Aadorfer Fachforum Psychotherapie

Privat Klinik Aadorf

Persönlich und diskret.

Workshop D:

Esstörungen und Mikronährstoffmangel

Esstörungen und Mikronährstoffe

- Den Esstörungen immanent ist ein dysfunktionaler Umgang mit der Nahrungsaufnahme
- Dadurch bedingt kommt es sehr häufig zu Ungleichgewichten und Mangelzuständen
- Die Grunderkrankung bedingt das Ausmass und die Richtung des Mangels
- Bei Anorexia nervosa Mangel an verschiedenen Mikronährstoffen häufig
- Auch bei Adipositas z.B. bei einer Binge-Eating-Störung Mikronährstoffmangel häufig
- In diesem Workshop werden wir uns anhand der aktuellen Studienlage mit der Diagnostik und Therapie von Mikronährstoffmangelzuständen bei Essstörungen auseinandersetzen.

Anorexia Nervosa: Allgemeine Empfehlungen zur Supplementation

World Journal of Psychiatry

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REVIEW

Anorexia nervosa: Outpatient treatment and medical management

Stein Frostad, Mette Bentz

Allgemeine Empfehlungen:

- Supplementation mit einem Multivitamin, in dem Vitamin D und Calcium enthalten ist
- Bei Bedarf Thiaminsupplementation
- Bei Bedarf Magnesiumsupplementation
- Bei Bedarf Zinksupplementation
- Bei Bedarf Phosphatsupplementation, ggf. auch I.V.
- Bei Bedarf Kaliumsupplementation
- Bei Bedarf Eisensupplementation

Anorexia nervosa: Outpatient treatment and medical management

Stein Frostad, Mette Bentz

Aber:

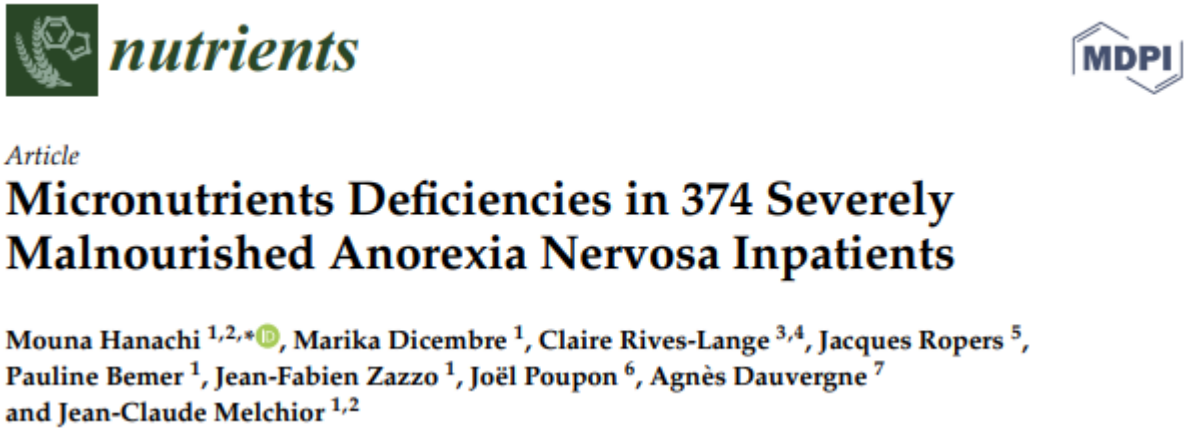
- Welche Patientengruppe profitiert von welcher Supplementation?
- Welche Labordiagnostik ist sinnvoll?
  - ➢ Genauere Untersuchungen erforderlich
  - ➢ Zwei grosse Studien mit schwer und leichter erkrankten Patientinnen

Anorexia nervosa: Outpatient treatment and medical management

Stein Frostad, Mette Bentz

Studie 1: Schwere Anorexie – BMI 12.5

Mikronährstoffmangel bei extremer AN



nutrients

MDPI

Article

Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Mouna Hanachi [1,2,*], Marika Dicembre [1], Claire Rives-Lange [3,4], Jacques Ropers [5], Pauline Bemer [1], Jean-Fabien Zazzo [1], Joël Poupon [6], Agnès Dauvergne [7] and Jean-Claude Melchior [1,2]

Paris, Frankreich

Studie 1: Extreme AN – Welche Patienten?

Table 1. Characteristics of all patients at admission and comparison between Anorexia Nervosa restricting type (AN-R) and AN binge-purging (AN-BP).

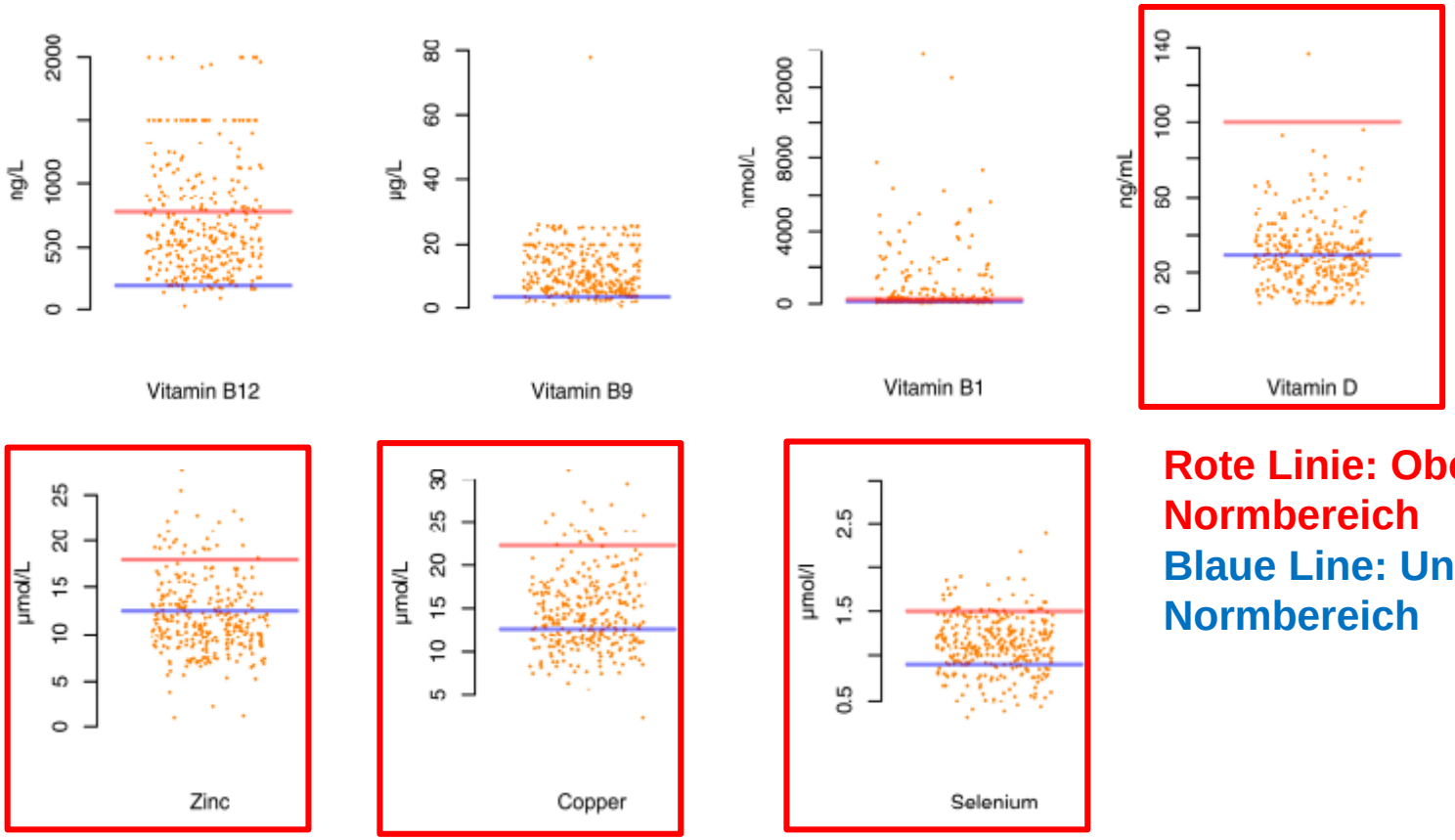
Clinical and Biological Parameters	*N*	AN-BP	AN-R	*P*
Patients (*n*, %)	374	121 (32%)	253 (68%)	
Gender	360 (96.3%) Women	117 (96.7%)	243 (96%)	NS
	14 (3.7%) Men	4 (3.3%)	10 (4.1%)	NS
Age (year)	31.3 ± 12.9	29,41 (23.73, 37.09)	26,22 (19.98, 38.29)	0.029
Weight (kg)	33.7 ± 5.9	34.40 (30.00, 39.10)	32,40 (29.60, 36.80)	0.048
Height (m)	1.6 ± 0.07	1.64 (1.60, 1.68)	1.62 (1.59, 1.68)	NS
BMI (18.5–24.9 kg/m2)	12.5 ± 1.7	12.80 (11.25, 14.20)	12.30 (11.30, 13.50)	NS
Fat Mass (g)	3098 ± 1744	2715.00 (2236.75, 4073.73)	2505.60 (2165.75, 3381.02)	NS
Fat Mass (%)	9702 ± 4057	8.60 (7.75, 12.45)	8.50 (7.40, 9.43)	NS
Disease duration (year)	9.4 ± 9.2	10.00 (5.00, 16.00)	4.50 (2.00, 12.00)	<0.001
Albumin (38–52 g/L)	37 ± 6.8	38.00 (32.00, 42.00)	37.75 (34.00, 41.00)	NS
CRP (<5 mg/L)	4 ± 14.5	0.71 (0.50, 1.20)	0.70 (0.50, 1.45)	NS
TSH (0.55–4.78 mUI/L)	1.9 ± 1.5	1.35 (0.90, 2.23)	1.62 (1.14, 2.35)	NS
Ca (2.12–2.52 mmol/L)	2.2 ± 0.17	2.25 (2.17, 2.38)	2.22 (2.14, 2.30)	NS
Ph (0.8–1.45 mmol/L)	1.2 ± 1.6	1.15 (0.94, 1.27)	1.15 (0.98, 1.31)	NS
AST (15–37 UI/L)	60.4 ± 116.3	28.00 (21.00, 38.00)	29.00 (21.00, 54.00)	NS
ALT (12–78 UI/L)	109 ± 232.8	36.00 (26.25, 65.75)	51.00 (30.00, 103.50)	0.001
GGT (5–55 UI/L)	63.1 ± 135.6	29.00 (18.00, 56.50)	34.00 (20.00, 59.00)	NS
ALP (46–116 UI/L)	90.2 ± 105.3	70.00 (52.75, 95.50)	70.00 (53.00, 94.00)	NS
BNP (<100 ng/L)	47.3 ± 68.4	20.00 (9.00, 46.00)	31.00 (15.00, 52.75)	0.021
LVEF (>50%)	64.87 ± 7708	65.00 (57.00, 67.00)	66.00 (61.00, 71.00)	0.009

Values are presented on median and Standard Deviation or on median and extremes; NS: not significant; BMI: body mass index; CRP: C reactive protein; TSH: thyroid balance; Ca: Calcium; Ph: Phosphoremia; AST: aspartate aminotransferase; ALT: alanine aminotransferase; GGT: gamma glutamyl transferase; ALP: alkaline phosphatase; BNP: brain natriuretic peptide; LVEF: left ventricular ejection fraction.

