Nutrients to Reverse Aging

Thierry Hertoghe, MD

Physical Aging = Amino acid + Nutritional deficiencies

- Diffuse hair loss all over head
- Zinc, iron, silicon, vit. A, B5, B7, Cysteine, methionine, cystine, tyrosine
- Upper head (vertex) hair loss
- Excess meat intake
- Thin hair lacking volume
- Cysteine, methionine, cystine, tyrosine
- Gray, white hair
- Copper, B12, PABA, Tyrosine, Cysteine, phenylalanine
- Shorter hair
- Forehead Wrinkles

Amino acid deficiency

Droopy nose tip
Slagging cheeks
Nasal fold
Thinner lips
Skinfolds under the chin
Muscle hypotrophy Valine, Leucine, arginine, glutamine, del. Amino acid deficiency
Thickened skin elasticity
Age spots Selenium, vit. C, A, E, (prorenogen) deficiencies

What Nutrients may Reverse Aging?

Nutrients that (may)
- prolong life
- stimulate hormone productions

Physical Aging = Amino acid + Nutritional deficiencies

- Sunken eyes Water, sodium (intake) deficiency
- Dry eyes Vit. A, Omega-6 deficiencies
- Enflamed eye globe (Scleritis) Vit. B2, B6 deficiency
- Enflamed eye borders (Col infectious) Omega-3 defic.
- Enflamed eye globes (Col infectious) Omega-3 defic.
- Dropy upper/lower eyelids IGF-1/GH deficiency
- Acne foot wrinkles Water, sodium (intake) deficiency
- Swollen lower eyelids Iodine deficiency
- AGING FACE Amino acid + nutritional deficiencies
- Lack of sun tan
- Tyrosine, vit. D deficiencies
- Slagging cheeks
- Nasal fold
- Thinner lips
- Skinfolds under the chin

Physical Aging = Amino acid + Nutritional deficiencies

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- Zinc, iron, silicon, vit. A, B5, B7, Cysteine, methionine, cystine, tyrosine
- Dry hair Zinc, omega-3 fatty acid, vit. A deficiencies
- Split hair Iron, silicone deficiencies
- Upper head (vertex) hair loss
- Excess meat intake
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• Shorter hair

Forehead Wrinkles
• Wrinkled nose
• Hallowing cheeks
• Droopy nose tip
• Enflamed eye borders ( Conjunctiva)
• Crow's feet wrinkles
• Sunken eyes
• Forehead Wrinkles
• Enlarged, sunken eye sockets
• Enflamed eye globe (Scleritis)
• Skinfolds under the chin
• Muscle hypotrophy
• Wrinkled skin

Very small fleshes in the skin

AGING FACE
• Lack of sun tan
• Droopy nasal bridge
• Very small fleshes in the skin
• Skinfolds under the chin
• Thinner lips

Dry hair
• Enflamed eye borders ( Conjunctiva)

Very small fleshes in the skin
• Skinfolds under the chin

図 57x568 to 288x742
図 310x335 to 539x479
図 322x135 to 520x229

Reversal of Physical Aging

 Longer Life
 Nutrients to Reverse Aging

Vit. A ...

Vit. A Treatment

⇒ ↑ Thyroid, IGF-1, Testosterone

Reversal of Physical Aging

⇒ Amino acid + Nutritional Supplements

Reversal of Physical Aging

Longer Life
Nutrients to Reverse Aging

Vit. A Supplements
⇒ ↓ Mortality in children w/ pneumonia

VIT. A SUPPLEMENTS
400,000 IU (or half that for infants)

Figure: Vitamin A supplements, a low cost intervention, reduce mortality of HIV-infected children.

(667 children ages 6 months to 3 years w/ pneumonia, children who were severely malnourished or had clinical signs of vitamin A deficiency were excluded; mean follow-up = 24.4 months)


Vit A => ↑ IGF1 mRNA

Response of the insulin-like growth factor system to vitamin A depletion and repletion in rats.

