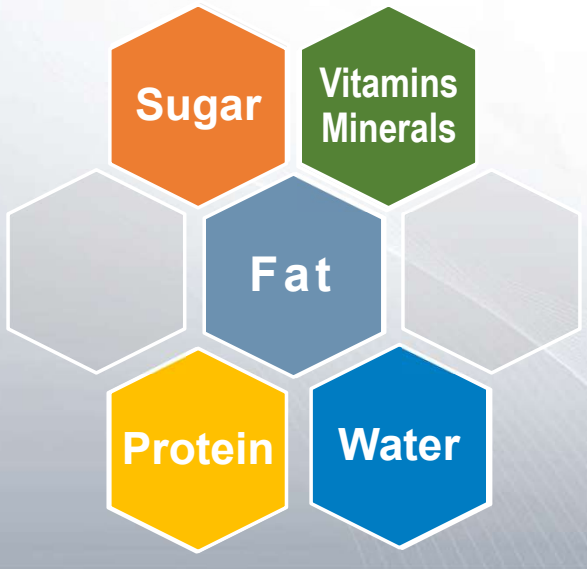
Nutritional Therapy: Top Ten Tenets

For Wound Care

Bob Bartlett MD

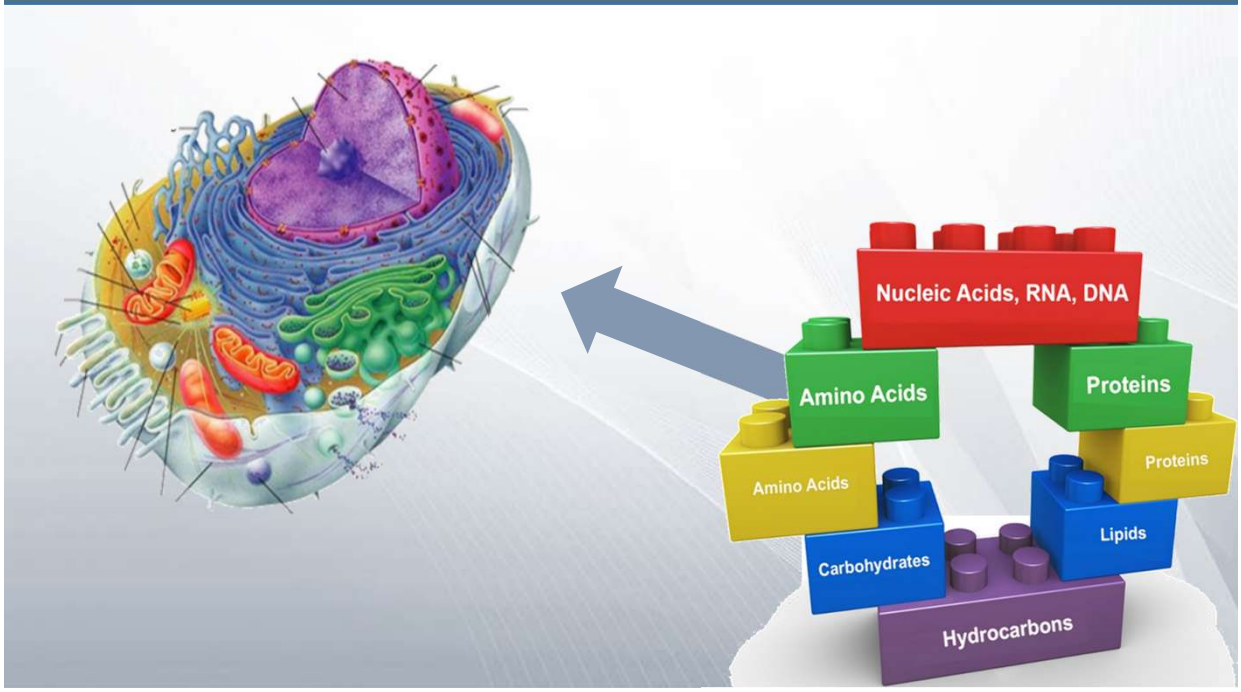
Chief Medical Officer

Nutritional Therapy: Top Ten Tenets

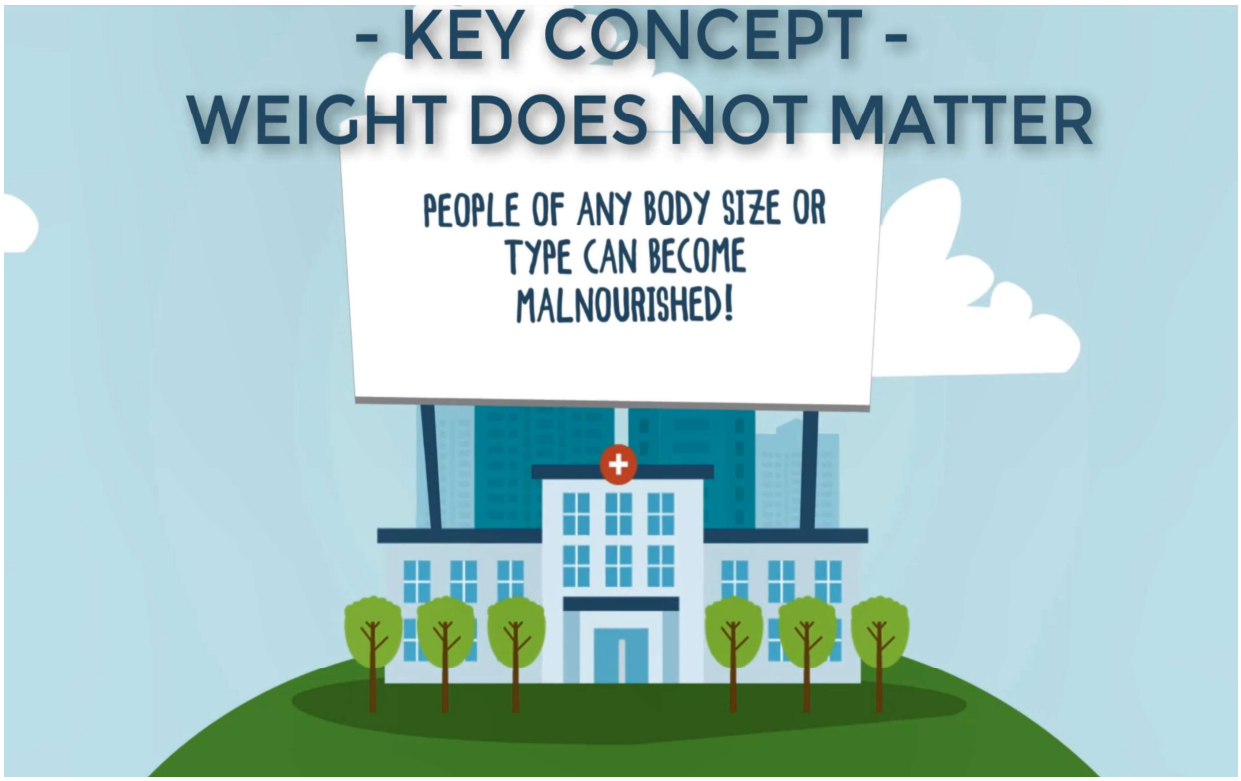


Periodic Table of Nutrition

You can't build cells with antibiotics, dressings or debridement!