Paris, Frankreich

Article
Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Studie 1: Extreme AN – Welcher Mangel?



Rote Linie: Obergrenze Normbereich

Blaue Line: Untergrenze Normbereich

Figure 1. Micronutrients status in AN patients. Lower limits of normal micronutrients concentrations values are represented by blue lines and upper limits of normal values with redlines. Dots under the blue lines represent micronutrient deficiencies in AN patients.

Article
Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Studie 1: Extreme AN - Häufigkeiten

Nährstoff	Anteil Mangel	Normal bev.	Cutoff
Zink	64.3%	5%	<12.5 umol/l
Kupfer	37.1%	14%	<12.7 umol/l
Selen	20.5%	2%	<0.9 umol/l
Vitamin D	54.2%		<30 ng/ml
Vitamin B1	15%		
Vitamin B12	4.7%		<126 ng/l
Vitamin B9	8.9%		<3.9 ug/l

Paris, Frankreich

Article
Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Studie 1: Extreme AN – Kombinierte Häufigkeiten und Besonderheiten

Table 2. Deficiencies detected in AN patients.

Deficiencies Number	% of Patients
None deficiency	7.2%
One deficiency	28.3%
Two deficiency	33.2%
Three deficiency	18.8%
Four and more deficiency	12.6%

Table 3. Comparison of micronutrients status between AN-R and AN-BP.

Micronutrients	AN-BP	AN-R	*p*
Zinc (12.5–18 micromol/L)	11.10 (9.05, 13.20)	11.45 (9.50, 14.07)	NS
Copper (12.7–22.2 micromol/L)	15.25 (11.75, 17.70)	13.50 (11.30, 16.72)	0.022
Selenium (0.9–1.5 micromol/L)	1.02 (0.83, 1.21)	1.18 (1.00, 1.38)	<0.001
Vitamin B12 (197–77 1ng/L)	516.00 (337.00, 804.00)	608.50 (394.25, 976.75)	0.036
Vitamin B9 (>3.90 microg/L)	9.32 (6.18, 14.70)	10.66 (6.19, 17.55)	NS
Vitamin D (30–100 ng/mL)	26.00 (15.75, 35.25)	29.00 (20.75, 36.25)	NS
Vitamin B1 (126–250 nmol/L)	188.50 (150.00, 334.25)	195.00 (148.00, 457.00)	NS

Values are presented on median (interquartile range). AN-R, anorexia nervosa-restricting subtype; AN-BP, anorexia nervosa-binge-purging subtype; NS, not significant.

Paris, Frankreich

Article
Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Weitere Auffälligkeiten:

Clinical Research

Hypomagnesemia in Adolescents With Eating Disorders Hospitalized for Medical Instability

Kristin S. Raj MD, Casey Keane-Miller RD, Neville H. Golden MD

Background: Hypomagnesemia in patients with eating disorders is poorly characterized, particularly among adolescents. *Methods*: To determine the prevalence of hypomagnesemia (Mg ≤1.7 mg/dL) and clinical characteristics of adolescents hospitalized with a *DSM-IV*-diagnosed eating disorder who developed hypomagnesemia, a retrospective chart review was conducted on all adolescents aged 10–21 years with an eating disorder were hospitalized at a tertiary care children's hospital from 2007 to 2010. Patients were refed orally with standard nutrition and high-energy liquid supplements. Serum magnesium and phosphorus were obtained on admission, every 24–48 hours for the first week, and thereafter as clinically indicated. Clinical characteristics of patients with hypomagnesemia were compared with those of individuals with normal magnesium levels and those with hypophosphatemia. *Results*: Eighty-six of 541 eligible participants (15.9%) developed hypomagnesemia. Forty (47%) with hypomagnesemia admitted to purging in the year before admission, with 88% purging during the prior month. Compared with those with normal serum magnesium levels, patients with hypomagnesemia were older ($P = .0001$), ill longer ($P = .001$), more likely to be purging ($P = .04$), and more likely to have an alkaline urine ($P = .01$). They did not differ in eating disorder diagnosis, BMI, or other electrolyte disturbances. Hypomagnesemia developed 4.9 ± 5.5 days after refeeding was initiated, significantly later than the onset of hypophosphatemia, 0.95 ± 2.6 days ($P < .001$). *Conclusions*: Hypomagnesemia is prevalent in adolescents hospitalized for an eating disorder and is associated with purging and alkaline urine. Hypomagnesemia develops later in the course of refeeding than hypophosphatemia. Magnesium levels should continue to be monitored after the more immediate risk of hypophosphatemia has passed, especially in those with alkaline urine.

Article

Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Was noch?

Significant nutritional variables in patients with eating disorders

Leanne Barron[1,2*], Robert Barron[3], Jeremy Johnson[4], Shannon Ward[5], Ingrid Wagner[6], Warren Ward[7]

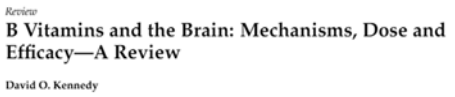
Retrospective chart analysis of 113 patients presenting to a general practitioner with eating disorders was conducted, in an attempt to identify statistically and clinically significant nutritional variables. Blood tests are a useful diagnostic tool in eating disorders, and this research suggests that current testing should be broadened to include trace minerals such as zinc and manganese.

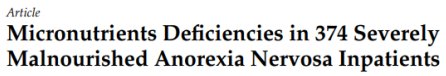
Results were analysed for cholesterol, red blood cell folate, vitamin B12, magnesium, manganese, zinc, vitamin D, phosphate, ferritin, white cell count, red cell count and platelets. Patients were analysed as an entire group, but also separately as those suffering from Anorexia Nervosa (1), Bulimia Nervosa (2), EDNOS (3) and classic AN followed by BN (4).

Analysis using T tests and chi squared showed that variables most likely to lie outside the population reference range were manganese, cholesterol, ferritin, vitamin B12, zinc and vitamin D.

Konsequenzen (1)

- **Zink:**
  - Wichtige Rolle in der Neurotransmission im GABAergen System
  - Hinweise auf bessere Gewichtszunahme nach Gabe von Zink
  - ➢ Supplementation sinnvoll
- **Selen:**
  - Wichtig als Regulator von Angst und Stimmung
  - Wichtig als Antioxidans und zur Prävention von kardialen Komplikationen bei Hypokaliämie und Hypomagnesiämie
  - ✓ Supplementation sinnvoll
- **Thiamin:**
  - Wichtig für emotionale Stabilität, Schlaf, Gedächtnis
  - ✓ Supplementation sinnvoll
- **Magnesium:**
  - Wichtig für den Elektrolythaushalt
  - ✓ Supplementation sinnvoll


Review
B Vitamins and the Brain: Mechanisms, Dose and Efficacy—A Review
David O. Kennedy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Micronutrients Deficiencies in 374 Severely Malnourished Anorexia Nervosa Inpatients

Konsequenzen (2)

- **Kupfer:**
  - Rolle bei der Erkrankung unklar
  - ➢ Keine klare Indikation für Supplementation
- **Vitamin B9 (Folsäure) und B12:**
  - Eher seltenere Mängel
  - Kognitive Störungen, Affektive Störungen, Verhaltensstörungen, Psychotische Störungen
  - Im Labor kostengünstig zu ermitteln (Holo-TC, Folsäure i.d. Erythrozyten)
  - ➢ Supplementation bei Mangel
- **Vitamin D:**
  - Osteoporose, Osteomalazie, Schlafstörungen, Schmerzen, depressive Symptomatik
  - Im Labor messbar
  - ➢ Supplementation bei Mangel

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Exkurs: Refeeding Syndrom

Studie 2: Leichtere Verläufe

Mikronährstoffe bei moderater AN

Article

Micronutrient Status in 153 Patients with Anorexia Nervosa

Najate Achamrah [1,2,3,4,*], Moïse Coëffier [1,2,3], Agnès Rimbert [1,2,3], Jocelyne Charles [1], Vanessa Folope [1,2,3], André Petit [1,2,3], Pierre Déchelotte [1,2,3] and Sébastien Grigioni [1,2,3]

Rouen, Frankreich

Studie 2: Moderate AN – die Daten

Table 1. Comparison of AN-R and AN-BP anthropometric features.

	AN-R (n = 91)	AN-BP (n = 62)	t-Test (p)
Mean age (years)	29.42 ± 11.4	27.1 ± 10.4	NS
Disease duration (years)	6.8 ± 7.6	8.4 ± 9.3	NS
BMI (kg/m^2)	16.5 ± 2	18.8 ± 3.0	<0.05
FM (%)	18.7 ± 6.5	20.8 ± 5.9	p = 0.054
FFM (%)	80.6 ± 8.9	76.9 ± 12.6	<0.05

AN-R, anorexia nervosa—restricting subtype; AN-BP, anorexia nervosa—binge–purging subtype; BMI, body mass index; FM, fat mass; FFM, fat-free mass; NS, not significant. Values are means ± SD.

Table 2. Micronutrients mean values in AN-R and AN-BP patients.

	Mean Values	AN-R	AN-BP	t-Test (p)
Zn (9–17 µmol/L)	12.8 ± 1.9	13.04 ± 2.03	12.6 ± 1.7	NS
Cu (10–40 µmol/)	18.76 ± 5.8	18.40 ± 5.9	19.36 ± 5.5	NS
Ph (0.87–1.50 mmol/L)	1.19 ± 0.2	1.20 ± 0.17	1.17 ± 0.23	NS
Mg (0.75–1 mmol/L)	0.86 ± 0.64	0.87 ± 0.60	0.85 ± 0.72	NS
Ca^{2+} (2.15–2.55 mmol/L)	2.36 ± 0.92	2.35 ± 0.91	2.37 ± 0.09	NS
Se (0.90–1.65 µmol/L)	0.97 ± 0.21	0.99 ± 0.23	0.93 ± 0.18	NS
Vit E (7–17 mg/L)	11.89 ± 3.51	11.4 ± 3.07	12.68 ± 4.02	<0.05
B12 (141–489 pmol/L)	413.1 ± 193.1	426.3 ± 183.0	391.46 ± 177.7	NS
B9 (10.4–42.4 nmol/L)	19.42 ± 13.07	19.45 ± 10.59	19.38 ± 16.40	NS
Vit A (430–800 µg/L)	521.6 ± 164.6	511.6 ± 152.4	537.5 ± 182.7	NS

AN-R, anorexia nervosa—restricting subtype; AN-BP, anorexia nervosa—binge–purging subtype; NS, not significant. Values are means ± SD.

