ATP.
- All cells require energy to survive
- NAD(H) reacts with oxygen to form water and energy.

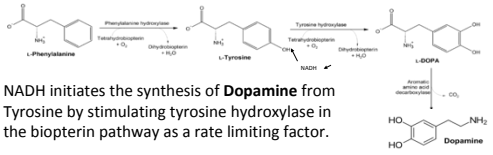
NAD

- 1 molecule of NADH = 3 molecules of ATP
  - with the amount of NADH present dictating the amount of energy produced.
- **Cardiac muscle** cells contain **90 mcg/gm of NADH (per gm of tissue)**
- **Brain and muscle tissue containing 50 mcg/gm**.
- **Red blood** cells contain **NADH with 4 mcg/gm**.
- The more NADH present in cells, theoretically, the greater function of energy production.



NAD

- It is used in the synthesis of Tyrosine



- NADH initiates the synthesis of **Dopamine** from Tyrosine by stimulating tyrosine hydroxylase in the biopterin pathway as a rate limiting factor.

https://upload.wikimedia.org/wikipedia/commons/thumb/d/dd/Conversion_of_phenylalanine_and_tyrosine_to_its_biologically_important_derivatives.png/800px-Conversion_of_phenylalanine_and_tyrosine_to_its_biologically_important_derivatives.png

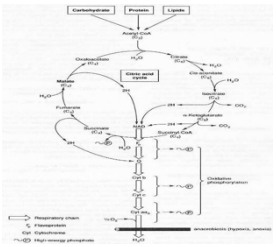
The role of mitochondrial dysfunction in disease:

Damage to cell organelles (mitochondria, nucleus, ER, membrane) disrupts active transport, enzyme production and function & cell reproduction.

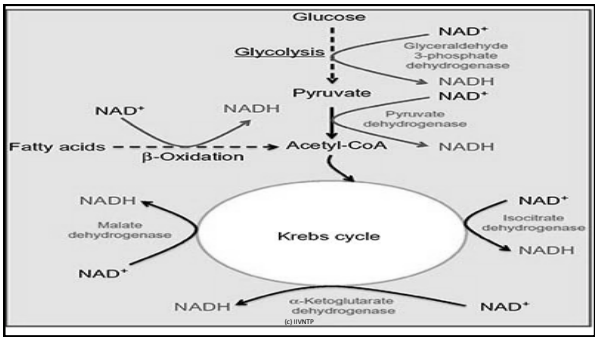
These changes disrupt cell osmotic balance, transporters and other factors causing degenerative changes in the cell, tissue and eventually the person.

Mitochondria: ELECTRON TRANSPORT / OX-PHOS – 1

- **TCA cycle and Glycolysis only form a few ATP molecules each.**
- **Most of the ATP from glucose metabolism comes from the electron transport system.**
- **The main function of all the earlier steps is to make the Hydrogen of the Glucose molecule available in forms that can be utilized for oxidation.**



(c) IVNTP



(c) IVNTP

NAD and Enzyme Activation

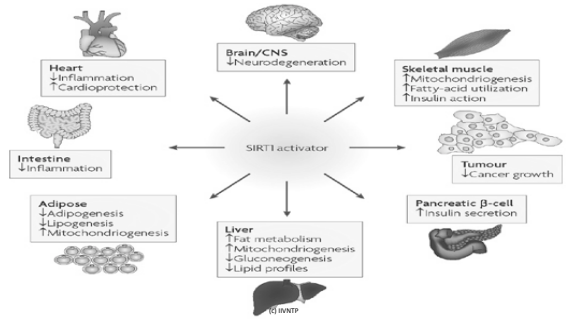
- **SIRTs (**Sirtuins) **-** Enzymes that turn off certain genes that promote aging, such as those involved in inflammation, fat synthesis, insulin & circadian rhythm
  - 7 different Sirtuins but SIRT 1 & 3 are of most importance
  - ^ NAD+ levels or ^ NAD:NADH ratio in the body = more active SIRTs
- **PARPs** (Poly ADP-ribose polymerases) - play an important role in various cellular processes, including modulation of chromatin structure, transcription, replication, recombination, DNA repair, cell proliferation and cell death
  - catalyze the transfer of ADP-ribose to target proteins.
- **CD38 -** The main enzyme involved in the degradation of the NAD precursor nicotinamide mononucleotide (NMN); overexpression is specifically responsible for the decline of mitochondrial NAD+ that occurs with aging
  - Important for immune, endothelial & CV function