Vit. A Treatment

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Reversal of Physical Aging

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Response of the insulin-like growth factor system to vitamin A depletion and repletion in rats.
VIT. A => TESTOSTERONE

Figure: decreased testosterone steroidogenesis in testicular tissue of rats fed a vit. A: < 10 μg vit.A/per gram wet liver weight


↓ Serum level of oxygenated carotenoids in diets => ↑ mortality

Beta-carotene & other carotenoids...

Dietary Intake of Vitamin B6 => ↓ Mortality.

SUBJECTS: 23 119 men and 35 611 women, age 40 to 79 years, completed a food frequency questionnaire in the Japan Collaborative Cohort Study. During the median 14-year follow-up, there were 986 deaths from stroke, 424 from coronary heart disease, and 2087 from cardiovascular disease.

RESULTS: Dietary folate & vitamin B(6) intakes => inversely associated with mortality from heart failure for men and with mortality from stroke, coronary heart disease, and total cardiovascular disease for women.

These inverse associations did not change materially after adjustment for cardiovascular risk factors. No association was found between vitamin B(12) intake and mortality risk.

B vitamin supplementation => sign. protective effect on stroke, but none on the risk of CVD, MI, CHD, cardiovascular death, or all-cause mortality.

STUDIES: 19 studies including 47921 participants

RESULTS: The overall relative risks with 95% CI of outcomes for patients treated with B vitamin supplementation compared with placebo = 0.98 (0.94-1.03) for CVD, 0.98 (0.92-1.05) for coronary heart disease (CHD), 0.97 (0.90-1.05) for myocardial infarction (MI), 0.88 (0.82-0.95) for stroke, and 0.97 (0.91-0.92) for cardiovascular death, 0.99 (0.95-1.04) for all-cause mortality. Blood Hcy levels were ↓ in all included RCTs.

CCL: B vitamin supplementation has a significant protective effect on stroke, but none on the risk of CVD, MI, CHD, cardiovascular death, or all-cause mortality.


Vit. B6 Treatment

⇒ ↑ GH, Melatonin, Testosterone, (Estradiol?)

Vitamin B6

⇒ ↑ night peak of serum GH in women

SUBJECTS: 10 normal women
TREATMENT: 2x 300 mg/day vitamin B6 in 24 h
• unchanged circadian rhythm of serum ACTH & cortisol
• Nonsign. slightly lower serum prolactin,
• sign. higher night peak of growth hormone (p. 0.05)
MECHANISM: The effect of vitamin B6 is likely to be mediated by dopaminergic receptors at hypothalamic level as previous studies by other authors appear to prove.


ViT. B6 inj.

⇒ ↑ serum melatonin in rats

SUBJECTS: 30 laboratory rats ⇒ 2 groups – control group & experimental group + vit. B6 inj. ⇒ Every other day at 22h, melatonin concentration ⇒ for 2 months.
VIT. B6 TREATMENT: vit. B6 inj. after 2 months
⇒ + 35.95% ↑ plasma melatonin at 22h
CCL: Vit. B6 injections strengthens melatonin biosynthesis; Vit. B6 deficiency, may cause disease by reduction of endogen melatonin production.


↓ Vit. B6 ⇒ ↓ serum testosterone in rats

Fig.: vit. B6 deficiency resulted in an increased & prolonged nuclear retention of the steroid. The results suggest that vit. B6 has a th in the action of testosterone ($\&$ other steroid hormones), possibly in the recycling of receptors from the nucleus back into the cytosol after initial translocation

Vit. B9
Dietary intake of Folic acid => ↓ mortality.

SUBJECTS: 23119 men and 35611 women, age 40 to 79 years, completed a food frequency questionnaire in the Japan Collaborative Cohort Study. During the median 14-year follow-up, there were 986 deaths from stroke, 424 from coronary heart disease, and 2087 from cardiovascular disease.

RESULTS: Dietary folate & vitamin B(6) intakes were inversely associated with mortality from heart failure for men and with mortality from stroke, coronary heart disease, and total cardiovascular disease for women. These inverse associations did not change materially after adjustment for cardiovascular risk factors. No association was found between vitamin B(12) intake and mortality risk.