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Micronutrient Status in 153 Patients with Anorexia Nervosa

Studie 2: Moderate AN – Mikronährstoffe

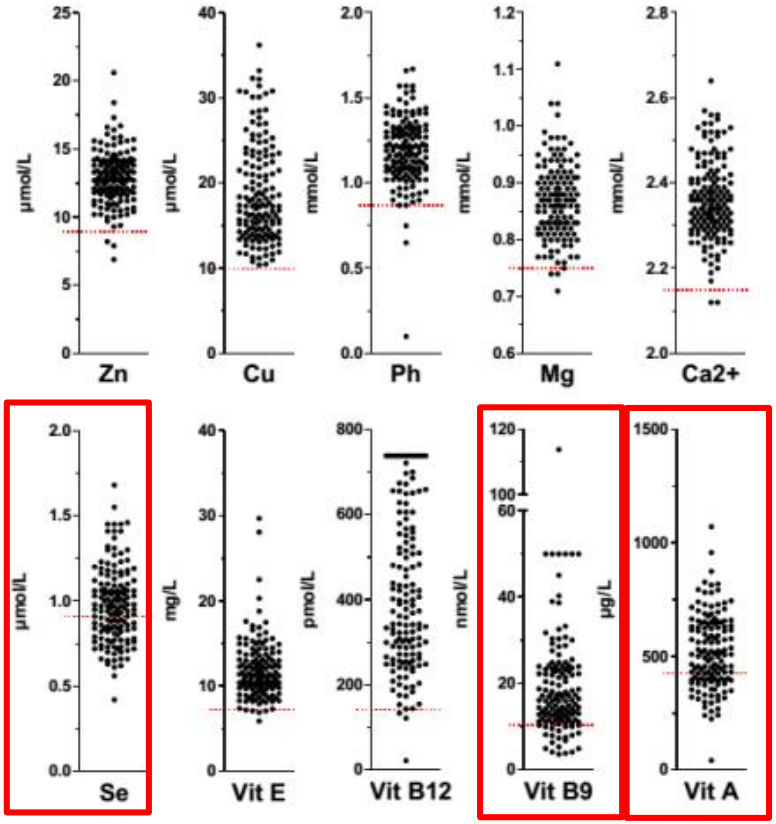

Figure 1. Micronutrients status in AN patients. Zinc (Zn), copper (Cu), phosphorus (Ph), magnesium (Mg), calcium (Ca2+), selenium (Se), vitamin E (Vit E), vitamin B12 (Vit B12), vitamin B9 (Vit B9), and vitamin A (Vit A) values represented by plots are expressed in µmol/L (Zn, Cu, and Se), mmol/L (Ph, Mg, and Ca2+), mg/L (Vit E), pmol/L (Vit B12), nmol/L (Vit B9) or µg/L (Vit A). Low micronutrients concentrations are represented by red lines. Plots under the red lines represent micronutrient deficiencies in AN patients.

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Micronutrient Status in 153 Patients with Anorexia Nervosa

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Studie 2: Moderate AN - Besonderheiten

Table 3. Patients with micronutrient deficiency in AN-R and AN-BP.

	Deficiency % (*n*)	AN-R % (*n*)	AN-BP % (*n*)	*p* (Chi2)
Zn (9–17 µmol/L)	2.1 (3)	2.2 (2)	1.9 (1)	NS
Cu (10–40 µmol/L)	0 (0)	0 (0)	0 (0)	-
Ph (0.87–1.50 mmol/L)	3.5 (5)	3.5 (3)	3.6 (2)	NS
Mg (0.75–1 mmol/L)	3.0 (4)	1.2 (1)	6.0 (3)	NS
Ca^{2+} (2.15–2.55 mmol/L)	1.3 (2)	1.1 (1)	1.7 (1)	NS
Se (0.90–1.65 µmol/L)	40.6 (58)	40.0 (36)	41.5 (22)	NS
Vit E (7–17 mg/L)	1.5 (2)	1.2 (1)	1.9 (1)	NS
B12 (141–489 pmol/L)	2.2 (3)	2.4 (2)	1.9 (1)	NS
B9 (10.4–42.4 nmol/L)	15.9 (22)	14.1 (12)	18.9 (10)	NS
Vit A (430–800 µg/L)	32.8 (45)	35.7 (30)	28.3 (15)	NS
At least one oligoelement deficiency	43.7 (55)	43.0 (34)	44.7 (21)	NS
At least one vitamin deficiency	45.7 (59)	46.9 (38)	43.8 (21)	NS

AN-R, anorexia nervosa—restricting subtype; AN-BP, anorexia nervosa—binge–purging subtype; NS, not significant.

Article
Micronutrient Status in 153 Patients with Anorexia Nervosa

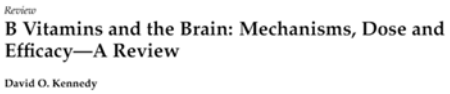
Moderate AN: Konsequenzen (1)

- **Zink:**
  - Seltener Mangel
  - ➢ Supplementation nur bei Mangel
- **Selen:**
  - Sehr häufiger Mangel
  - Wichtig als Regulator von Angst und Stimmung
  - Wichtig als Antioxidans
  - Wichtig für Kardiale Komplikationen, welche aufgrund von Hypokaliämie und Hypomagnesiämie relevant sind
  - ✓ Supplementation sinnvoll
- **Vitamin A:**
  - Häufiger Mangel
  - ✓ Supplementation sinnvoll

Article
Micronutrient Status in 153 Patients with Anorexia Nervosa

Moderate AN: Konsequenzen (2)

- **Kupfer:**
  - Sehr seltener Mangel
  - ➢ Keine Indikation für Supplementation
- **Vitamin B9 (Folsäure) und B12:**
  - Häufiger Mängel
  - Kognitive Störungen, Affektive Störungen, Verhaltensstörungen, Psychotische Störungen
  - Im Labor kostengünstig zu ermitteln (Holo-TC, Folsäure i.d. Erythrozyten)
  - ➢ Supplementation bei Mangel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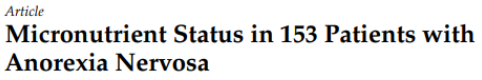
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Fragen und Diskussion

Binge Eating und Adipositas

plants

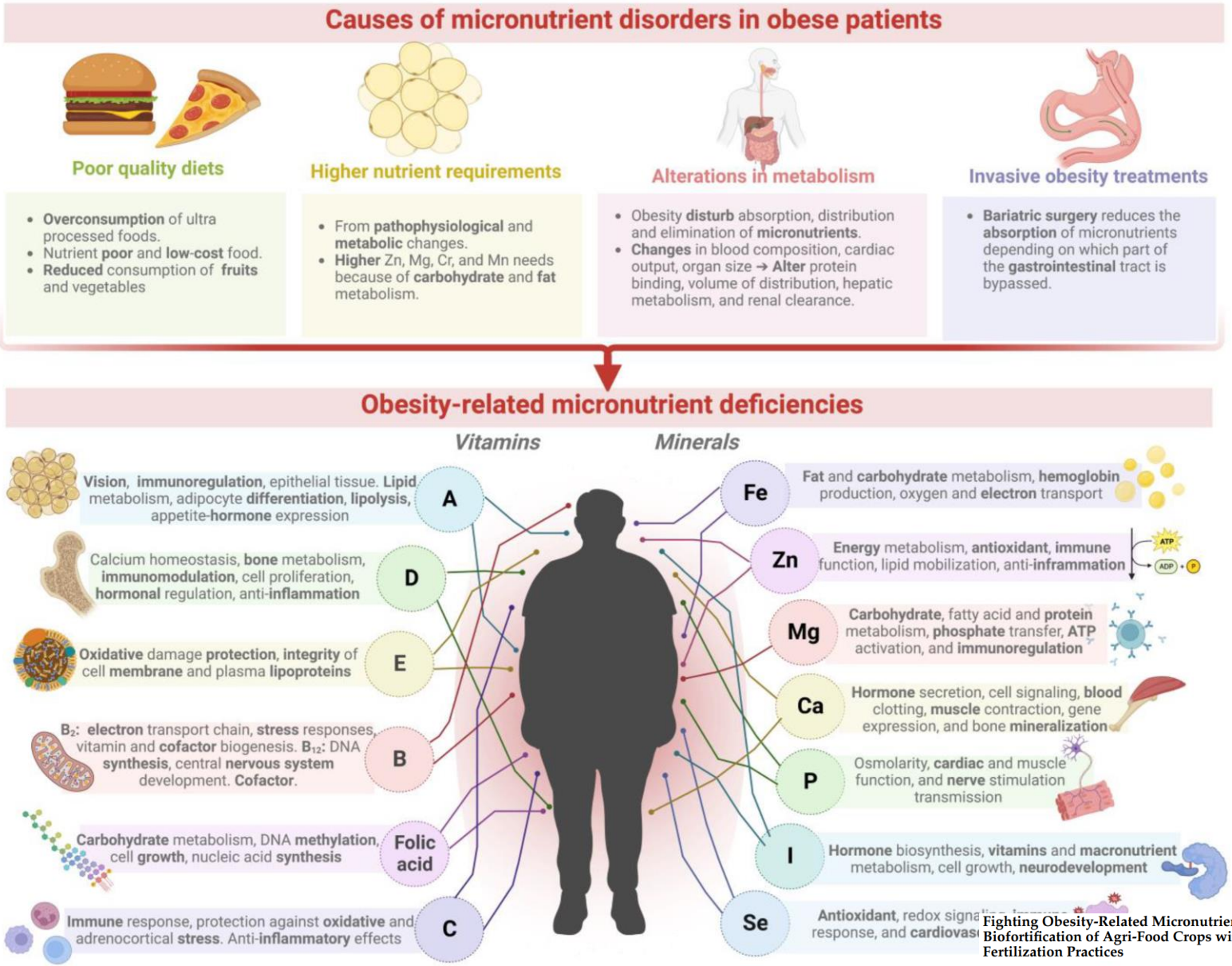
MDPI

Review

Fighting Obesity-Related Micronutrient Deficiencies through Biofortification of Agri-Food Crops with Sustainable Fertilization Practices

Carlos Esteban Guardiola-Márquez [1], María Teresa Santos-Ramírez [1], M. Eugenia Segura-Jiménez [1], Melina Lizeth Figueroa-Montes [1] and Daniel A. Jacobo-Velázquez [1,2,*]

Fighting Obesity-Related Micronutrient Deficiencies through Biofortification of Agri-Food Crops with Sustainable Fertilization Practices



Fighting Obesity-Related Micronutrient Deficiencies through Biofortification of Agri-Food Crops with Sustainable Fertilization Practices