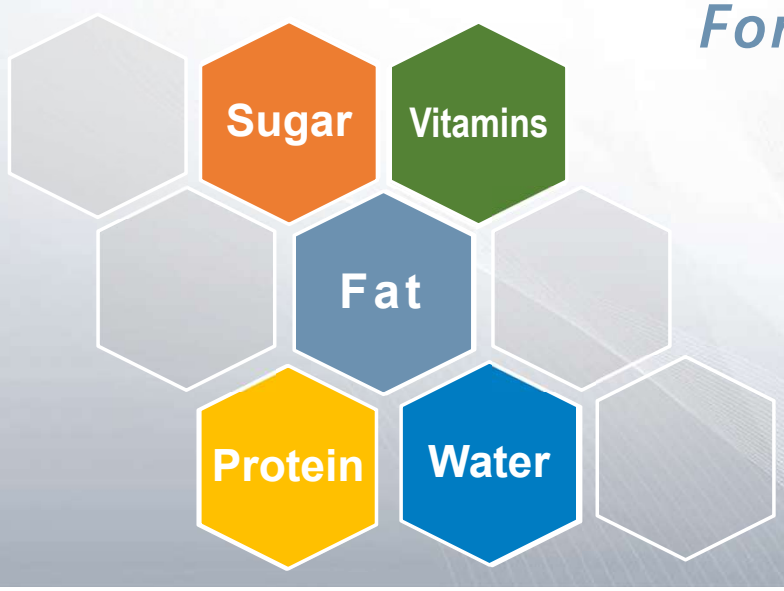
Practical Therapy


- KEY CONCEPT -
WEIGHT DOES NOT MATTER
PEOPLE OF ANY BODY SIZE OR TYPE CAN BECOME MALNOURISHED!


Consumption ≠ Absorption
LEAKY GUT

Nutritional Therapy: Top Ten Tenets

For Wound Care



Sugar

1 - GLYCEMIC CONTROL

It's essential to healing

Routine HbA1c screening for all chronic wounds

Glycemic Control

- Hyperglycemia delays healing, contributes to infection, wound dehiscence, and amputation.
- The importance of glycemic control is *underestimated*. It is often viewed as merely desirable, when in fact it is *essential*.

Target is HbA1c <7% (~*Glucose150 mg %*)

Surgical Site Dehiscence

RECONSTRUCTIVE

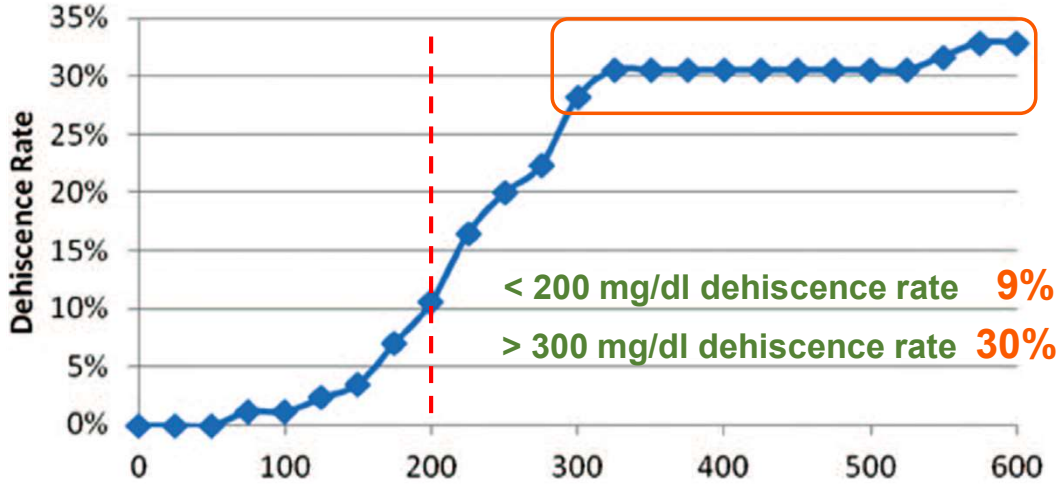
The Role of Chronic and Perioperative Glucose Management in High-Risk Surgical Closures: A Case for Tighter Glycemic Control

Matthew Endara, M.D.
Derek Masden, M.D.
Jesse Goldstein, M.D.
Stephen Gondek, M.D., M.P.H.
John Steinberg, D.P.M.
Christopher Attinger, M.D.
Washington, D.C.; Baltimore, Md.; Philadelphia, Pa.; and Boston, Mass.

Background: The exact risk that poor glucose control introduces to patients undergoing surgical closure has yet to be fully defined.
Methods: The authors retrospectively analyzed a prospectively collected database of patients seen at their wound care center to evaluate the effects of chronic and perioperative glucose control in high-risk patients undergoing surgical wound closure. Hemoglobin A1c and blood glucose levels for the 5 days before and after surgical closure were recorded and compared with the primary endpoints of dehiscence, infection, and reoperation. Univariate and multivariate analyses were performed.
Results: Seventy-nine patients had perioperative glucose levels and 64 had hemoglobin A1C levels available for analysis. Preoperative and postoperative hyperglycemia (defined as any blood glucose measurement above 200 mg/dl) as well as elevated A1C levels (above 6.5 percent or 48 mmol/ml) were significantly associated with increased rates of dehiscence (odds ratio, 3.2, $p = 0.048$; odds ratio, 3.46, $p = 0.028$; and odds ratio, 3.54, $p = 0.040$, respectively). Variability in preoperative glucose (defined as a range of glucose levels exceeding 200 points) was significantly associated with increased rates of reoperation (odds ratio, 4.14, $p = 0.025$) and trended toward significance with increased rates of dehiscence ($p = 0.15$). In multivariate regression, only perioperative hyperglycemia and elevated A1c were significantly associated with increased rates of dehiscence.
Conclusions: In primary closure of surgical wounds in high-risk patients, poor glycemic control is significantly associated with worse outcomes. Every effort should be made to ensure tight control in both the chronic and subacute perioperative periods. (*Plast. Reconstr. Surg.* 132: 996, 2013.)
CLINICAL QUESTION/LEVEL OF EVIDENCE: Risk, II.