(c) IVNTP

Sirt1, when activated by NAD+: activates:

- **PGC-1alpha** - stimulates mitochondrial biogenesis and increased fatty acid oxidation
- **P53** - a tumor-suppressor gene that prevents cells with DNA damage from growing into cancers
- **SREBP1c** - controls blood sugar, fatty acid, and fat production in response to insulin. It also controls cholesterol levels
- **PPAR-gamma** – increases insulin sensitivity
- **FOXO1** – reduces adipogenesis

(c) IVNTP



(c) IVNTP

SIRT 3 Activates:

- **SOD2**- a mitochondrial antioxidant enzyme
- **PARP**- enzyme that repairs damaged DNA, etc.
- **LCAD, SDH, AceSC2**- assist in fatty acid & CHO metabolism

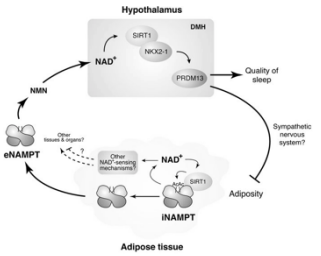
(c) IVNTP

Circadian Rhythm

- SIRT1 is also responsible for metabolic reprogramming, stress resistance & circadian control/rhythm
- It activates the central pacemaker to maintain robust circadian control in young animals, and a decay in this activity may play an important role in aging.
- SIRT1 is part of a regulatory loop that amplifies circadian gene expression.

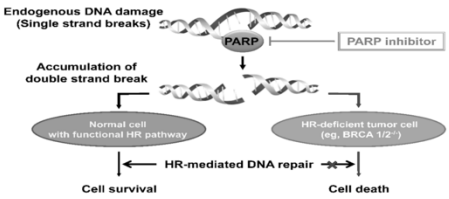
http://www.cell.com/cell/fulltext/S0092-8674(13)00594-1

Disruption to Circadian Rhythm

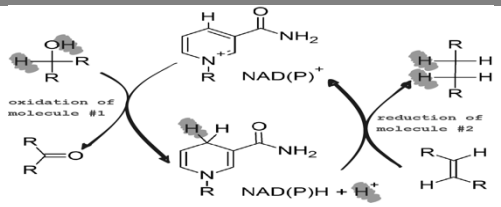


NAD+ & DNA Repair via PARPs

- DNA damage by free radicals occurs constantly in every cell
- This changes chromatin structure which activates PARPs > NAD+ dependent



NAD(P) – NAD(P)H Cycle



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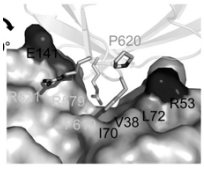
NAD

- **NAD** and **NADH** also serve to activate various enzymes.
- **NAD** is the first of 5 enzyme complexes of the electron transport chain, where ATP is synthesized and runs every biological process of the body.

Mitochondrial Cofactors to consider:

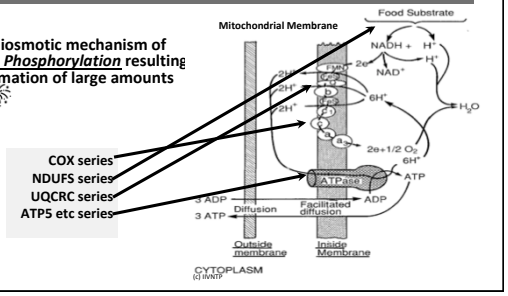
- Nicotinamide / NAD(H)
- CoQ10
- Riboflavin-5-phosphate
- Iron
- Proline
- Ca, Mg, K, Zn,Cu,Cr [positive]
- Cd [negative = mito poison]



•Structure 19, 833–843, June 8, 2011
•African Journal of Food Science Vol. 4(5) pp. 200-222, May 2010
•J. Anim. Sci. Vol. 91, E-Suppl. 2/J. Dairy Sci. Vol. 96, E-Suppl. 1

Mitochondria: ELECTRON TRANSPORT / OX-PHOS SNP's – 4

The chemiosmotic mechanism of *Oxidative Phosphorylation* resulting in the formation of large amounts of ATP.