Mikronährstoffe und Adipositas

Micronutrient	Micronutrient Physiologic and Metabolic Function	Deficiency in Obese Patients	Type of Condition	Reference
Vitamin A and carotenoids	Retina and epithelial tissue development, lipid metabolism, immune system function. Inhibition of adipocyte differentiation by enhancing lipolysis. Reduction in leptin and resistin expression [14,47].	Carotenoids (α-carotene, β-carotene, ζ-carotene, lutein, and lycopene) ≈ 44.4%.	Male (n = 29) and female (n = 37) individuals between 49 and 58 years old with a body mass index (BMI) > 30 kg/m^2.	[48]
		All evaluated patients presented a deficiency of vitamin A (<30 µg/dL).	Individuals with a BMI over 25 kg/m^2 (overweight) and 30 kg/m^2 (obesity) aged 18–65 years (n = 127).	[6]
Vitamin D	Calcium homeostasis, bone metabolism, immunomodulation, cell proliferation, and control of hormonal systems. Upregulates anti-inflammatory cytokines [49].	Approximately 16.5% presented a deficiency of serum 25 hydroxy vitamin D (<30 nmol/L).	Danish individuals; 6–18 years old (n = 1484) with overweight/obesity; body mass index standard deviation score (BMI Z-score) > 2.33.	[50]
		The prevalence of deficiency (≤20 ng/mL) is around 90%.	Obese individuals class II and III (BMI ≥ 35 and ≥40 kg/m^2).	[2]
Vitamin E	Protection of cell constituents from oxidative damage, such as polyunsaturated fatty acids found in the membrane and plasma lipoproteins [51].	Deficiency of 61.5% (11.5 ± 12.2 mg/L), and 47.8% (15.6 ± 12.2 mg/L) in obese and metabolic syndrome patients, respectively.	Individuals 10–16 years old from Central Turkey with obesity (BMI Z-score > 2) (n = 73) or metabolic syndrome (waist circumference ≥ 90 cm (n = 64).	[52]
Vitamin B_2	Mitochondrial electron transport chain function and homocysteine metabolism. Its derivatives, flavin mononucleotide and flavin adenine dinucleotide, are implicated in stress responses and vitamin and cofactor biogenesis [53].	Deficit of 48.9% in the obese group (89.1 ± 35 µg/L); 33.1% in the metabolic syndrome group (116.7 ± 65.2 µg/L).	Individuals 10–16 years old from Central Turkey with obesity (BMI Z-score > 2) (n = 73) or metabolic syndrome (waist circumference ≥ 90 cm (n = 64).	[52]
		Deficiency of 38.8% (<5 ng/mL).	Children 11–17 years old (n = 50) with obesity (BMI Z-score ≥ 2).	[54]
Vitamin B_{12}	DNA synthesis, conversion of homocysteine to methionine, and central nervous system development. Cofactor in the one-carbon metabolism and propionate catabolism [55,56].	Insufficiency of 23% (< 150 pmol/l) in cohort 1 and 18.3% in cohort 2.	Two cohorts of pregnant women (16–18 weeks) (n = 244 and n = 60) with average BMI = 26.5 ± 5.5 kg/m^2 for cohort 1 and BMI = 32.6 ± 11.2 kg/m^2 for cohort 2.	[57]
		Deficiency of around 29% (397.5 ± 26.3 ng/L).	Forty obese adults (BMI > 35 kg/m^2) aged 21–49 underwent bariatric surgery.	[58]
Folic acid	Well-functioning carbohydrate metabolism (15). DNA methylation, cell growth, and nucleic acid synthesis [56].	Prevalence of 54% (obese) and 65% (patients after bariatric surgery).	Patients with morbid obesity before (BMI > 30 kg/m^2) and after bariatric surgery (BMI > 35 kg/m^2).	[56]
		Inadequacies (<10 nmol/L) per area: America (0.8–2.1%), Europe and Eastern Mediterranean (40.9%), Africa (24.4%), Southeast Asia, and Western Pacific (1.1–3.7%).	Women with a rising prevalence of overweight and/or obesity (BMI > 18.5 kg/m^2) in reproductive age (15–49 years old) in 17 population surveys.	[59]

Fighting Obesity-Related Micronutrient Deficiencies through Biofortification of Agri-Food Crops with Sustainable Fertilization Practices

Vitamin C	Immune response, protection against oxidative and adrenocortical stress. Anti-inflammatory effects [60].	Deficit of 24.6%, 32.8%, and 34.6% for sarcopenic, osteopenic, and osteosarcopenic obese individuals.	Korean women (n = 1344) postmenopausal (>50 years old) with osteosarcopenic (BMI = 27.15 kg/m^2), sarcopenic (BMI = 28.12 kg/m^2), and osteopenic (BMI = 26.24 kg/m^2) obesity.	[61]
Iron	Fat and carbohydrate metabolism, hemoglobin production, oxygen transport, DNA synthesis, and electron transport [14,62].	Deficiency of 31.8% in male and 25.9% in female patients.	Children 8–9 years old (n = 160) with high body fat (BMI Z-score > 1) in Sri Lanka.	[63]
		Insufficiency in patients with peripheral (16.9%) and central (10.7%) adiposity.	Overweight and/or obese American young women (23–43 years old; BMI ≥ 25 kg/m^2; n = 81).	[64]
Zinc	Energy metabolism with antioxidant and immunological properties. Stimulates the function of zinc-α2-glycoprotein (adipokine with lipid mobilizing and anti-inflammatory activity) [65].	Prevalence of 24–74% after bypass surgery: biliopancreatic bypass (45–91%), gastric bypass (15–21%), laparoscopic sleeve gastrectomy (11–14%).	Patients with morbid obesity before (BMI > 30 kg/m^2) and after bariatric surgery (BMI > 35 kg/m^2).	[56]
		Deficiency prevalence of 84.7% (<70 µg/dL fasted).	Women rising prevalence of overweight and/or obesity (BMI > 18.5 kg/m^2) in reproductive age (15–49 years old).	[59]
Magnesium	Carbohydrate metabolism, phosphate transfer reactions, fatty acid and protein synthesis, ATP activation, and immune system function [62,66].	Deficiency in males was 6.6%, and, in females was 7.7%.	Children 8–9 years old (n = 160) with high body fat (BMI Z-score > 1) in Sri Lanka.	[63]
Calcium	Hormone secretion, intracellular signaling, blood clotting, muscle contraction, gene expression, and bone mineralization [67,68].	Deficiency of 50.2% in obese women.	Obese women (35.37 ± 2.09 years old) with average BMI = 34.68 ± 0.61 kg/m^2 (n = 70).	[69]
Potassium	Cellular osmolarity, acid–base equilibrium, cardiac and muscle function, and nerve stimulation transmission [70].	Deficiency of 59.6% in obese women.	Obese women (35.37 ± 2.09 years old) average BMI= 34.68 ± 0.61 kg/m^2 (n = 70)	[69]
		100% of patients showed deficiency (<3.5 mmol/L).	Individuals with a BMI over 25 kg/m^2 (overweight) and 30 kg/m^2 (obesity) aged 18–65 years (n = 127).	[6]
Iodine	Thyroid hormones biosynthesis, vitamins, macronutrient metabolism, and cell growth fetal and child neurodevelopment [71,72].	Insufficiency prevalence of 24.4%.	Overweight (BMI > 25 kg/m^2) and obese (BMI > 30 kg/m^2) children (11–13 years old) residing in iodine-sufficient areas (IS) and mildly iodine-deficient areas (ID).	[73]
Selenium	Antioxidant defense, redox signaling, immune response, and cardiovascular function [74].	Deficiency of 25.9% in plasma and 34.2% in the erythrocyte.	Obese women aged 20–50 years (BMI ≥ 35 kg/m^2, n = 63).	[75]
Copper	Electron transport, protein structure, mitochondrial respiratory chain, immune function, antioxidant defense. Cofactor of redox enzymes [56,76].	Concentration decreased by 16% 12 months after bariatric surgery.	Norwegian patients (85% women) 27–59 years old, eligible for bariatric surgery (BMI = 42.4 ± 3.6 kg/m^2, n = 46).	[77]
		Prevalence of 46.7%.	Overweight/obese children aged 6–16 years (average BMI = 24.78 ± 3.93 kg/m^2, n = 69).	[78]

Fighting Obesity-Related Micronutrient Deficiencies through Biofortification of Agri-Food Crops with Sustainable Fertilization Practices

Mikronährstoffmangel bei Adipositas

International Scholarly Research Network
ISRN Endocrinology
Volume 2012, Article ID 103472, 8 pages
doi:10.5402/2012/103472

Review Article

The Malnutrition of Obesity: Micronutrient Deficiencies That Promote Diabetes

Mikronährstoffmangel bei Adipositas

Table 1: Prevalence of micronutrient deficiencies in obesity and diabetes [5, 6, 46, 58–61].

Micronutrient	Prevalence of deficiency	
	Obesity	Type 2 diabetes
Thiamine B1	15–29%	17–79%[a]
Pyridoxine B6	0–11%	—
Cobalamin B12	3–8%	22%
Folic Acid	3-4%	—
Ascorbic acid C	35–45%	—[b]
Vitamin A	17%	—
Vitamin D	80–90%[c]	85–90%[c]
Vitamin E	0%	0%
Zinc	14–30%	—
Chromium	—	20–40%
Selenium	58%	—

—Prevalence data not available
[a]Data includes type 1 diabetic patients
[b]Decreased levels of ascorbic acid have been reported in diabetes
[c]Prevalence reflects rates vitamin D insufficiency.

Thiamine deficiency measured by direct plasma levels of thiamine or by elevated erythrocyte transketolase activity has been observed in 15–29% of obese individuals planning to undergo weight loss surgery [75, 76]. The prevalence of thiamine deficiency has been reported in 17–79% of diabetic patients, although these studies include both type 1 and type 2 diabetic patients [77–79] (Table 1).

High rates of vitamin D insufficiency and frank deficiency have been reported in obese individuals and in diabetics. The prevalence of vitamin D insufficiency (defined as <30 mg/dL) in obese individuals ranges from 80–90% [5, 9]. While some controversy exists over treatment targets in individuals with mild insufficiency of vitamin D especially for the purported extraskeletal effects of vitamin D supplementation, [10, 11] a significant amount of evidence suggests there may be some beneficial effect in using vitamin D supplementation for improvement in glucose metabolism and insulin signaling in patients with type 2 diabetes or impaired glucose tolerance [12].

Further studies in diabetic patients and in those at risk for developing diabetes may help to elucidate the role of vitamin C supplementation in this population. The high rate of vitamin C deficiency in obese individuals suggests that supplementation may be beneficial. Increasing dietary intake of fruits and vegetables can also address this deficiency and is currently recommended as part of a lifestyle intervention for the prevention and treatment of type 2 diabetes [101].