The important role that tight glucose control plays in optimizing patient outcomes has been accepted in practically all facets of medicine. Large population studies have proven that hyperglycemia interferes with wound healing in patients with ulcers and contributes to increased rates of infection.[1–3] Poor glycemic control has also been associated with increased rates

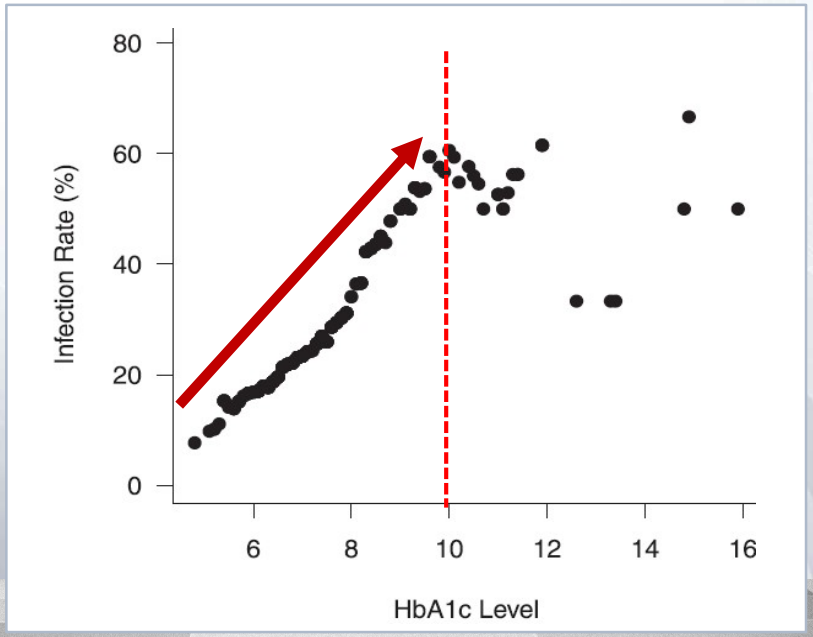
Surgical Site Dehiscence



Preoperative Blood Glucose

Plastic & Recon Surg2013; 132 (4): 996-1004

Infection rates increase steadily as HbA1c increases to 10% and then levels off

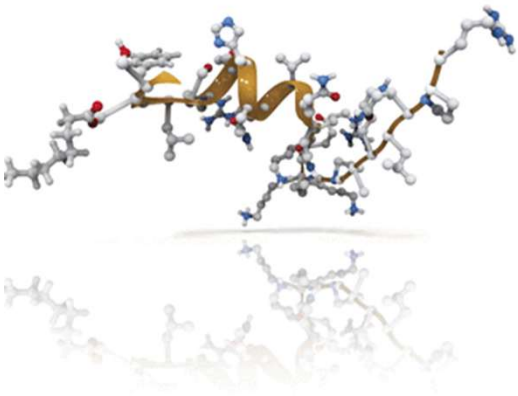


J Foot Ankle Surg. 2014 May-Jun;53(3):307-11

Infection Risk

diabetes

Nielson CP 1989 Aug;38(8):1031-5

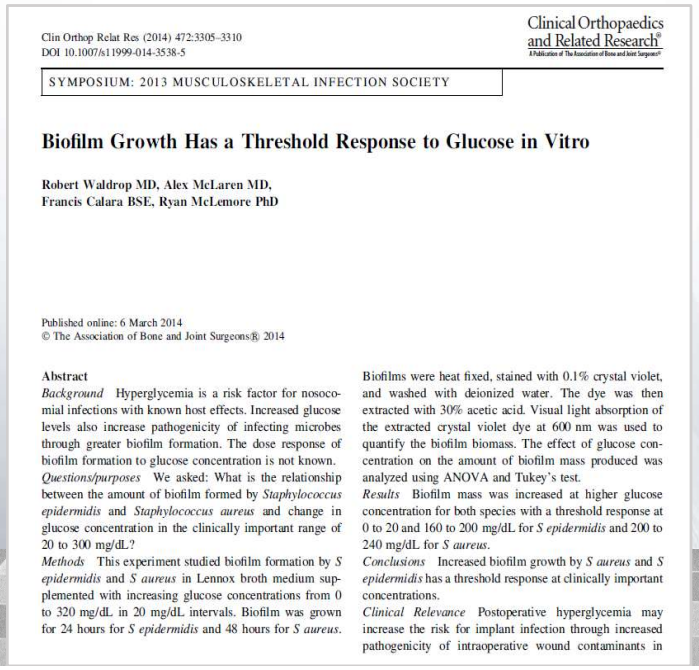


American Diabetes Association.

Neutrophilic function is reduced in proportion to an increase in the glucose level, and 200 mg/dL is the threshold of neutrophil dysfunction.

The respiratory burst was reduced 28% after a 30 min exposure at 200 mg/dL.

Biofilm Growth - Glucose Dependent



Clin Orthop Relat Res (2014) 472:3305–3310
DOI 10.1007/s11999-014-3538-5

Clinical Orthopaedics and Related Research®
A Publication of The Association of Bone and Joint Surgeons®

SYMPOSIUM: 2013 MUSCULOSKELETAL INFECTION SOCIETY

Biofilm Growth Has a Threshold Response to Glucose in Vitro

Robert Waldrop MD, Alex McLaren MD, Francis Calara BSE, Ryan McLemore PhD

Published online: 6 March 2014
© The Association of Bone and Joint Surgeons® 2014

Abstract

Background Hyperglycemia is a risk factor for nosocomial infections with known host effects. Increased glucose levels also increase pathogenicity of infecting microbes through greater biofilm formation. The dose response of biofilm formation to glucose concentration is not known.

Questions/purposes We asked: What is the relationship between the amount of biofilm formed by *Staphylococcus epidermidis* and *Staphylococcus aureus* and change in glucose concentration in the clinically important range of 20 to 300 mg/dL?

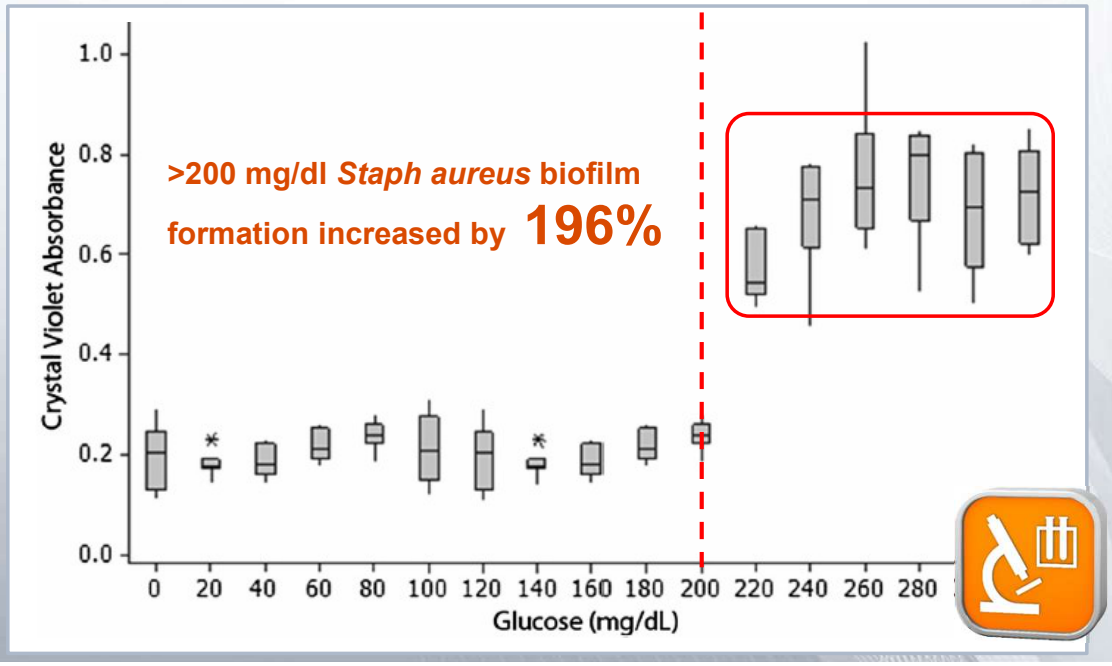
Methods This experiment studied biofilm formation by *S epidermidis* and *S aureus* in Lennox broth medium supplemented with increasing glucose concentrations from 0 to 320 mg/dL in 20 mg/dL intervals. Biofilm was grown for 24 hours for *S epidermidis* and 48 hours for *S aureus*. Biofilms were heat fixed, stained with 0.1% crystal violet, and washed with deionized water. The dye was then extracted with 30% acetic acid. Visual light absorption of the extracted crystal violet dye at 600 nm was used to quantify the biofilm biomass. The effect of glucose concentration on the amount of biofilm mass produced was analyzed using ANOVA and Tukey's test.