Mitochondria: ELECTRON TRANSPORT / OX-PHOS – 2

- **Oxidation of Hydrogen is accomplished by Splitting the Hydrogen Atom into:**
  - ***Hydrogen Ion***
  - ***Electron***
- **Electron used to change dissolved O2 from fluids to Hydroxyl ions.**
- **Hydroxyl ions combine to form H2O**
- **During these reactions, ATP produced.**
- **Occurs in the Mitochondria**
- **Occurs via Enzymatically Catalyzed Reactions**

Mitochondria: ELECTRON TRANSPORT / OX-PHOS – 3

SNP's and Poisoning can disorder mitochondrial function:

- **Inhibition is Blocking the respiratory chain at places other than the ADP+P position.**
  - **Poisons can generate this:**
    - **Barbiturates**
    - **Rotenone (insecticide)**
    - **Sulfuric Acid, CO, Cyanide**
    - **Antibiotics: *Oligomycin, Piericidin-A***
- **Uncoupling (derailing) Ox-Phos:**
  - **Ox-Phos is the addition of 'P' to ADP = ATP**
  - **Uncoupling is the dissociation of oxidation from phosphorylation** (Incr. permeability of mitochondria to protons / reduces electromechanical potential – short circuits ATP-synthase [ATPase])
    - **2-4-dinitrophenol, dinitrocresol, pentachlorophenol**

Brief Summary of NAD

- A good majority of enzymatic metabolic reactions in the mitochondria of our cells require NAD+; specifically, the Krebs Cycle
- NAD+ (with the help of O2) drives ATP production & activates enzymes (**SIRTs, PARPs, etc**) > this is how O3 Therapy works for energy/anti-aging
- These NAD-induced enzymes are responsible for various vital processes such as DNA repair, circadian rhythm, healthy inflammation & insulin levels, etc.
- NAD can get depleted with exposure to free radicals & oxidative stress (alcohol, drugs, pesticides, GMO foods, hydrogenated fats, heavy metals, etc.) which then causes NT imbalances, DNA changes, mitochondrial dysfunction, etc.
- This can translate to immune issues, addiction tendencies, chronic fatigue, neurological/mental/emotional disorders, accelerated aging, cancer, etc.

Side Effects

- "Flushing" skin
- General physical flush/rush
- Cardiac
- Phlebitis
- Overall improved circulation

- Time of onset of symptoms: seconds, minute to several minutes

NAD IV Cautions

- **Dilute as indicated**
- Overdose or speed shock can lead to **excessive mitochondrial activity**
  - Tachycardia
  - Arrhythmia
  - Neuro-psych issues
  - Tremor
  - And more

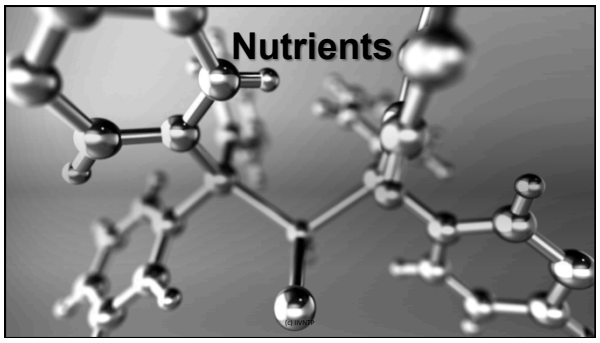
Cantó C, Menzies K, Auwerx J. NAD+ metabolism and the control of energy homeostasis - a balancing act between mitochondria and the nucleus. *Cell metabolism*. 2015;22(1):31-53. doi:10.1016/j.cmet.2015.05.023.