↑ Serum Vit. B12 => ↑ mortality but unclear assoc.

DESIGN: Retrospective cross-sectional study.

PARTICIPANTS: 1570 patients aged 65 & older hospitalized:

RESULTS: Vit. B12 deficiency (<or=200 pmol/L) was found in 15% of older hospitalized patients. 50% of the patients had high vitamin B12 levels (>or=350 pmol/L), 68.2% of whom were aged 80 and older. Metformin use was clearly associated with lower vitamin B12 levels. In patients aged 65 and older, an inverse correlation was found between vit. B12 levels & albumin, metformin, & angiotensin-converting enzyme (ACE) inhibitor use. "Age," number of medications, & mortality => linearly correlated with vit. B12 levels.

CCL: ↑ vit. B12 levels => ↑ mortality, but it is unclear whether vit. B12 = marker or a surrogate marker or even a substance that directly causes death.


↑ Vit. B12 in diet => ↓ mortality in men & women

High vitamin B12 in the diet
-14% in women
-26% in men
but not from cerebrovascular disease


↑ Serum Vit. B12 => ↑ light-induced phase-shift by Melatonin

DESIGN: 8 young males => single blind cross-over test to see the effects of vit. B12 (methylcobalamin; VB12) => phase-response of the circadian melatonin rhythm to a single bright light exposure.

TREATMENT: IV of Vit. B12 (0.5 mg/day) at 12h30 for 11 days => after:
oral (3x 2 mg/day) for 7 days (vs placebo)
• Blood sampling => under dim light condition (< 200 lx) & plasma melatonin before & after a single bright light exposure (2500 lx for 3 h) at 0700 h.
• smaller amplitude of the melatonin rhythm before the light exposure in the Vit. B12 trial
• The light exposure phase-advanced the melatonin rhythm sign, in the Vit. B12 trial, but not in the placebo.

Vit. C ...

Large doses of Vit. C => ↑ survival & ↑ quality of life of terminal cancer patients

SUBJECTS: a healthy male volunteer.
TREATMENT: Vitamin C infusion
• initial to moderate stages: distinct ↓ plasma cortisol & vit. C from the circulation
• middle stage: a small ↑ plasma cortisol
• terminal stage: skyrocket-like ↑↑ plasma ACTH & cortisol

Methylprednisolone in vit. C infusion
• initial to moderate stages: suppressed the depletion of plasma vitamin C & cortisol of the initial tomoderate stages.
• middle stage: No effect on the small surge of plasma cortisol.
• terminal stage: ↓ ↓ completely suppressed the emergence of the plasma ACTH/cortisol surges of the terminal stage


ACTH, Cortisol, Estradiol

Vit C Treatment

Vit. C supplements => partially recovered testosterone levels in hyperglycemic rats

SUBJECTS: hyperglycemic adult male rats => 4 experimental groups:
hyperglycemic control (Hy), hyperglycemic + 150 mg/day vitamin C (HyC), hyperglycemic + 100 mg/day vitamin E (HyE) or hyperglycemic + vitamins C and E (HyCE); normoglycemic group (n = 10) received only the vehicles

RESULTS:
• Both vitamins => sign. ↓ TBARS (thiobarbituric acid reactive species) level in the hyperglycemic groups.
• sign. ↓ testosterone level in the Hy and HyE groups when compared to the normoglycemic group (n = 10) received only the vehicles
• ↑ sensitivity of the α-1 adrenoceptor in the vas deferens of the hyperglycemic control group was observed.

CCL: Vit. C treatment => more efficient than vitamin E in attenuating the effects of hyperglycemia on the male reproductive system of adult rats.

Co Q10 ...