Results Biofilm mass was increased at higher glucose concentration for both species with a threshold response at 0 to 20 and 160 to 200 mg/dL for *S epidermidis* and 200 to 240 mg/dL for *S aureus*.

Conclusions Increased biofilm growth by *S aureus* and *S epidermidis* has a threshold response at clinically important concentrations.

Clinical Relevance Postoperative hyperglycemia may increase the risk for implant infection through increased pathogenicity of intraoperative wound contaminants in

Biofilm Growth - Glucose Dependent



Sugar 1 - GLYCEMIC CONTROL

Sugar 2 – SUGAR SUBSTITUTES

It's complicated

Sugar

2 - Sugar Free Pitfalls

Physiol Behav. 2015 Dec 1;152(Pt B):450-5

Metabolic effects of non-nutritive sweeteners

M. Yanina Pepino *

Center for Human Nutrition, Washington University School of Medicine, St. Louis, MO, United States

Until recently, the general belief was that non-nutritive sweeteners (NNS) were healthy substitutes because they provide sweet taste without calories or glycemic effects.

However, data from several studies found they are associated with ***increased risk*** of **obesity, metabolic syndrome**, and **type 2 diabetes**.

Intense sweeteners
Non-caloric sweeteners
Sucralose
Metabolism

homeostasis, 2) NNSs interfere with gut microbiota and induce glucose intolerance, and 3) NNSs interact with sweet-taste receptors expressed throughout the digestive system that play a role in glucose absorption and trigger insulin secretion. In addition, recent findings from our laboratory showing an association between individual taste sensitivity to detect sucralose and sucralose's acute effects on metabolic response to an oral glucose load are

Clinical Care/Education/Nutrition/Psychosocial Research

ORIGINAL ARTICLE

Diabetes Care. 2013

Sucralose Affects Glycemic and Hormonal Responses to an Oral Glucose Load (Splenda)

M. Yanina Pepino, phd
Courtney D. Tiemann, mph, ms, rd
Bruce W. Patterson, phd
Burton M. Wice, phd
Samuel Klein, md

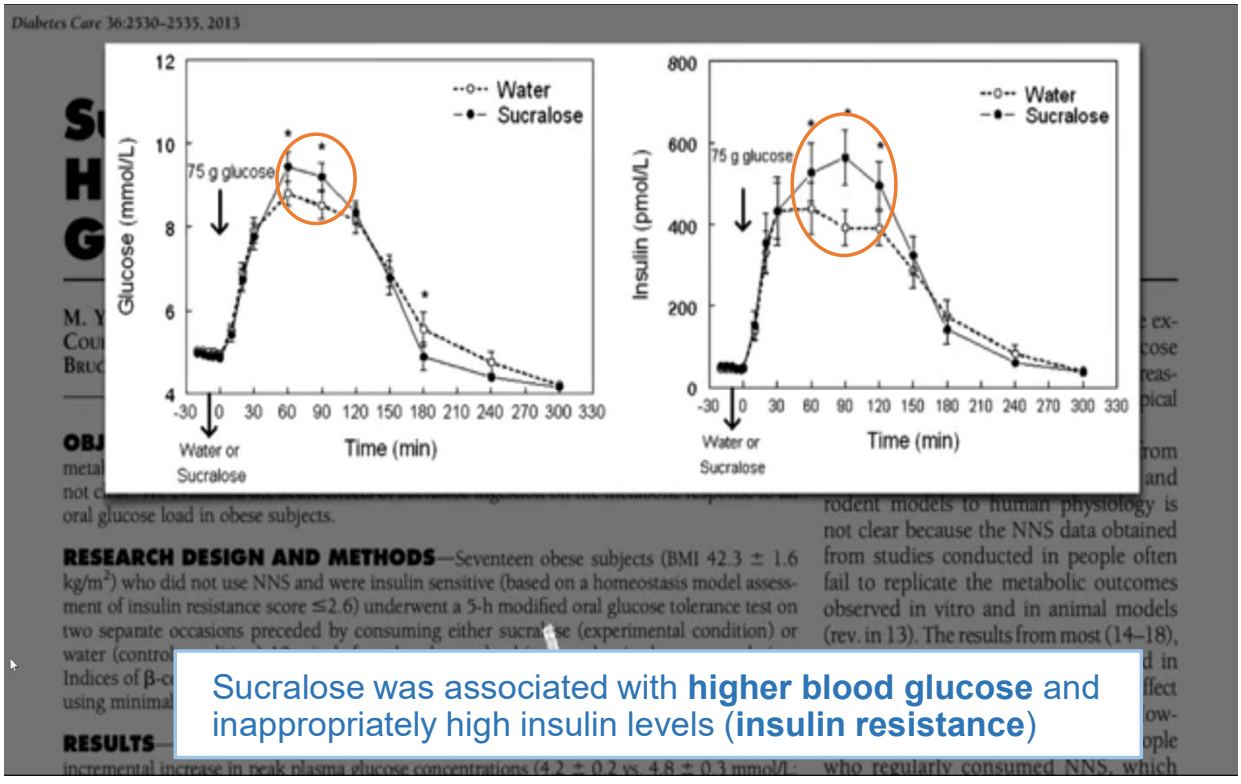
OBJECTIVE—Nonnutritive sweeteners (NNS), such as sucralose, have been reported to have metabolic effects in animal models. However, the relevance of these findings to human subjects is not clear. We evaluated the acute effects of sucralose ingestion on the metabolic response to an oral glucose load in obese subjects.