Highlights

- Adaptive cellular metabolism relies on NAD+ to mediate energy signaling
- NAD+ therapeutics is showing its potential to treat disease
- Metabolic syndrome, cancer and aging all involve NAD+ signaling

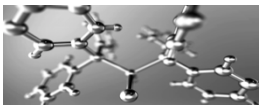
Oral Studies

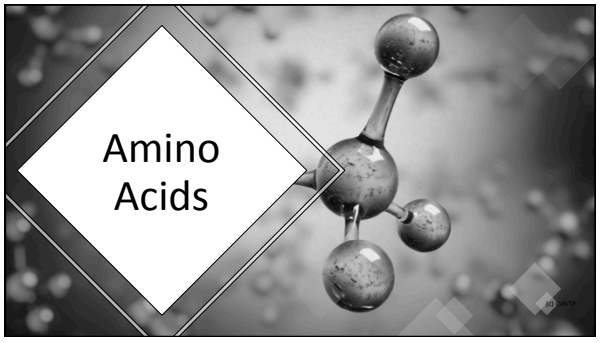
- Samuel A. J. Trammell, Mark S. Schmidt, Benjamin J. Weidemann, Philip Redpath, Frank Jaksch, Ryan W. Dellinger, Zhonggang Li, E. Dale Abel, Marie E. Migaud & Charles Brenner. **Nicotinamide riboside is uniquely and orally bioavailable in mice and humans.** Nature Communications volume 7, Article number: 12948 (2016)
- Santaella ML, Font I, Disdier OM. **Comparison of oral nicotinamide adenine dinucleotide (NADH) versus conventional therapy for chronic fatigue syndrome.** P R Health Sci J. 2004 Jun;23(2):89-93.
- Alegre J, Rosés JM, Javierre C, Ruiz-Baqués A, Segundo MJ, de Sevilla TF. [Nicotinamide adenine dinucleotide (NADH) in patients with chronic fatigue syndrome]. [Article in Spanish] Rev Clin Esp. 2010 Jun;210(6):284-8. doi: 10.1016/j.rce.2009.09.015. Epub 2010 May 5.
- Castro-Marrero J, et.al. **Effect of coenzyme Q10 plus nicotinamide adenine dinucleotide supplementation on maximum heart rate after exercise testing in chronic fatigue syndrome - A randomized, controlled, double-blind trial.** Clin Nutr. 2016 Aug;35(4): 826-34. doi: 10.1016/j.clnu.2015.07.010. Epub 2015 Jul 17.



Supportive Nutrients to potentiate NAD+

- Intravenous and Oral options:
- Amino Acids - complete or precursors specific to their neurotransmitter deficiency (ex: GABA, DLP, 5HTP,)
- NAC
- GSH (IL-2)
- Ca/Mg/K+
- B vitamins
- Protandim
- Kavinace
- Lavella/lavender oil
- SAMe





Amino Acid Infusions, Crystalline
Combination Products

- Examples of what is available

- Aminosyn II, Aminosyn PF
- Plenamine 15%

Vitamin-Amino Acid Interactions
B vitamins required for all amino acid formulations

- B vitamins all contain an amino group - nitrogen
- Pyridoxine, B6, is most important vitamin for amino acid metabolism
- B6 is cofactor for transaminase enzymes which metabolize amino acids
- Riboflavin, B2, is required by GSH reductase
- Niacin, B3, is synthesized from tryptophan
- Others will be covered with specific amino acids

Amino Acids

- Amino acids that are not used for protein synthesis:

- Are used for energy,
- as sugars (glycogenic) or
- fats (ketogenic)

Plenamine 15%

Essential Amino Acids in mg/100 ml

Isoleucine 740
Leucine 1.04 gms
Lysine 1.18 gms
Methionine 749
Phenylalanine 1.04 gms
Threonine 749
Tryptophan 250
Valine 960