Elderly sick patients with ↓ serum coenzyme Q 10

⇒ ↑↑ mortality

Mortality at 6 months (%)

<table>
<thead>
<tr>
<th>Patients with low serum coQ10</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 0.55 μg/ml</td>
</tr>
<tr>
<td>53 %</td>
</tr>
</tbody>
</table>

A low CoQ10 level was found in patients with chemotherapy, cytotoxic or on cholesterol-lowering agents, or suffering from heart failure or severe myopathies

n = 94

Figure: Severe increase of mortality at 6 months in patients wiserum coenzyme Q10 under one standard deviation of the mean of a population of patients over age 50 (mean age = 72 yrs)

W. Judkins. Statistic data support prediction of death within six months in low levels of coenzyme Q10 and other factors. Cite Heart. 1994; 71: 317-9

Vit. D

Vit. D supplements ⇒ ↓ mortality

- Ecological & observational studies ⇒ low vitamin D status ⇒ could be associated with higher mortality from life-threatening conditions including cancer, cardiovascular disease, and diabetes mellitus that account for 60% to 70% of total mortality in high-income countries.

- RESULTS: 18 independent randomized controlled trials, incl. 57,311 participants. A total of 4777 deaths from any cause occurred during a trial size-adjusted mean of 5.7 years. Daily doses of vitamin D supplements varied from 300 to 2000 IU. The trial size-adjusted mean daily vitamin D dose was 528 IU. In 9 trials, there was a 1.4-5.2 fold difference in serum 25-hydroxyvitamin D between the intervention & control groups. The summary relative risk for mortality from any cause was 0.93 (95% CI, 0.87-0.99). There was neither indication for heterogeneity nor indication for publication biases. The summary relative risk did not change according to the addition of calcium supplements in the intervention.

- CONCLUSIONS: Intake of ordinary doses of vitamin D supplements seems to be associated with decreases in total mortality rates. Population-based, placebo-controlled randomized trials with total mortality as the main end point should be organized for confirming these findings.


CCL: A nonlinear decrease in mortality risk as serum 25(OH)D ↑, + optimal concentrations 75-87.5 nmol/L.
**Vit. D Treatment**

- \( \uparrow \) IGF-1
- \( \uparrow \) or \( \downarrow \) PTH; \( \uparrow \) calcitonin

2 µg/day CALCITRIOL =>
=> \( \uparrow \) serum IGF-1 in osteoporotic women

**SUBJECTS:** 18 osteoporotic women \([1,25(OH)2D3] \Rightarrow 2 \text{ doses (1µg/day & 2 µg/day)} \text{ for 14 days}

**The biological effectiveness of the treatment => controlled by ↓ serum intact parathyroid hormone**

**TREATMENT:** CALCITRIOL 1,25(OH)2 vitamin D3 ---
- ↑ sign. plasma IGF-I, beta 2 microglobulin, & serum osteocalcin => effects were only apparent after the higher dose of the drug (169 vs 134 ng/ml, \( P < 0.01 \); 2.08 vs 1.92 µg/ml, \( P < 0.05 \); & 8.5 vs 5.4 ng/ml, \( P < 0.01 \), resp.).

**CCL:** exogenous 1,25(OH)2D3 => ↑ production of IGF-I & beta 2 microglobulin in osteoporotic patients in parallel to the marker of osteoblastic function, osteocalcin


**Vit. E Treatment**

\( \uparrow \) Serum α-tocopherol => \( \Rightarrow \) ↓ total stroke mortality & hemorrhagic stroke mortality in women;\( \uparrow \) Serum γ-tocopherol => ↓ ischemic stroke mortality in men

**SUBJECTS:** 39 242 participants (age range, 40-79 years) between 1988 & 1990. During 13-year follow-up => 530 stroke deaths (302 ischemic strokes & 210 hemorrhagic strokes) & 211 deaths from coronary heart disease. Controls were matched for sex, age, area of residence (case-control study)

**RESULTS:** Serum α-tocopherol => no ass in men => in women \( \Rightarrow \) inversely assici. w/ total stroke mortality & hemorrhagic stroke mortality. RR for highest vs lowest quintile of serum α-tocopherol in women = 0.35 (0.16-0.77; \( P = 0.009 \)) for total stroke & 0.26 (0.07-0.97; \( P = 0.048 \)) for hemorrhagic stroke.