RESEARCH DESIGN AND METHODS—Seventeen obese subjects (BMI 42.3 ± 1.6 kg/m^2) who did not use NNS and were insulin sensitive (based on a homeostasis model assessment of insulin resistance score ≤2.6) underwent a 5-h modified oral glucose tolerance test on two separate occasions preceded by consuming either sucralose (experimental condition) or water (control condition) 10 min before the glucose load in a randomized crossover design. Indices of β-cell function, insulin sensitivity (S_I), and insulin clearance rates were estimated by using minimal models of glucose, insulin, and C-peptide kinetics.

RESULTS—Compared with the control condition, sucralose ingestion caused *1*) a greater incremental increase in peak plasma glucose concentrations (4.2 ± 0.2 vs. 4.8 ± 0.3 mmol/L; $P = 0.03$), *2*) a 20 ± 8% greater incremental increase in insulin area under the curve (AUC) ($P < 0.03$), *3*) a 22 ± 7% greater peak insulin secretion rate ($P < 0.02$), *4*) a 7 ± 4% decrease in insulin clearance ($P = 0.04$), and *5*) a 23 ± 20% decrease in S_I ($P = 0.01$). There were no significant

glucose absorption by upregulating the expression of sodium-dependent glucose transporter isoform 1 (5,10,11) and increasing the translocation of GLUT2 to the apical membrane of intestinal epithelia (12).

The relevance of the findings from studies conducted in cell systems and rodent models to human physiology is not clear because the NNS data obtained from studies conducted in people often fail to replicate the metabolic outcomes observed in vitro and in animal models (rev. in 13). The results from most (14–18), but not all (19,20), studies conducted in people have found that NNS do not affect plasma glucose, insulin, or GLP-1. However, these studies did not exclude people who regularly consumed NNS, which could have chronic effects on glucose metabolism (5,10,11) that would blunt any



Sucralose was associated with **higher blood glucose** and inappropriately high insulin levels (**insulin resistance**)

Nature Reviews Endocrinology 10, 637 (2014)

Not so sweet—artificial sweeteners can cause glucose intolerance by affecting the gut microbiota

Claire Greenhill

Although NASs are not absorbed, they do contact the *gut microbiota*, which is now known to have a range of important effects on human physiology.

the past few decades, which is probably linked to changes in human nutrition. A notable change over this period is the increased use of NAS in common foods, in an effort to reduce caloric intake and normalize blood levels of glucose.

However, "the safety and efficacy of NAS use remains controversial and

of NAS in obesity, C57Bl/6 mice were fed a high-fat diet with or without saccharin.

receiving NAS and the control mice no longer had different levels of glucose intolerance, both in the lean and obese states. This result suggests that glucose intolerance induced by NAS is mediated by changes to the gut microbiota. Faecal transplantation was used to determine whether the gut microbiota had a causal

be linked to susceptibility to the metabolic syndrome," say Segal and Elinav.

fasting blood levels of glucose. Similarly to the findings in mice, 16S ribosomal RNA gene sequencing showed that participants who consumed NAS had a different microbiota composition compared with those who did not consume NAS, independently of BMI.

When seven healthy human volunteers

Cell Metabolism 20, November 4, 2014, 701-703

A Bitter Aftertaste: Unintended Effects of Artificial Sweeteners on the Gut Microbiome

dietary sugar alternative meant to stave off the risk of obesity and diabetes appear to increase disease risk due to microbial alterations

Microbial communities populate the mammalian gastrointestinal tract, closely associating with the host throughout its life span. The gut is an important site for metabolic and immune regulation, and microbial cells here substantially outnumber human cells in the entire

both human physiology and our microbial inhabitants. In parallel with modernization, rates of noncommunicable, "post-modern" diseases—such as diabetes, obesity, allergies, and asthma—have increased alarmingly (Blaser and Falkow, 2009). To combat this trend without

ulum given to the germ-free recipients, rather than to direct effects due to treatment. This approach has been employed with great success in defining how intestinal microbiota influence host metabolism under conditions of disturbance, such as comparing obese versus lean individuals

9 OCTOBER 2014 | VOL 514 | NATURE | 181-186

Artificial sweeteners induce glucose intolerance by altering the gut microbiota

"our results link NAS consumption, dysbiosis and metabolic abnormalities, thereby calling for a reassessment of massive NAS use."

ations to the intestinal microbiota. These NAS-mediated deleterious metabolic effects are abrogated by antibiotic treatment, and are fully transferrable to germ-free mice upon faecal transplantation of microbiota configurations from NAS-consuming mice, or of microbiota anaerobically incubated in the presence of NAS. We identify NAS-altered microbial metabolic pathways that are linked to host susceptibility to metabolic disease, and demonstrate similar NAS-induced dysbiosis and glucose intolerance in healthy human subjects. Collectively, our results link NAS consumption, dysbiosis and metabolic abnormalities, thereby calling for a reassessment of massive NAS usage.

Non-caloric artificial sweeteners (NAS) were introduced over a century ago as means for providing sweet taste to foods without the associated high energy content of caloric sugars. NAS consumption gained much

drinking water of lean 10-week-old C57Bl/6 mice (Extended Data Fig. 1a). Since all three commercial NAS comprise ~5% sweetener and ~95% glucose, we used as controls mice drinking only water or water supple-

European Journal of Clinical Nutrition (2012) **66**, 972

GUT bacteria and aspartame: why are we surprised?

That artificial sweeteners can modify the gut microbiome is no surprise. Even small concentrations can affect the gut biology with subsequent physiologic effects.

direct contact with the sweetener and its metabolic compounds. During obesity or periods of weight management regimes, where patients might use APM (as part of their management program), it is perhaps more crucial to have optimum bacterial community

frequently been under vigorous scientific discussion. Currently, it is still approved by the FDA, as well as the EFSA; even though on consumption, each molecule of APM releases a molecule of methanol, which metabolizes into a molecule of formaldehyde.[3] Formaldehyde (which is a highly reactive substance) is classified as a known human carcinogen, with no safe level of consumption. Therefore, it is not unexpected that very small amounts of the

E-mail: resia.pretorius@up.ac.za

REFERENCES

1 Wu GD, Chen J, Hoffmann C, Bittinger K, Chen YY, Keilbaugh SA *et al.* Linking long-term dietary patterns with gut microbial enterotypes. *Science* 2011; **334**: 105–108.
2 Gophna U. Microbiology. The guts of dietary habits. *Science* 2011; **334**: 45–46.

European Journal of Clinical Nutrition (2012) 66, 972

GUT bacteria and aspartame: why are we surprised?

(NutraSweet | Equal)

direct contact with the sweetener and its metabolic compounds. During obesity or periods of weight management regimes, where patients might use APM (as part of their management program), it is perhaps more crucial to have optimum bacterial community

Each molecule of aspartame releases a molecule of methanol which metabolizes to formaldehyde which kills gut bacteria – changing the gut microbiome.

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2 Gophna U. Microbiology. The guts of dietary habits. *Science* 2011; **334**: 45–46.