Plenamine 15%

- mOsm/ml 1.38 (1358 per liter)
- pH 5.6

Non-essential Amino Acids in mg/100 ml

Alanine 2.17 grams
L-Aspartic acid 434
Histidine 894
Serine 592
N-Acetyl-L-Tyrosine 39
L-Glutamic Acid 749
Arginine 1.47 grams
Glycine 1.04 gms
Proline 894

Single Amino Acid Infusions
Introduction

- See Therapeutic Application of Amino Acid Therapy page for clinical uses
- Commonly available AA's
- Commonly available concentrations
- Safe dosing for single amino acids given IV has a very wide safe range
- Gram doses are noted in literature

N-Acetyl-Cysteine

- 100 mg/ml
- Increases intracellular Glutathione
- NAC increases GSH levels in liver, helps prevent oxidative drug damage
- Transport mechanisms exist for movement of NAC:HM complexes both into and out of the brain

- Increase GSH levels in the kidneys.
  - Nephroprotective and regenerative

N-Acetyl-Cysteine

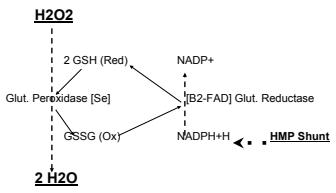
- Typical dosage recommendations are in the range of IV is 250-1500 mg of NAC daily for most therapeutic benefits.
- Typical oral can vary from 500 mg to 2000 mg
  - Increase as tolerated
  - Side effect nausea if consuming oral in to high a dose

Glutathione

- 60 to 200 mg/ml
- A tripeptide synthesized from Glycine, Glutamic acid and Cysteine
- Primary intracellular antioxidant – essential to life
- Useful in any condition where there is risk for oxidative damage
- 500 mg is a reasonable starting dose for conditions benefiting from GSH
  - MCS patients may do better starting at 100-200 mg
- Evaluate how patient tolerates dose before giving high doses, e.g. build up dose over time incrementing 500 mg per infusion

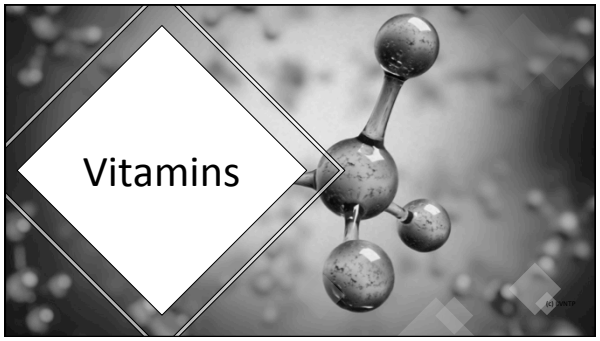
Glutathione

The Glutathione Redox Cycle and Peroxide



Glutathione

- Important role in immune function via white blood cell production and is a potent antiviral agent
- Glutathione levels decrease with age. It is involved in cellular differentiation and slows the aging process



Vitamins: The B's

- Thiamine
- Riboflavin
- Niacinamide
- Dexpanthenol
- Pyridoxine
- B-12
  - Hydroxocobalamin
  - Methylcobalamin
- B- complex

Vitamin B1, Thiamine

- Thiamine is a coenzyme in oxidative decarboxylation reactions
- Important for reactions in energy metabolism
- Requirement is related to energy intake in the form of carbohydrates
- Essential in aldehyde metabolism

Vitamin B1: Thiamine HCl

- Deficiency leads to slow function of the HMP Shunt and the TCA cycle
  - Consider in:
    - Chronic fatigue
    - Depleted patients
    - Therapies attempting to boost glutathione function
- Deficiency can be caused by:
  - Loop diuretics
  - Digitalis
  - Ethanol intake
  - Chronic aldehyde exposure

Acetaldehyde and Nutrient Deficiencies: Thiamine

In addition to its toxic effects, acetaldehyde induces deficiencies of nutrients used for its detoxification. Ex: vitamin B1 (thiamine) is depleted through alcohol and acetaldehyde detoxification. (Thiamine) B1 is essential in carbohydrate metabolism for energy production, of which the brain uses 20 percent.