Serum γ-tocopherol was inversely associ. w/ ischemic stroke mortality in men but positively associated with hemorrhagic stroke mortality in women. The respective multivariate odds ratios (95% CI) for the highest versus the lowest quintile and for a 1 standard deviation increment in γ-tocopherol level were 0.48 (0.22-1.06; \( P \text{ for trend} = 0.07 \)) & 0.77 (0.58-1.02), respectively, for ischemic stroke in men and 3.10 (0.95-10.12; \( P \text{ for trend} = 0.052 \)) & 1.49 (1.04-2.13) for hemorrhagic stroke in women.


**Vit. E**

2 000 IU Vitamin E/day =>
=> ↓ mortality in Alzheimer’s disease patients

**SUBJECTS:** 847 probable or mixed AD patients followed in a research center between 1990 and the censoring date of December 31, 2004. Standard practice during this period was to recommend vitamin E at 1,000 IU twice daily to all patients.\( \Rightarrow \) Approx. 2/3 of the patients took vitamin E with a cholinesterase inhibitor (ChEI), I, 10% took vitamin E alone, & 15% took no antideementia drug.

**RESULTS:** RR of dying
- - 29% with vitamin E use (with or without a cholinesterase inhibitor (ChEI)) \( \Rightarrow \) (0.71; 95% CI: 0.57-0.89; \( P = 0.003 \))
- - 23% with vitamin E alone or with another drug was 0.77 (95% CI: 0.66-1.0)
- - 20% for cholinesterase inhibitor use compared to the no drug treatment group (95% CI: 0.67-1.60)


**Vit. E Treatment**

\( \Rightarrow \), Thyroid, DHEA
Vit E => restores Thyroid activity after oxidative damage, by inc. deiodinase.

Thyroid activity

Ca ...

↓ urinary calcium => ↑ mortality

Kobayashi E, Okubo Y, Suwazono Y, Kid T, Nishi M, Nakagawa H, Nogawa K. Association between urinary calcium excretion level and mortality in inhabitants of the Jinzu River basin area of Japan. Biol Trace Elem Res. 2002 Nov;89(2):145-53. Department of Occupational and Environmental Medicine, Graduate School of Medicine, Chiba University, Japan. A follow-up study on 5442 inhabitants (2699 men, 2743 women) was conducted to determine the association between urinary Ca excretion level and mortality of the general population of three different areas of the Jinzu River basin area, namely, non-Jinzu River, mixed, and the Jinzu River water systems, over a period of 6127 d. More than 98% of the subjects were followed completely in each area. In comparison with the low- and high-Ca excretion groups (cutoff values; 25.1 mg/dL in men, 20.4 mg/dL in women), the mortality rates per 1000 person-years and standardized mortality ratios (SMRs) tended to be greater in the low-Ca-excretion groups than in the high-Ca-excretion groups in both sexes for all the three areas. Moreover, Cox's hazard ratios in men and women of the three areas exhibited negative values, 0.99 except for men of the mixed watersystem. These values were statistically significant in both sexes for the Jinzu River water system and in women for the non-Jinzu River water system. We conclude that the life-span becomes shorter as urinary Ca excretion levels become lower. PMID: 12449238 [PubMed-indexed for MEDLINE]

Calcium Treatment

⇒ GH, IGF-1, ACTH, Cortisol, Aldosterone, Testosterone

⇒ PTH

Calcium => ↑ serum ACTH in humans

⇒ ↑ serum cortisol & aldosterone

SUBJECTS: 4 normal subjects, one patient + hypoparathyroidism & one patient + hypothyroidism & 6 normal subjects and 4 hypothyroidized patients as controls

TREATMENT: iv calcium gluconate

• a decrease in plasma renin activity but only in the normal subjects.
• higher plasma ACTH, cortisol & aldosterone were when calcium was administered.
• Plasma ACTH and cortisol responses to calcium were abolished whereas plasma aldosterone response persisted in 2 normal subjects pre-treated + dexamethasone.
• Plasma renin activity was not statistically different whether or not calcium had been injected in the subjects studied twice.