Nutrition 29 (2013) 1293–1299

Non-nutritive sweeteners: Review and update

Padmini Shankar Ph.D., R.D.[a,*], Suman Ahuja Ph.D.[b], Krishnan Sriram M.B.B.S., F.R.C.S(C), F.A.C.S.[c]

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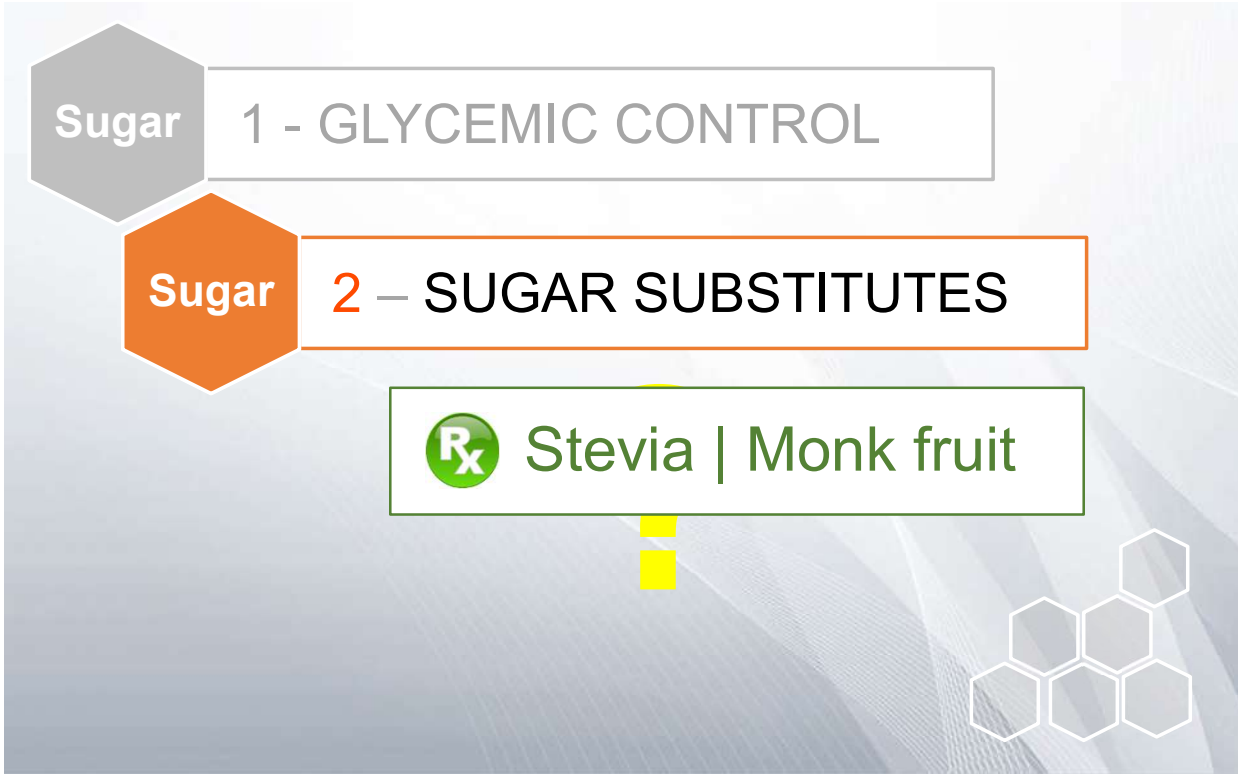
[b] *Department of Cooperative Research, Human Nutrition/Obesity Research, Lincoln University, Jefferson City, MO, USA*
[c] *Division of Surgical Critical Care, Surgical Nutrition Section and Nutrition Support Team, Stroger Hospital of Cook County, Chicago, IL, USA*

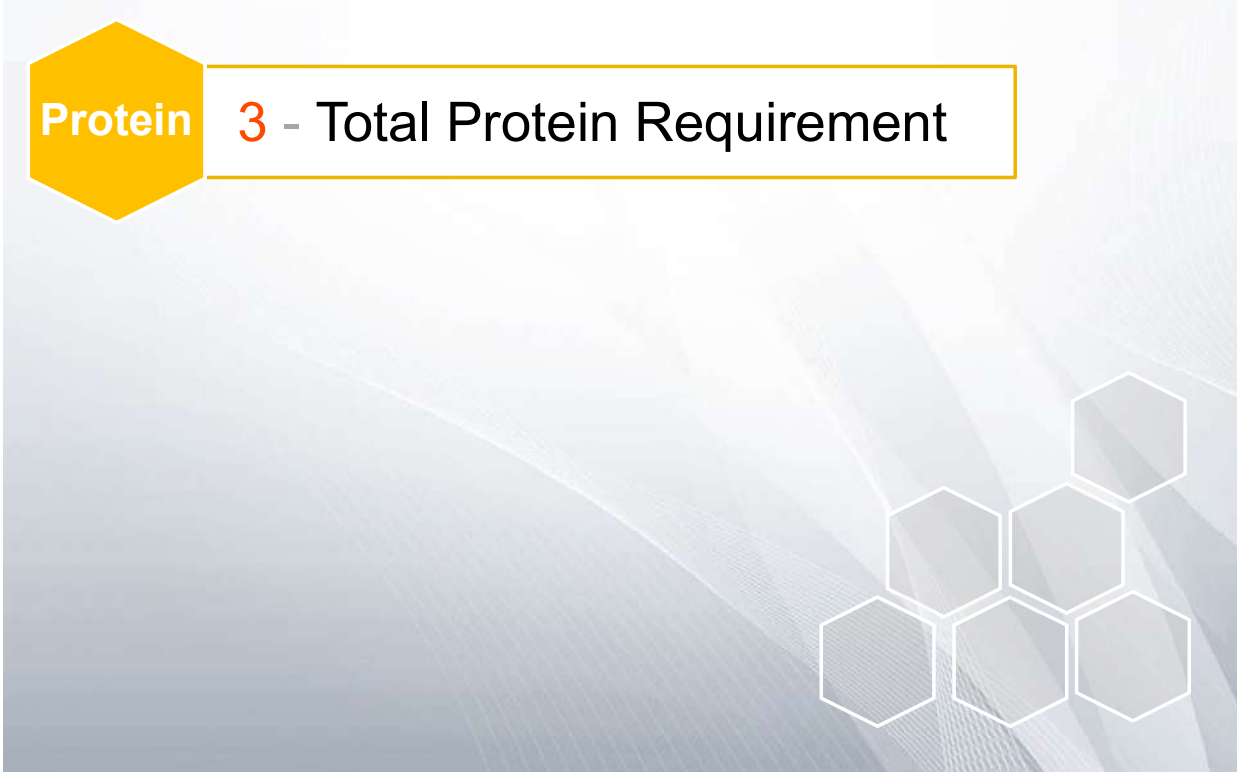
There are mixed reports about the safety of aspartame. All of the studies funded by industry vouch for its safety. In contrast, *92% of independent studies report adverse health effects*.

all ages. A systematic review of several databases and reliable websites on the internet was conducted to identify literature related to NNS. Keywords that were used individually or in combination included, but were not limited to, artificial sweeteners, non-nutritive sweeteners, non-caloric sweeteners, obesity, sugar substitutes, diabetes, and cardiometabolic indicators. The


Sugar
1 - GLYCEMIC CONTROL
Sugar
2 – SUGAR SUBSTITUTES
Stevia | Monk fruit


Nutritional Therapy: Top Ten Tenets
For Wound Care
Sugar
Vitamins
Minerals
Fat
Protein
Water

Protein 3 - Total Protein Requirement



Nutrition

Historically, clinicians used serum protein levels, including albumin and pre-albumin, to determine nutritional status. *However, current research indicates serum protein levels are affected by inflammation, renal function, hydration, and other factors*

During periods of inflammatory stress, albumin and prealbumin levels drop because they are negative acute-phase reactants.