Acetaldehyde-induced B1 depletions exacerbate the already low B1 levels common in the population due to diuretics and other drugs, over-consumption of simple carbohydrates (dysglycemia) and adrenal stress. In addition to its many functions, thiamine, the "nerve vitamin," is critical to nerves and neurotransmitters. Even mild, chronic B1 deficiency can produce brain-related symptoms such as emotional instability, confusion, depression, fatigue, irritability, headaches, sensitivity to noise, insomnia, decreased short-term memory, brain-fog and a feeling of impending doom. (Lonsdale, Williams)

-Sulfites also destroy vitamin B1's biological activity, contributing to a deficiency. Nutrient depletion leads to sensitivity to other chemicals that use these same pathways.

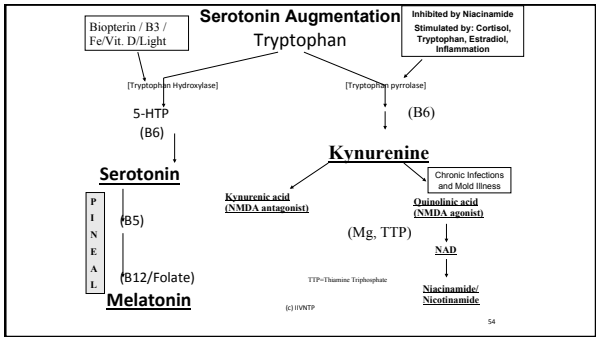
- Takabe M, Itokawa Y. Thiamine depletion after ethanol and acetaldehyde administration to rabbits. J Nutr Sci Vitaminol (Tokyo). Oct. 1983;29(5):509-14.
- Lonsdale D, Shamberger. Red cell transketolase as an indicator of nutritional deficiency. Am J Clin Nutr 1980;33:205-11.
- Williams RR, et al. Induced Thiamin (vitamin B1) Deficiency in Man. Arch Int Med. 1942;69:721-38.

Vitamin B2: Riboflavin

- Low toxicity due to renal dumping of excess
- Depressed B2 status leads to slow beta-oxidation of fats
- Deficiencies can allow for more sensitivities to aflatoxin
  - PMID: 323066 (Interrelationships of mycotoxins with nutrition)
- Helps prevent against mycotoxin hepatotoxicity
- Part of Glutathione Reductase & Synthase enzyme systems
  - Increase use with oxidative therapies
    - H2O2
    - Vit C
- Average IV dose 1 to 10 mg.

Vitamin B3: Niacinamide

- Supports SEROTONIN synthetic pathways
- Part of the glucose tolerance factor
- Heavy use in enzyme systems
  - Used in *Dehydrogenase reactions*.
  - H+ Transfers
  - NAD
- Average IV dose: 100 to 1000 mg.



Dexapanthenol

- B5
- Supplied as the alcohol form of pantothenic acid, Dexpanthenol
- Pharmacologic doses of 2500-3000 mg may prolong the effects of succinylcholine
- Consider in adrenal support, acylation reaction, NAT2 genomics support
- Drug reactions:
  - Rare allergic reactions during use of parenteral dexpanthenol and some antibiotics, barbiturates and opiates when used concomitantly

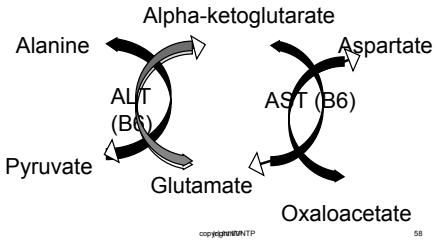
Dexpanthenol

- Pantothenic acid functions as a component of coenzyme A and as part of the acyl carrier protein for fatty acid synthetase
- Has major influences on the synthesis and breakdown of carbohydrates and fatty acids as well as the synthesis of steroid hormones and hemoglobin
- Physiologic doses are very safe

Pyridoxine

- B6
- Pyridoxine acts as a coenzyme in metabolic reactions for carbohydrate, lipid and amino acid metabolism
- Ethanol increases the breakdown of pyridoxine
- B6 deficiency can be induced by malabsorption, malignancies,
- many diseases, long-term drug therapy as well as total parenteral nutrition (TPN).