CCL: direct effect of calcium on ACTH and aldosterone secretion which is not mediated by calcitonin & parathyroid hormone. The stimulatory effect of calcium on cortisol secretion depends on the increase in plasma ACTH.


± 2 g of Calcium supplementation

⇒ free & total testosterone

SUBJECTS: 30 healthy male athletes => 3 groups:
1. Group 1 non-exercising subjects +35 mg calcium/kg body weight
2. Group 2 subjects receiving 35 mg calcium/kg body weight undergoing training routines for 90 min/day, 5 days a week
3. Group 3 subjects undergoing training routines for 90 min/day, 5 days a week

RESULTS: 4 weeks of calcium supplementation on free & total testosterone levels

• plasma free- & total testosterone levels at exercise before & after supplementation relative to resting values (p < 0.05).
• also true when active subjects were compared to inactive subjects (p < 0.05).

CCL: training results => ↑ testosterone levels in athletes and that the ↑ is greater if accompanied by calcium supplementation, which may be useful for increasing overall athletic performance

Cinar V, Baltaci AK, Mogulkoc R, Klic M. Testosterone levels in athletes at rest and exhaustion: effects of calcium
Potassium from food => stroke mortality

Potassium deficiency => GH, IGF-1

For every 100 mg/L Mg levels in drinking water => -0.248 in the RR of dying
Magnesium Treatment

 rencontrement

$\Rightarrow \uparrow$ GH, Melatonin, IGF-1, $T_3$, PTH, Testosterone

$\Rightarrow \downarrow$ Estradiol in men

Mg $\Rightarrow \downarrow$ myocardial infarct mortality

Maternal mortality from eclampsia (% of patients)

$\forall n = 1974$ patients + AAMI (1723 deaths) - magnesium, 1494 controls - magnesium, 1753 deaths data from all available randomized controlled trials of magnesium in patients + AAMI

Figure: Since the introduction of low dose magnesium sulphate to treat eclamptic patients at Dhaka Medical College, mortality rates have fallen from 16% to 8%.

Personal communications from other centres in Bangladesh show similar findings.

Two AS, Tsui SS. Role of magnesium in reducing mortality in severe myocardial infarction: a review of the evidence. Drug: 1998 Sep 6(6);447-51 Division of Cardiology, University of Alberta, Edmonton

Zinc Treatment

$\Rightarrow \uparrow$ IGF-1, $T_3$, Testosterone

$\Rightarrow \downarrow$ PTH

↑Magnesium $\Rightarrow$ ↑ bioavailable testosterone

SUBJECT: SHBG of blood of young adults $\Rightarrow$ exposed to magnesium ions.

RESULTS: Higher concentration of magnesium $\Rightarrow$ slower is the binding of testosterone to SHBG.


Zinc supplements $\Rightarrow \downarrow$ all-cause mortality

Figure: Sign. reduced mortality in participants who were randomly assigned to receive zinc supplements in Age-Related Eye Disease Study (AREDS).

Zinc stimulates IGF-1…. More than meat??

Graph: 226 Free living healthy men; 42-76 yr

Zinc => ↑serum testosterone, sperm count & fertility in men + low testosterone

Carnitine => ↓ mortality after myocardial infarction

Acetyl-L-carnitine (3 g/day) => ↑ serum IGF-1

SUBJECTS: 11 asymptomatic, HIV-1-infected subjects
TREATMENT: Acetyl-L-carnitine (3 g/day) for 5 months
- substantial impact on the main immunologic abnormality associated with HIV infection, ↓ loss of CD4 cells, by ↓ rate of apoptotic lymphocyte death.

2 imp mechanisms underlying the observed anti-apoptotic effects of acetyl-L-carnitine
- ↓ ceramide generation
- ↑ serum IGF-1, a major survival factor able to protect cells from apoptosis by different stimuli & conditions.
Acetyl-L-carnitine => ↑ serum cortisol

Acetyl-L-carnitine => acutely administered
=> raises serum beta-endorphin & cortisol in humans


Thank you for your attention!