Nutrition

- Elevated energy expenditure and *catabolism of lean body mass* are associated with chronic inflammation.
  - Acute-phase inflammatory response triggers a sequence of reactions leading to *elevated energy expenditure* and *nitrogen excretion*, which increases energy and protein requirements concurrently with anorexia and pathologically altered utilization of nutrients.

Rx 1.5-2.0 gm/kg/day IBW (100-140 gm)

Protein 3 - Total Protein Requirement

Protein 4 - Arginine (CE amino acid)

Protein 4 - Arginine (CE amino acid)

Conditionally amino acids are usually not essential, *except in times of illness and stress*; hence the term "conditionally essential" (CE). There are 8 CE amino acids.

Essential amino acids cannot be made by the body and must come from food. There are 9 essential amino acids.

Protein 4 - Arginine (CE amino acid)

- Numerous effects on wound **healing** and **immune** function.
- It is a precursor to proline – required for **collagen** synthesis
- It is a precursor for ornithine – required for NO synthesis
- Increased lymphocyte mitogensis and activity occurs.
- Multiple studies show supplemental arginine **accelerates healing** by increasing collagen deposition in wounds.

Protein 4 - Arginine (CE amino acid)

- Impaired healing with diabetes and malnutrition are associated with *low wound NO levels*.
- Inhibition of NO synthesis in wounded animals results in weaker wounds and decreased collagen synthesis.

Protein 3 - Total Protein Requirement

Protein 4 - Arginine (CE amino acid)

Protein 5 - Glutamine (CE amino acid)

Protein 5 - Glutamine (CE amino acid)

- Glutamine is the most abundant amino acid in plasma and is a primary energy source for rapidly proliferating cells.
- Glutamine supplementation **decreases infectious complications**. (Wischemeyer PE, 2001)
- Glutamine **protects against inflammatory injury** by inducing the expression of heat shock proteins - providing cellular protection from inflammation, injury, and stress. (Wischemeyer PE, 2001)
- Glutamine can modulate and **preserve gut function**, which is compromised in severe stress. (Ward E, 2009)

Protein 4 - Arginine (CE amino acid)

Protein 5 - Glutamine (CE amino acid)

Beyond daily protein requirements, specific amino acid supplements may provide benefit

Nutritional Therapy: Top Ten Tenets

For Wound Care

FAT No clear consensus

Nutritional Therapy: Top Ten Tenets

For Wound Care

Vitamins Minerals

6 - Vitamin C

Vitamins Minerals

6 - Vitamin C

RDA to prevent disease vs "optimal" RDA?

Year	RDA Edition Publication	Vitamin C RDA for healthy adult male.
1974	8th	45 mg
1980	9th	45 mg
1989	10th	60 mg
2000	DRI for Vitamin C, Vitamin E, Selenium, and Carotenoids.	90 mg

Source: Passwater Library

Vitamins Minerals

6 - Vitamin C

Vitamin C has been shown to:

- Enhance neutrophil ***migration*** in response to chemoattractants
- Enhance ***phagocytosis*** of microbes
- Stimulate reactive oxygen species (ROS) generation and ***killing*** of microbes.

(Carr AC, Nutrients 2017)

Vitamins Minerals

6 - Vitamin C

- Mice supplemented with vitamin C improved full thickness wound healing after radiation therapy along with increased collagen and fibroblast numbers. (Jagetia GC 2007)
- PRCT: Surgical patients with pressure ulcers given large doses of ascorbic acid had significant acceleration in healing. (Taylor TV 1974)

Overall, vitamin C supplementation has consistently shown benefit to wound healing.

Vitamins Minerals

6 - Vitamin C

- ✔ *Routine supplementation beyond the RDA is recommended for cigarette smokers*
- ✔ *Opinions on "optimal" vitamin C suggest doses 3-5 X the current RDA (90 mg).*

℞ 500 mg/day slow release

Vitamins Minerals

6 - Vitamin C

Vitamins Minerals

7 - Vitamin A

Vitamins Minerals

7 - Vitamin A

Vitamin A has multiple positive effects on wound healing even in non-deficient states.

- It **increases collagen** cross-linking and breaking strength.
- It increases the inflammatory response in wounds through enhanced lysosomal membrane lability, increased macrophage influx, and stimulation of collagen synthesis.
- It increases the number of monocytes and macrophages in the inflammatory phase and facilitating epithelial cell differentiation.
- Importantly, it **reverses corticosteroid-induced inhibition** of cutaneous wound healing.

Vitamins Minerals

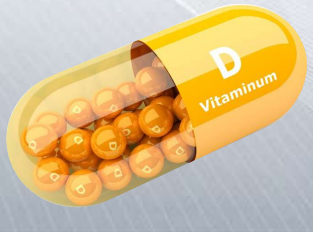
7 - Vitamin A

Rx 10,000 units for 4 weeks

Vitamins Minerals 6 - Vitamin C

Vitamins Minerals 7 - Vitamin A

Vitamins Minerals 8 - Vitamin D



Vitamins Minerals 8 - Vitamin D

- Vitamin D is more than a simple vitamin. Research has shown that vitamin D is most likely the oldest hormone
- All human cells have vitamin D receptors and the receptor effect is cell dependent
- Normal levels of serum vitamin D levels have been shown to positively affect a number of diseases such as cancer, heart disease, diabetes, hypertension, autoimmune diseases, and insufficiency fractures.

Vitamins Minerals

8 - Vitamin D

- Low levels of vitamin D associated with development of diabetic foot infections. (Yakob 2014)
- PRCT Vitamin D - Positive effects on T-cell-mediated **immunity**, **insulin secretion** and **receptor action**, cell **growth** and **healing**. (Asemi 2013)
- Vitamin D restoration of antimicrobial peptide production and improved in vitro wound-healing assays.(Gonzalez-Curiel 2014)

Effects of vitamin D supplementation on glucose metabolism, lipid concentrations, inflammation, and oxidative stress in gestational diabetes: a randomized, double-blind trial. Asemi, Z, et al. Am. J. Clin. Nutr. 2013: 98; 1425–1432.