Transaminase Reactions

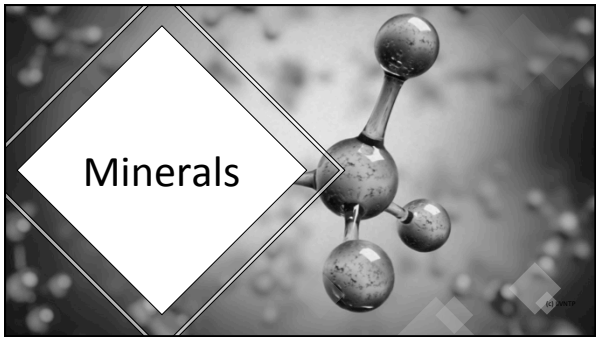


Cobalamins

- Hydroxocobalamin
  known as long acting B12, binds to serum proteins better than Cyano. Suitable for i.m. or i.v. use
  - Hydroxocobalamin is used as a treatment for cyanide poisoning, 5-10 g i.v.
- Methylcobalamin
  - is a metabolically active form of B12, especially suited for neurological complaints

B Complex

- B-complex 100 contains the following ingredients/mL:
  - Thiamine HCl 100 mg
  - Riboflavin-5-phosphate 2 mg
  - Niacinamide 100 mg
  - Dexpanthenol 2 mg
  - Pyridoxine 2 mg
  - NOTE: SOME B-VITAMINS DO HAVE CORN IN THEM!



Minerals

- Magnesium
- Calcium
- Potassium

Activities of agents on the CNS

AGENT	EXCITE CNS	SEDATE CNS
Calcium		****
Magnesium		****
H+		****
Bicarbonate	****	
Phosphate	****	

Magnesium

- Dosage & Administration
  - IM: Up to 3 mL 50% adults
    - 1-3 mL 20% pediatric
  - IV: given normal renal function, up to 50 mEq over 4-6 hours (12.5 mL 50% Mg Sulfate)
  - Clinical use
    - Up to 3 mL 50% over 20-30 min, watch for O.D. Sx
    - Up to 6 mL 50% over 2 hours in drip
- Many (most likely most) patients are Mg deficient and benefit from an appropriate degree of Mg replacement during IVMT

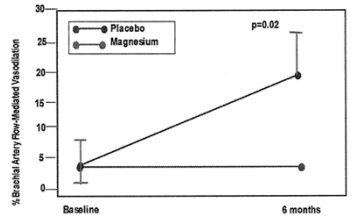


Fig. 8. Percent change in endothelium-dependent brachial artery flow-mediated vasodilation (%FMD) in placebo (horizontal line) (n = 25) and magnesium (diagonal line) (n = 25) at baseline and after 6 months.

Burton B. Silver, PhD

Development of Cellular Magnesium Nano-Analysis in Treatment of Clinical Magnesium Deficiency Journal of the American College of Nutrition, Vol. 23, No. 6, 732S-737S (2004)

Calcium and Magnesium IV Products

- Between 'salt' forms calculating mg / mL based on product concentration may not yield an equivalent dose of the electrolyte in the IV solution.
- Example:
  - Calcium in the chloride and gluconate forms are both 10% solutions (100 mg/ mL).
  - 10 mL Calcium gluconate (1000 mg) yields 4.65 mEq Ca++
  - 10 mL Calcium chloride (1000 mg) yields 13.6 mEq Ca++

Calcium and Magnesium IV Products

Product	Percent	mg/mL	mEq / mL
Calcium Gluconate	10%	100	0.465
Calcium Chloride	10%	100	1.36
Magnesium Sulfate	50%	500	4.06
Magnesium Chloride	20%	200	1.97

(c) IVNTP

Calcium and Magnesium IV Products

- Therefore on an equivalent ionic basis:
  - 1 mL Calcium chloride is equal to 2.92 mL Calcium gluconate
  - 1 mL Calcium gluconate is equal to 0.34 mL Calcium chloride
  - 1 mL Magnesium sulfate is equal to 2.06 mL magnesium chloride
  - 1 mL Magnesium chloride is equal to 0.49 mL Magnesium sulfate

(c) IVNTP

Calcium

- 10% Calcium gluconate
- 10% Calcium chloride,
- 10%Calcium glycerophosphate (Calphosan), contains 50 mg calcium glycerophosphate and 50 mg calcium lactate
- Dosage and administration
  - Gluconate 100-2000 mg (0.465-9.3 mEq) i.v.
    - Infusion rate never greater than 1 mEq/min.