The Malnutrition of Obesity: Micronutrient Deficiencies That Promote Diabetes

Aber:

Nutritional status of selenium in overweight and obesity: A systematic review and meta-analysis

Larissa Cristina Fontenelle [1], Diana Stefany Cardoso de Araújo [2], Tamires da Cunha Soares [3], Kyria Jayanne Clímaco Cruz [4], Gilberto Simeone Henriques [5], Dilina do Nascimento Marreiro [6]

Results: This systematic review included 73 articles, of which 65 articles were eligible for meta-analysis. There were no significant differences between the overweight/obesity and eutrophy groups in terms of dietary intake and plasma/serum levels of selenium and selenoprotein P levels. The activity of plasma/serum glutathione peroxidase was decreased in the overweight/obese children and adolescents; however, the difference was no statistically significant when the enzyme activity was analyzed in erythrocytes and whole blood. In the adult age group, overweight/obese individuals had decreased activity of both glutathione peroxidase isoforms compared to those individuals with eutrophy. In particular, the activity of erythrocyte glutathione peroxidase was significantly decreased in obese individuals and those living in regions with low-to-moderate selenium content in the soil. Regarding selenium excretion markers, overweight/obese individuals had lower levels of selenium in the urine and nails and a trend of higher levels of selenium in the hair than eutrophic individuals.

Conclusions: In conditions of excess adiposity, the main alteration in the nutritional status of selenium is a decrease in glutathione peroxidase activity, particularly in adults with obesity. In addition, reduced levels of selenium in urine and nails can be found in overweight or obese individuals.

Zusammenfassung Mangel:

Anorexie	Adipositas
Vitamin D	Vitamin D
Zink	Zink
Selen	Selen
Folsäure	Folsäure
Vitamin B1	Vitamin B1
Vitamin A	
Magnesium (?)	

Fragen und Diskussion

Fokus auf Selen:

ELSEVIER

Psychoneuroendocrinology

Volume 140, June 2022, 105723

Selenium deficiency is associated with disease severity, disrupted reward processing, and increased suicide risk in patients with Anorexia Nervosa

Robertas Strumila [a b c 1], Aiste Lengvenyte [a b c 1], Emilie Olie [a b], Maude Seneque [a b], Kathlyne Dupuis-Maurin [a b], Adrian Alacreu-Crespo [a b f], Laurent Maimoun [g h], Patrick Lefebvre [d], Eric Renard [a d e], Philippe Courtet [a b], Sebastien Guillaume [a b]

Montpellier, Frankreich

Fokus auf Selen:

	Low selenium Mean ± SD or N(%)	Normal Selenium Mean ± SD or N(%)	T-statistic, U score, or χ^2	P-value
Sociodemographic and clinical characteristics				
Age, years	25.48 ± 9.20	26.76 ± 11.17	2777.0	0.623
Age at AN onset, years	**16.24 ± 2.93**	**19.04 ± 6.25**	**-2.96[t]**	**0.005***
Disease duration, years	8.42 ± 8.78	8.58 ± 9.58	2650.5	0.77
Education, years	14.19 ± 9.98	13.63 ± 2.31	2137	0.18
BMI	18.12 ± 3.07	17.75 ± 2.40	0.83[t]	0.41
BMI, minimal lifetime	15.31 ± 2.17	15.9 ± 2.09	-1.66[t]	0.10
Calorimetry	1183.60 ± 220.54	1120.67 ± 197.4	1.79[t]	0.07
Fat mass	21.99 ± 7.99	20.16 ± 6.94	1.43[t]	0.15
Vitamin B12	**308.31 ± 122.87**	**378.18 ± 162.73**	**-2.94[t]**	**0.004****
Irregular menstruation	17/41 (41.46%)	7/35 (20.00%)	4.03	0.045*
Suicide attempt history	**23/81 (28.39 %)**	**7/71 (9.85 %)**	**8.2[x2]**	**0.004****
Violent suicide attempt history	6/22 (27.27 %)	0/7 (0 %)	2.4 [x2]	0.121
Suicide risk (MINI)	25/75 (33%)	10/58 (17.24%)	4.37	0.037*
Hospitalization history	19/82 (23.17%)	7/71 (9.8%)	4.78	0.029*

Montpellier, Frankreich

Selenium deficiency is associated with disease severity, disrupted reward processing, and increased suicide risk in patients with Anorexia Nervosa

Fokus auf Selen:

	Low selenium Mean ± SD or N(%)	Normal Selenium Mean ± SD or N(%)	T-statistic, U score, or χ^2	P-value
Clinical questionnaires scores				
EDEQL score	1.58 ± .803	1.32 ± .729	1.72[t]	0.089
SATAQ-4 score	62.65 ± 15.96	58.84 ± 15.61	1.40[t]	0.165
BSQ-34 score	**122.09 ± 44.22**	**99.17 ± 40.40**	**1477.5[u]**	**0.004****
EDE-Q score	**3.63 ± 1.34**	**2.82 ± 1.73**	**1.596[u]**	**0.010****
Neuropsychological characteristics				
Delayed discounting score (log)	**0.98 ± 3.39**	**0.03 ± 0.07**	**1163.5[u]**	**0.006****
Probabilistic discounting task score (log)	1.07 ± 2.09	0.84 ± 1.46	1568.5[u]	0.636
IGT score	5.13 ± 30.42	2.69 ± 33.01	0.394[t]	0.694
BSAT score	7.53 ± 2.047	7.41 ± 1.90	1987[u]	0.551
D2 test of attention score	0.4 ± .31	0.4 ± 0.25	1728.0[u]	0.418
ROCF Copy	1.97 ± 0.88	1.87 ± 0.85	1928.5[u]	0.546

Abbreviation: AN, anorexia nervosa; BSAT, Brixton spatial anticipation test; BSQ-34, body shape questionnaire 34; EDE-Q, eating disorder examination questionnaire; EDEQL, eating disorder quality of life questionnaire; IGT, Iowa gambling task; ROCF, Rey-Osterrieth complex figure; SATAQ-4, sociocultural attitudes towards appearance questionnaire 4; BMI – body mass index; SD, standard difference, MINI, Mini international neuropsychiatric interview.

* Significant after Benjamini-Hochberg correction, adjusted p-value (family wise error rate), with critical p value <0.01
** significant after standard unadjusted p value <0.05

Selenium deficiency is associated with disease severity, disrupted reward processing, and increased suicide risk in patients with Anorexia Nervosa

Fokus auf Selen:

Results

Se plasma levels were below the cut-off of 80µg/L in 53.6% (N=82) of patients. AN onset was earlier in patients with Se deficiency, (p=.005), whereas disease duration was comparable between groups (p=.77). General eating disorder symptomatology in the past 28 days (Eating Disorder Examination Questionnaire) was more severe in patients with Se deficiency (p=.010). The suicide risk (MINI International Neuropsychiatric Evaluation) tended to be higher (p=.037), and suicide attempt history was more frequent (28.39% vs 9.85%, p=.004) in patients with low Se levels. Se plasma concentration was negatively correlated with the performance in the temporal delayed discounting task (p=.006).

Conclusions

Our findings suggest that in patients with AN, Se plasma concentration might be implicated in disease severity and suicide risk. The finding that Se deficiency in patients with AN was associated only with reward-related processes, but not with other psychological functions suggests the selective involvement of dopamine-related pathways. Our results suggest that it might be useful to monitor the plasma micronutrient profile in patients with AN. Future studies should determine whether Se supplementation in AN might improve clinical outcomes.

Selenium deficiency is associated with disease severity, disrupted reward processing, and increased suicide risk in patients with Anorexia Nervosa

Fokus auf Selen

The role of selenium in depression: a systematic review and meta-analysis of human observational and interventional studies

Sana Sadat Sajjadi[1], Sahar Foshati[2], Sajjad Haddadian-Khouzani[1] & Mohammad Hossein Rouhani[1]✉

The results of human studies are inconsistent regarding selenium and depressive disorders. Therefore, we aimed to conduct a systematic review and meta-analysis of observational and interventional studies and provided an overview of the role of selenium in depression. Three databases including Medline, Scopus, and Web of Science were searched on June 30, 2020 and updated on April 12, 2021. Also, we searched in electronical databases of WHO Global Index Medicus and ClinicalTrials.gov. No time or language restrictions were used for the search. A random effects model was used to pool effect sizes. In total, 20 studies were included in the systematic review, and 15 studies were included in the meta-analysis. There were no significant differences in serum selenium levels between patients with depression and healthy subjects (WMD: 2.12 mg/L; 95% CI: − 0.11, 4.36; $I^2 = 98.0\%$, $P < 0.001$). Also, no significant correlation was found between serum levels of selenium and depression scores (r: − 0.12; 95% CI: − 0.33, 0.08; $I^2 = 73.5\%$, $P = 0.010$). Nevertheless, there was a significant negative association between high selenium intake and the risk of postpartum depression (OR: 0.97; 95% CI: 0.95, 0.99; $I^2 = 0.0\%$, $P = 0.507$). In addition, selenium supplementation significantly reduced depressive symptoms (WMD: − 0.37; 95% CI: − 0.56, − 0.18; $I^2 = 0.0\%$, $P = 0.959$). Taken these results together, selenium seems to have a protective role against postpartum depression and can be considered as a beneficial adjuvant therapy in patients with depression. Further studies are necessary to draw definitive conclusions.

Scientific Reports | (2022) 12:1045 | https://doi.org/10.1038/s41598-022-05078-1 nature portfolio

Fokus auf Selen

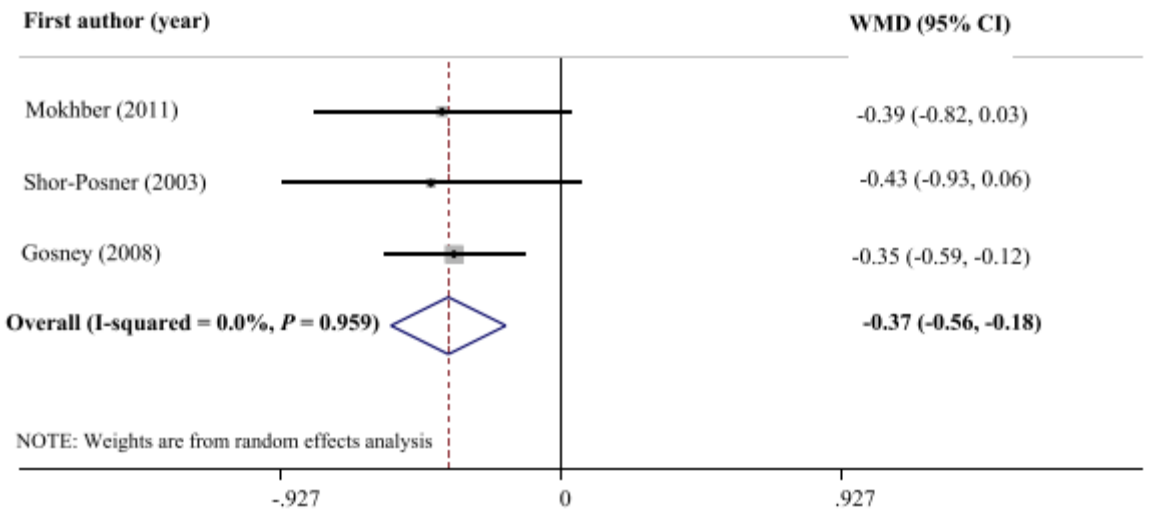


Figure 5. Forest plot of the effect of selenium supplementation on depression scores.