Vitamins Minerals

8 - Vitamin D

- In rats, the topical application of vitamin D **accelerated wound healing** in a dose-dependent manner. (Tian 1995)
- PRCT: Vitamin D supplementation associated with **improved healing**. (Razzaghi 2017)
- Vitamin D promotion of endothelial and keratinocyte cell migration in a DFU model. (Trujillo 2017)

The effects of vitamin D supplementation on wound healing and metabolic status in diabetic foot ulcers: A randomized, double-blind, placebo-controlled trial. Razzaghi R, et al. J Diabetes Complications. 2017 Apr;31(4):766-772.

Vitamins Minerals

8 - Vitamin D

Dai et al. *Nutrition and Diabetes* (2019)9:8
https://doi.org/10.1038/s41387-019-0078-9

Nutrition & Diabetes

REVIEW ARTICLE Open Access

Vitamin D and diabetic foot ulcer: a systematic review and meta-analysis

Jiezhi Dai[1], Chaoyin Jiang[1], Hua Chen[1] and Yimin Chai[1]

Abstract

Seven studies (1115 patients) were analyzed. Severe vitamin D deficiency is significantly associated with ulceration. Odds Ratio 3.2

Vitamins Minerals

8 - Vitamin D

10,000 units for 4 weeks than 4,000 units there after

Vitamins Minerals 6 - Vitamin C

Vitamins Minerals 7 - Vitamin A

Vitamins Minerals 8 - Vitamin D

Vitamins Minerals 9 - Zinc

Vitamins Minerals 9 - Zinc

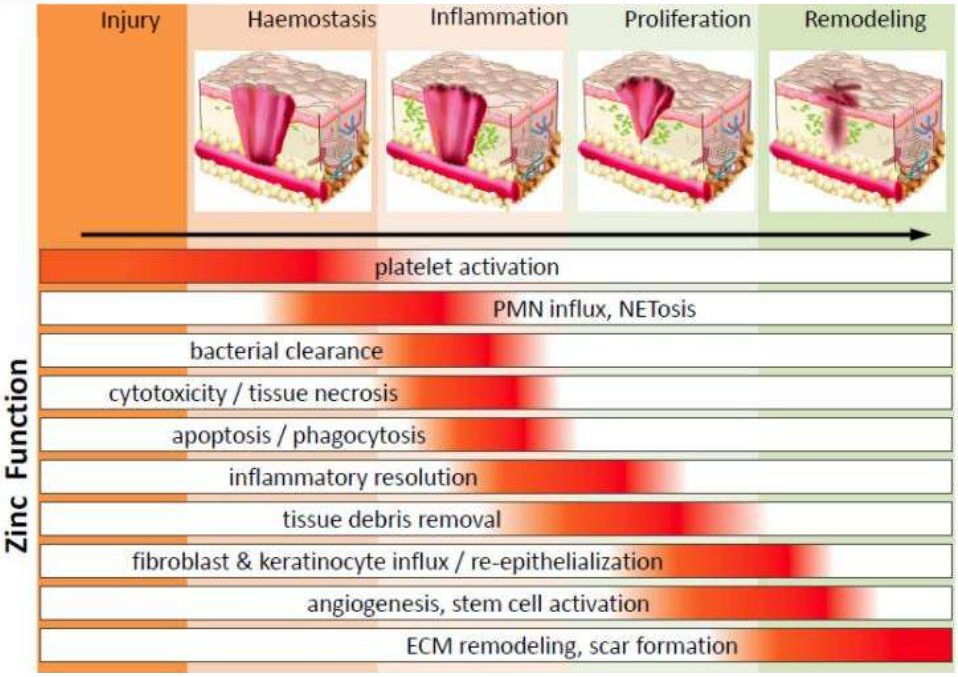
- Zinc serves as a cofactor in 3,000+ enzyme systems
- Zinc deficiency is associated with poor wound healing
- Zinc-dependent matrix metalloproteinases augment *autodebridement* and *keratinocyte migration.*
- Zinc confers resistance to epithelial apoptosis through *cytoprotection* against bacterial toxins and reactive oxygen species

*Demographic: A study of 600 nursing home residents found **50%** with low zinc levels.*

Vitamins Minerals

9 - Zinc

Summary of Zinc Functions



Vitamins Minerals

9 - Zinc

5 mg/day

Nutritional Therapy: Top Ten Tenets

For Wound Care

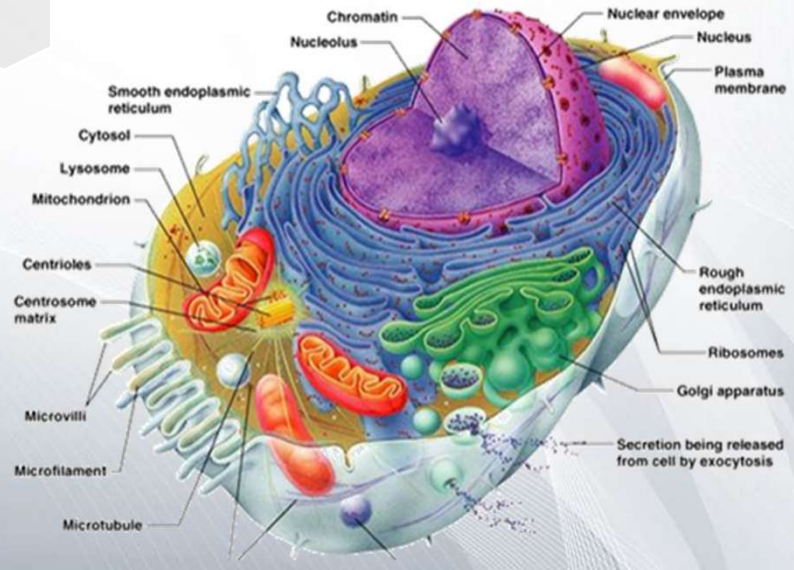
Water 10 - Hydration

30 ml/kg/day (~ 2 liters)

Increase necessary with elevated temp, vomiting, sweating, diarrhea, or heavily draining wounds.

SUMMARY

You can't build cells with antibiotics & dressings



SUMMARY

K I S Keep It Simple

SUMMARY

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Protein Supplement | Vitamin D | Vitamin C

NestléHealthScience

Gluten free / Lactose free

SUMMARY

K I S Keep It Simple

CE Amino Acids | CHMB

Abbott Nutrition

Arginine = Nitric Oxide
Glutamine = Collagen
CHMB* = Protein Synthesis

*calcium ß-hydroxy-ß-methylbutyrate

SUMMARY

- **Sugar** — Glycemic Control (Job 1) | Natural Sugar Substitutes
- **Protein** — 100 gm/day (Minimum) + Arginine & Glutamine
- **Vitamins A - C - D** — Vitamin A & D 10,000 IU/day | Vitamin C 500 mg/day
- **Water** — 2 liters (Qts) / day*