(c) IVNTP

Calcium

- Adverse reactions: hypotension, bradycardia, arrhythmia, tingling sensations, syncope, cardiac arrest
- Parenteral effects are mainly on nerve conduction and muscle contraction

(c) IVNTP

Calcium

- Role:
  - (8.5 – 10.5 mg. / dL Normal range)
  - **Neuromuscular regulation**
  - Skeletal / Bone maintenance
  - Influence enzyme activity
  - Prothrombin ? Thrombin conversion
  - **Calcium and Phosphate have a reciprocal relationship**
- Hypercalcemia:
  - Causes:
    - Hyperparathyroid / overdose of Vit. D or Antacids / Multiple myeloma / Parathyroid adenoma
  - Effects:
    - **Arrhythmias / N-V / Constipation / Lethargy** / Anorexia / Dehydration **/ Coma**

(c) IVNTP

Potassium

- Potassium chloride, 2 mEq/mL is principle form used in IVMT
  - Potassium phosphate and acetate are available
- Dose and administration
  - Not for i.m. use
  - Never add potassium to an i.v. push
  - Always dilute appropriately prior to infusion

(c) IVNTP

Potassium - Clinical

- Typical IVMT dose is 2-5 mL in drip of 200-500 mL given over 1-3 hours
- It is useful to include potassium
  - When giving high dose vitamin C
  - When infusing solutions using D5W as carrier
  - Both of these treatments induce insulin and when insulin moves either glucose or vitamin C into cells potassium is required

NEVER GIVE POTASSIUM IM OR SQ !!!!!

CRITICAL GUIDELINES FOR ADMINISTRATION OF POTASSIUM

Never give a potassium I.V. push.
Potassium chloride (KCl) should be added to a nondextrose solution such as isotonic saline to treat severe hypokalemia because administration of KCl in a dextrose solution may cause a small reduction in the serum potassium level.
Never administer concentrated potassium solutions without first diluting them as directed.
KCl preparations greater than 60 mEq/L **should not** be given in a peripheral vein. Concentrations greater than 8 mEq/100 mL can cause pain and irritation of peripheral veins and lead to postinfusion phlebitis (Rapp, 1987).
When adding KCl to infusion solutions, especially plastic systems, make sure the KCl mixes with the solution thoroughly. Invert and agitate the container to ensure mixing. ***Do not add KCl to a hanging container!***
For patients with any degree of renal insufficiency or heart block, Zull (1989) recommends reducing the infusion by 50 percent. For example, 5 to 10 mEq/h rather than 10 to 20 mEq/h.
Administer potassium at a rate not to exceed 10 mEq/h through peripheral veins (Kokko & Tannen, 1990; Gahart, 1994).
For patients with extreme hypokalemia, rates should be no more than 40 mEq/h while ECG is constantly monitored (Kokko & Tannen, 1990).
If KCl is administered into the subcutaneous tissue (infiltration), it is extremely irritating and can cause serious tissue loss. Use extravasation protocol in this situation.

A common oral supplement with CoQ10

Supplement Facts

Serving Size: 1 Lozenge
Servings Per Container: 30

	Amount Per Serving	%Daily Value
Coenzyme Q10	50 mg	*
PANMOL® NADHmicro (reduced nicotinamide adenine dinucleotide complex)	25 mg	*

* % Daily Value Not Established.

NAD Dosing Considerations

- Compounded 50 mg/ml
- Initial testing dose 1 ml
  - Admixture with other vitamin/mineral protocols
- Increase as tolerated