Scientific Reports | (2022) 12:1045 | https://doi.org/10.1038/s41598-022-05078-1 nature portfolio

Privat Klinik Aadorf

Fokus auf Zink:

frontiers in
BEHAVIORAL NEUROSCIENCE

REVIEW ARTICLE
published: 06 January 2015
doi: 10.3389/fnbeh.2014.00443

Behavioral impairments in animal models for zinc deficiency

Simone Hagmeyer[1†], Jasmin Carmen Haderspeck[1†] and Andreas Martin Grabrucker[1,2*]

[1] WG Molecular Analysis of Synaptopathies, Neurology Department, Neurocenter of Ulm University, Ulm, Germany
[2] Institute for Anatomy and Cell Biology, Ulm University, Ulm, Germany

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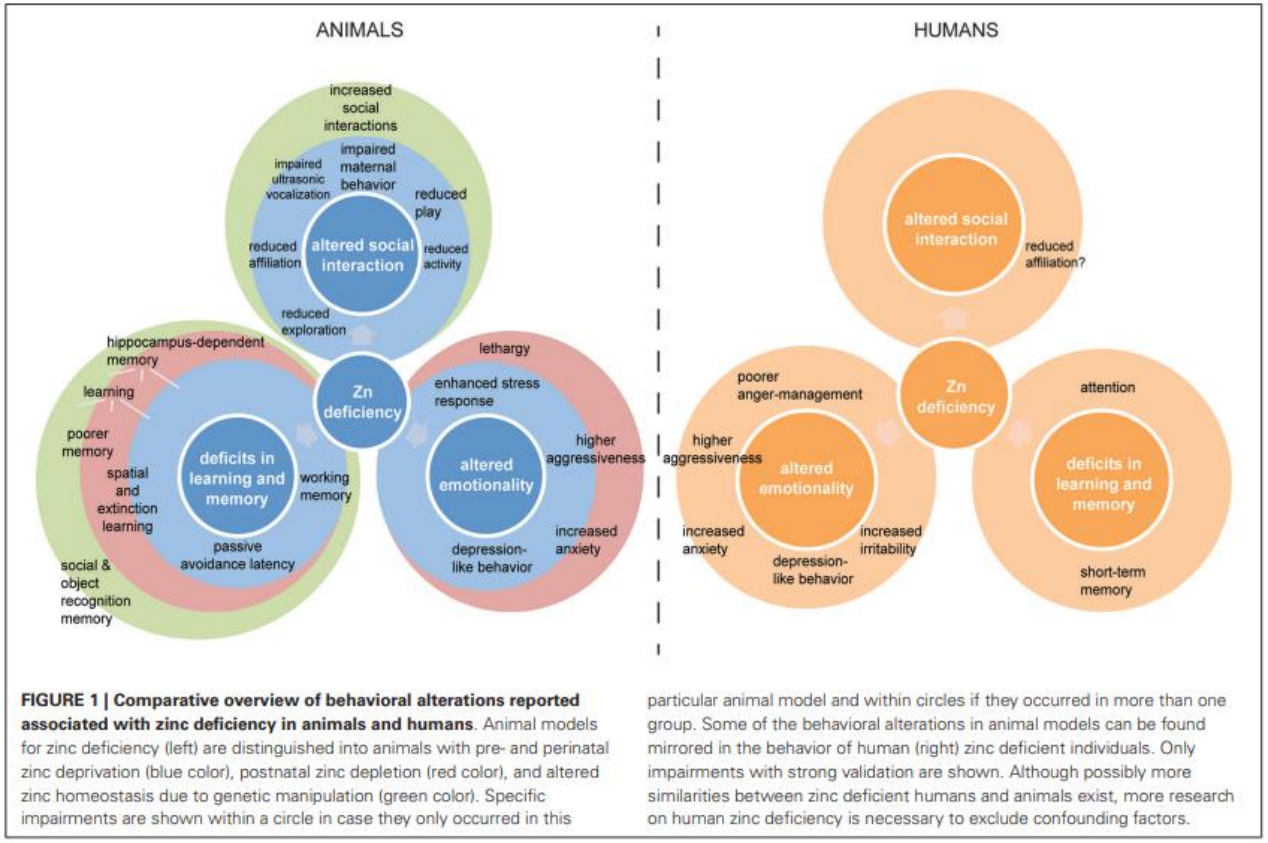

FIGURE 1 | Comparative overview of behavioral alterations reported associated with zinc deficiency in animals and humans. Animal models for zinc deficiency (left) are distinguished into animals with pre- and perinatal zinc deprivation (blue color), postnatal zinc depletion (red color), and altered zinc homeostasis due to genetic manipulation (green color). Specific impairments are shown within a circle in case they only occurred in this particular animal model and within circles if they occurred in more than one group. Some of the behavioral alterations in animal models can be found mirrored in the behavior of human (right) zinc deficient individuals. Only impairments with strong validation are shown. Although possibly more similarities between zinc deficient humans and animals exist, more research on human zinc deficiency is necessary to exclude confounding factors.

frontiers in BEHAVIORAL NEUROSCIENCE
REVIEW ARTICLE
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Behavioral impairments in animal models for zinc deficiency

*Simone Hagmeyer[1†], Jasmin Carmen Haderspeck[1†] and Andreas Martin Grabrucker[1,2]**

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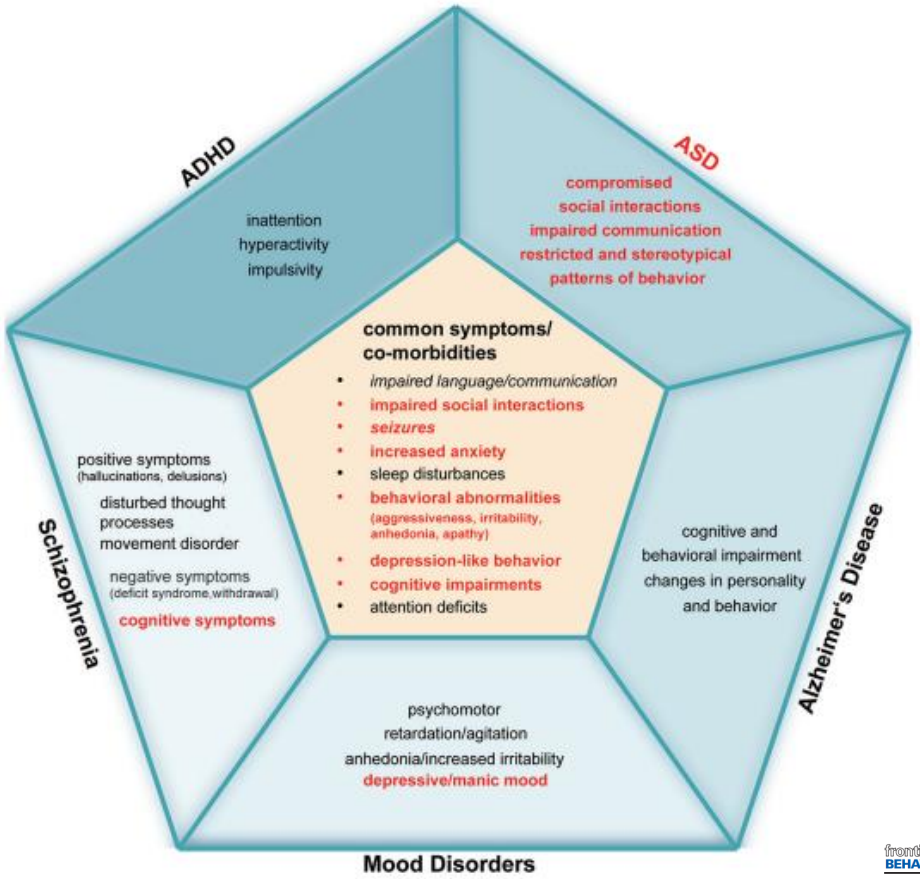

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Behavioral impairments in animal models for zinc deficiency

*Simone Hagmeyer[1†], Jasmin Carmen Haderspeck[1†] and Andreas Martin Grabrucker[1,2]**

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Fokus auf Zink:

Zinc Supplementation and Body Weight: A Systematic Review and Dose–Response Meta-analysis of Randomized Controlled Trials

Shima Abdollahi,[1,2] Omid Toupchian,[1] Ahmad Jayedi,[3] David Meyre,[4,5] Vivian Tam,[4] and Sepideh Soltani[2,6]

ABSTRACT

The aim of this study was to determine the effect of zinc supplementation on anthropometric measures. In this systematic review and dose–response meta-analysis, we searched PubMed, Scopus, ISI Web of Science, and the Cochrane Library from database inception to August 2018 for relevant randomized controlled trials. Mean differences and SDs for each outcome were pooled using a random-effects model. Furthermore, a dose–response analysis for zinc dosage was performed using a fractional polynomial model. Quality of evidence was evaluated using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) methodology. Twenty-seven trials ($n = 1438$ participants) were included in the meta-analysis. There were no significant changes in anthropometric measures after zinc supplementation in the overall analysis. However, subgroup analyses revealed that zinc supplementation increased body weight in individuals undergoing hemodialysis (HD) [3 trials, $n = 154$ participants; weighted mean difference (WMD) = 1.02 kg; 95% CI: 0.38, 1.65 kg; $P = 0.002$; $I^2 = 11.4\%$] and decreased body weight in subjects who are overweight/obese but otherwise healthy (5 trials, $n = 245$ participants; WMD = −0.55 kg; 95% CI: −1.06, −0.04 kg; $P = 0.03$; $I^2 = 31.5\%$). Dose–response analyses revealed a significant nonlinear effect of supplementation dosage on BMI ($P = 0.001$). Our data suggest that zinc supplementation increases body weight in patients undergoing HD and decreases body weight in individuals who are overweight/obese but otherwise healthy, although after normalization for study duration, the association observed in subjects who are overweight/obese disappeared. Although more high-quality studies are needed to reach a definitive conclusion, our study supports the view that zinc may be associated with body weight. *Adv Nutr* 2020;11:398–411.

Fokus auf Vitamin D:

Review

Molecular Basis Underlying the Therapeutic Potential of Vitamin D for the Treatment of Depression and Anxiety

Bruna R. Kouba [1], Anderson Camargo [1], Joana Gil-Mohapel [2,3,*] and Ana Lúcia S. Rodrigues [1,*]

Int. J. Mol. Sci. **2022**, 23, 7077. https://doi.org/10.3390/ijms23137077

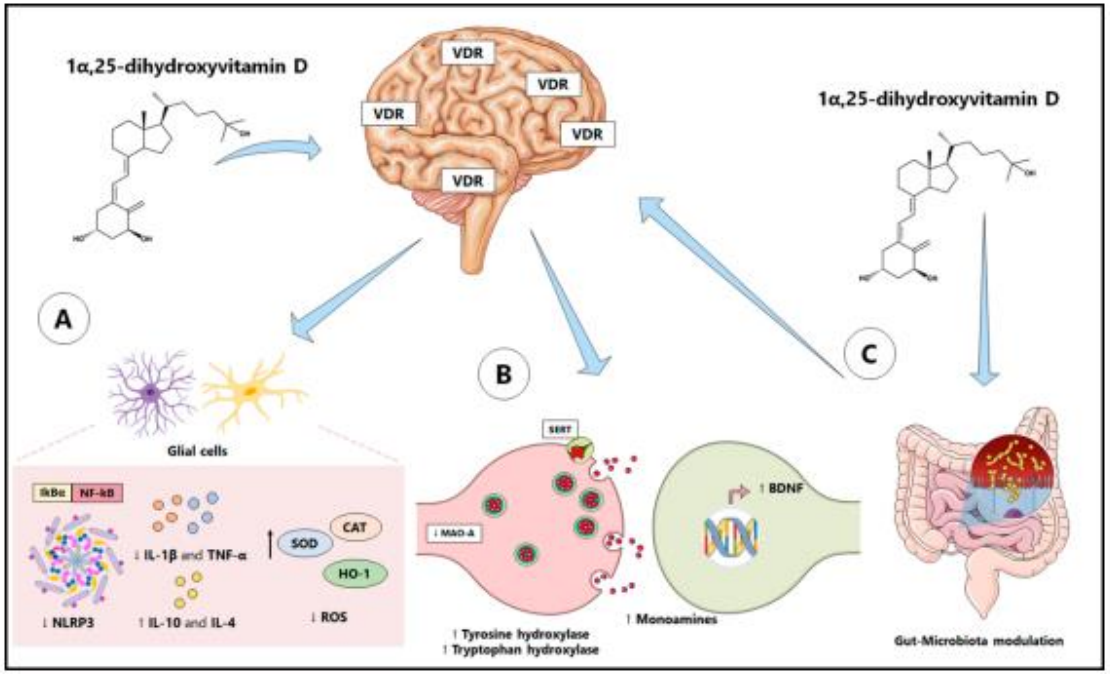

Figure 1. Antioxidant, anti-inflammatory, and neuromodulatory properties of vitamin D. Neurons and glial cells are able to express VDR in regions such as the prefrontal cortex and hippocampus. In these regions, the positive modulation of antioxidant enzymes, such as HO-1, CAT, and SOD, by calcitriol contributes to redox homeostasis and, consequently, to the attenuation of the neuroinflammatory process. Calcitriol is also able to increase the expression of IkBα, thus, inhibiting the nuclear translocation of NF-kB. As a consequence, less synthesis, oligomerization, and activation of the NLRP3 inflammasome occurs, resulting in attenuation of pro-inflammatory cytokines (such as IL-1β and TNF-α) and an increase in anti-inflammatory cytokines (such as IL-10 and IL-4) (**A**). In addition, vitamin D has also been shown to modulate the intestinal microbiota (**C**). Finally, vitamin D actively regulates the synthesis of monoamines and BDNF, thus, favoring the process of synaptic plasticity (**B**). Abbreviations: ↓: decreased; ↑: increased; BDNF: brain-derived neurotrophic factor; CAT: catalase; HO-1: heme oxygenase-1; IL-1β: interleukin-1β; IL-4: interleukin-4; IL-10: interleukin-10; IkBα: nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha; MAO: monoamine oxidase; NF-kB: nuclear factor kappa B; NLRP3: NOD-like receptor family pyrin domain-containing 3; ROS: reactive oxygen species; SERT: serotonin transporters; SOD: superoxide dismutase; TNF-α: tumor necrosis factor-alpha; VDR: vitamin D receptor.

Fokus auf Vitamin D:

The efficacy and safety of nutrient supplements in the treatment of mental disorders: a meta-review of meta-analyses of randomized controlled trials

Joseph Firth[1-3], Scott B. Teasdale[4,5], Kelly Allott[3,6], Dan Siskind[7,8], Wolfgang Marx[9], Jack Cotter[10], Nicola Veronese[11,12], Felipe Schuch[13], Lee Smith[14], Marco Solmi[15,16], André F. Carvalho[17,18], Davy Vancampfort[19,20], Michael Berk[6,9], Brendon Stubbs[21,22], Jerome Sarris[1,23]

Vitamin D was found to significantly reduce depressive symptoms in patients with clinical depression (N=948, n=4, SMD=0.58, 95% CI: 0.45-0.72, $p<0.01$, $I^2=0\%$). This estimate included data from non-blinded trials using intramuscular injections69. Nevertheless, in our re-analysis of data using only double-blind RCTs of oral supplements, similar positive effects were observed at doses of 1,500-7,143 IU/day (N=828, n=3, SMD=0.57, 95% CI: 0.43-0.71, $p<0.001$, $I^2=0\%$).

World Psychiatry 18:3 - October 2019

Vitamin D: Bedeutung der Dosis

Background: There have been several controversies about the correlation between vitamin D and depression. This study aimed to investigate the relationship between vitamin D supplementation and the incidence and prognosis of depression and to analyze the latent effects of subgroups including population and supplement strategy.

Methods: A systematic search for articles before July 2021 in databases (PubMed, EMBASE, Web of Science, and the Cochrane Library) was conducted to investigate the effect of vitamin D supplementation on the incidence and prognosis of depression.

Results: This meta-analysis included 29 studies with 4,504 participants, indicating that the use of vitamin D was beneficial to a decline in the incidence of depression (SMD: −0.23) and improvement of depression treatment (SMD: −0.92). Subgroup analysis revealed that people with low vitamin D levels (<50 nmol/L) and females could notably benefit from vitamin D in both prevention and treatment of depression. The effects of vitamin D with a daily supplementary dose of >2,800 IU and intervention duration of ≥8 weeks were considered significant in both prevention and treatment analyses. Intervention duration ≤8 weeks was recognized as effective in the treatment group.

Conclusion: Our results demonstrate that vitamin D has a beneficial impact on both the incidence and the prognosis of depression. Whether suffering from depression or not, individuals with low vitamin D levels, dose >2,800 IU, intervention duration ≥8 weeks, and all females are most likely to benefit from vitamin D supplementation.

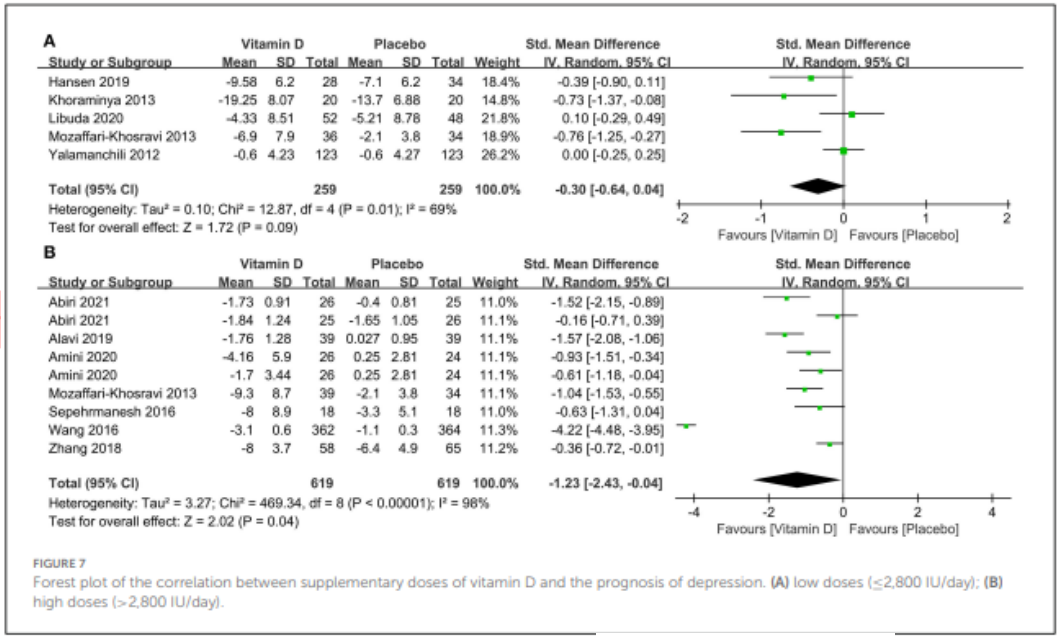

FIGURE 7
Forest plot of the correlation between supplementary doses of vitamin D and the prognosis of depression. **(A)** low doses (≤2,800 IU/day); **(B)** high doses (>2,800 IU/day).

Effect of vitamin D supplementation on the incidence and prognosis of depression: An updated meta-analysis based on randomized controlled trials

frontiers | Frontiers in Public Health

TYPE Review
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DOI 10.3389/fpubh.2022.903547

Fei Xie[†], Tongmin Huang[2†], Dandi Lou[3], Rongrong Fu[3], Chaoxiong Ni[4], Jiaze Hong[2] and Lingyan Ruan[1*]

Fragen und Diskussion

Die Kosten der Diagnostik (Auszug):

Analyse	Kosten TP
Vitamin A	68
Vitamin B1	76
Folsäure i. Ery.	9
Vitamin B12	25
Vitamin D	53
Ferritin	7.9
Magnesium i. Ery.	50
Phosphat	3.2
Selen	105
Zink	44

Vielen Dank für Ihre Aufmerksamkeit

Exkurs: Refeeding Syndrom

REVIEW

Refeeding syndrome : physiological background and practical management

Aminda De Silva ,[1] Jeremy M D Nightingale[2]

DEFINITION

Refeeding syndrome (RFS) is a potentially fatal condition commonly characterised by rapid changes in fluid and electrolyte balance leading to problems of cardiac arrthymias, cardiac and respiratory failure. Other manifestations include acute fatty liver, endocrine and haematological abnormalities, acute thiamine deficiency and neurological syndromes such as delirium and centropontine myelinolysis. Hidden sepsis, a separate dangerous problem, can also occur in malnourished individuals and sometimes is mistaken for a manifestation of RFS.

Key points

- Refeeding syndrome describes the adverse clinical and biochemical problems that may result from feeding malnourished patients via any route, be it oral, enteral or parenteral.
- Clinicians need to be aware of it and assume most malnourished patients are at risk.
- Hypophosphataemia is the most commonly used marker for refeeding problems and it commonly occurs when artificial nutritional support is started (especially with carbohydrate) and can rarely cause death.
- Hypophosphataemia is more common with oral/enteral feeding than parenteral nutrition.
- Hypomagnesaemia, hypokalaemia, hypoglycaemia (occasionally hyper) and thiamine deficiency may occur.
- Sodium retention (causing oedema) is common, especially after glucose (and sodium) are given.
- Non-protein energy is given as 50/50 carbohydrate (CHO)/lipid and initially at <50% requirements.

De Silva A, Nightingale JMD. *Frontline Gastroenterology* 2020;**11**:404–409. doi:10.1136/flgastro-2018-101065

Exkurs: Refeeding Syndrom

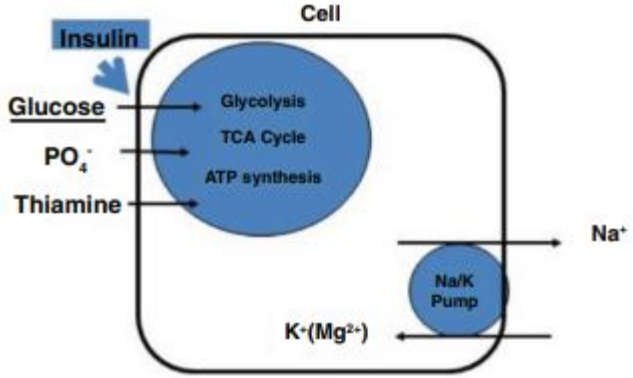

Figure 1 Simplified diagram to show key events in refeeding syndrome. TCA, tricarboxylic acid.

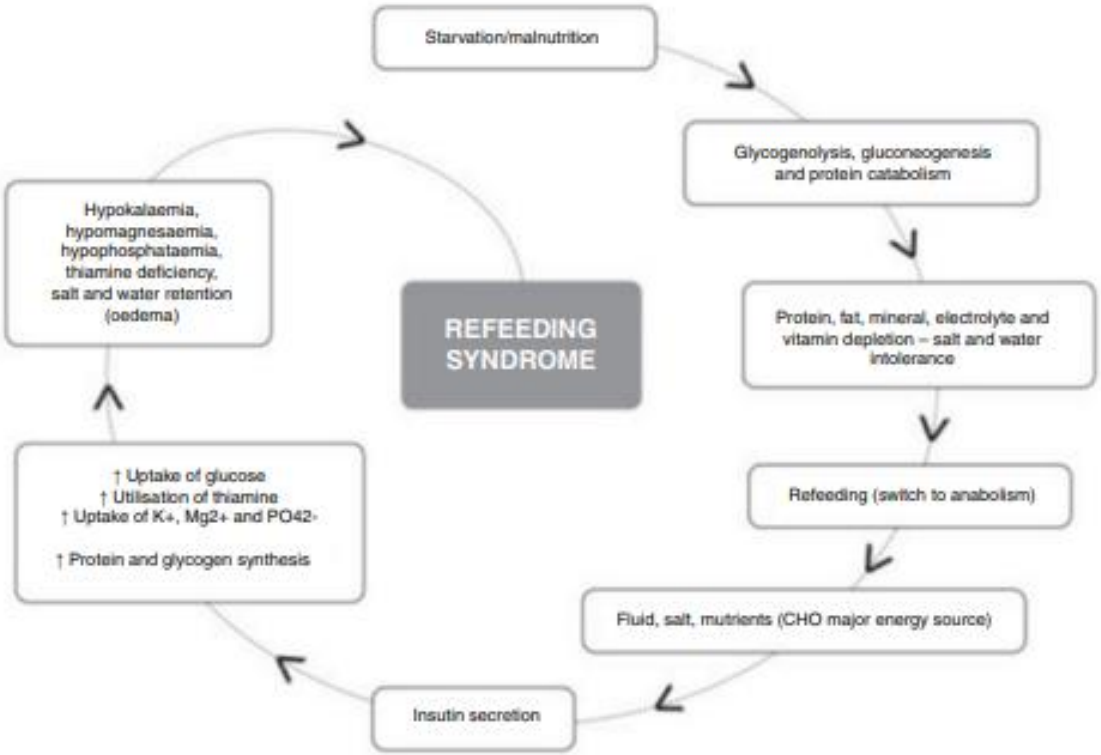

Figure 2 Diagram summarising events in refeeding syndrome.

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Table 1 Effects of hypophosphataemia	
Muscular	Weakness (diaphragm), respiratory failure
	Rhabdomyolysis
Cardiac	Biventricular failure, low blood pressure
	Arrhythmias, sudden death
Haematological	Low and dysfunctional white blood cells, red blood cells, platelets
	Haemolysis
Neurological	Weakness, lower motor neurone-type paralysis (loss of reflexes)
	Cranial nerve palsies
	Confusion, ataxia, tremors, fits, coma
Hepatic	Dysfunction (especially alcohol excess)

nutrition for over 5 *days*). Generous vitamin and electrolyte supplementation may be given while monitoring closely and increasing the calorie intake reasonably rapidly from 10 to 20 kcal/kg/24 hours. Often patients in this category are not hungry, but over the course of a few days, the restoration of their appetite is an indication that the risks of refeeding have been managed

Box 1 UK National Institute for Health and Care Excellence risk factors for developing refeeding syndrome[20]

One or more of the following:

- Body mass index (BMI) <16 kg/m^2;
- Unintentional weight loss >15% within last 3–6 months;
- Little or no nutritional intake for >10 days;
- Low potassium, magnesium or phosphate prior to feeding.

Two or more of the following:

- BMI <18.5 kg/m^2;
- Unintentional weight loss >10% within last 3–6 months;
- Little or no nutritional intake for >5 days;
- A history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics.

De Silva A, Nightingale JMD. *Frontline Gastroenterology* 2020;**11**:404–409. doi:10.1136/flgastro-2018-101065

Exkurs: Refeeding Syndrom

Article

Outcomes of an Accelerated Inpatient Refeeding Protocol in 103 Extremely Underweight Adults with Anorexia Nervosa at a Specialized Clinic in Prien, Germany

Thorsten Koerner [1], Verena Haas [2], Julia Heese [1], Matislava Karacic [1], Elmar Ngo [1], Christoph U. Correll [2,3,4], Ulrich Voderholzer [1,5,*] and Ulrich Cuntz [1,6]

Abstract: Background: In mildly to moderately malnourished adolescent patients with anorexia nervosa (AN), accelerated refeeding protocols using higher initial calory supply coupled with phosphate supplements were not associated with a higher incidence of refeeding syndrome (RS). It is unclear whether this is also a feasible approach for extremely malnourished, adult AN patients. Methods: Outcomes of a clinical refeeding protocol involving a targeted initial intake of ≥2000 kcal/day, routine supplementation of phosphate and thiamine as well as close medical monitoring, were evaluated. A retrospective chart review including AN patients with a body mass index (BMI) <13 kg/m^2 was conducted, to describe changes in weight, BMI, and laboratory parameters (phosphate, creatine kinase, hematocrit, sodium, liver enzymes, and blood count) over four weeks. Results: In 103 female patients (age, mean ± standard deviation (SD) = 23.8 ± 5.3 years), BMI between admission and follow-up increased from 11.5 ± 0.9 to 13.1 ± 1.1 kg/m^2 and total weight gain within the first four weeks was 4.2 ± 2.0 kg (mean, SD). Laboratory parameter monitoring indicated no case of RS, but continuous normalization of blood parameters. Conclusions: Combined with close medical monitoring and electrolyte supplementation, accelerated refeeding may also be applied to achieve medical stabilization in extremely underweight adults with AN without increasing the risk of RS.

J. Clin. Med. **2020**, *9*, 1535; doi:10.3390/jcm9051535

Exkurs: Refeeding Syndrom

Table 3. Current, national refeeding guidelines for malnourished patients with anorexia nervosa.

Guidelines	Age	Recommended Energy Intake
Australia and New Zealand	adult	1400 kcal/d [22]
Europe [23]	adult	start at 10, slowly increase to 15 kcal/kg/d (Day 1–3)
United Kingdom: Royal College of Psychiatrists [24]	adult	10–20 kcal/kg/d
United Kingdom: MARSIPAN [25]	adult	5–20 kcal/kg/d
American Psychiatric Association/American Dietetic Association [26]	adult	30–40 kcal/kg/d (1000–1600 kcal/d)
United Kingdom: Junior MARSIPAN [27]	<18 years	20 kcal/kg/d

The following criteria were used to define a case of RS:

- Critical deterioration of the general condition (e.g., severe edema, pericardial effusion, and weakness).
- Critical drop of serum phosphate to values <0.75 mmol/L.
- Increase in creatine kinase to >1000 U/L.
- Decrease of hematocrit to <25%.
- Decrease of serum sodium to <125 mmol/L.
- For the assessment of the clinical condition, the following laboratory parameters were also used:
  - Aspartate aminotransferase (GOT) (>35 U/L) and alanine aminotransferase (GPT) (>35 U/L) for estimation of liver cell damage.
  - Leukocytes (<3.98 G/L), hemoglobin (<11.2 g/L), and thrombocytes (<182 G/L) for the assessment of bone marrow function.

J. Clin. Med. **2020**, *9*, 1535; doi:10.3390/jcm9051535

Exkurs: Refeeding Syndrom

All patients received 200 mg thiamine daily for the first four weeks and 612–1024 mg phosphate daily for the first two weeks. With this supplementation, serum phosphate levels increased during the observation period (Table 2, Figure 1). On admission, 7 (6.2%) of the 103 subjects had hypophosphatemia with values <0.75 mmol/L, and one subject had a critical value <0.5 mmol/L. After one week, only five patients had slightly decreased serum phosphate levels (<0.75 mmol/L) and after two weeks only three patients.

The following data were collected on admission and weekly thereafter for 4 weeks in clinical care and are presented in this study: body weight, height, BMI, and blood work (creatin kinase, phosphate, sodium, GOT, GPT, leucocytes, hematocrit, hemoglobin, and thrombocytes).

The following cut-off values were used to define pathological blood values: creatin kinase, >170 U/L; phosphate, <0.75 mmol/L; sodium, <12 mmol/L; GOT, GPT >35 U/L; leucocytes, <3.98 G/L, hemoglobin, <25%; thrombocytes, <182 G/L.

Table 2. Laboratory values at admission (T1) and after four weeks (T5), Mean and SD for 103 pt.

	CK	Phos	Sodium	GOT	GPT	Leuco	Haematocrit	Hb	Pla
cut off	>170 U/L	<0.75 mmol/L	<125 mmol/L	>35 U/L	>35 U/L	<3.98 G/L	<25 %	<11.2 g/L	<182 G/L
At admission	222 ± 974 (27–9941)	1.15 ± 0.29 (0.46–1.92)	138.7 ± 4.7 (112.0–152.0)	67.9 ± 117.8 (15.3–1078.0)	85.6 ± 121.9 (8.3–742.0)	4.1 ± 1.8 (1.1–14.0)	37.0 ± 5.0 (22.9–44.7)	12.5 ± 1.8 (7.5–15.6)	242 ± 81 (45–494)
After four weeks	75 ± 54 *** (22–374)	1.34 ± 0.18 *** (0.94–1.88)	141.3 ± 2.7 *** (133.0–148.0)	25.9 ± 8.0 *** (12.7–53.0)	42.3 ± 21.6 *** (10.7–133.0)	4.8 ± 1.8 *** (1.6–12.8)	36.1 4.2 ** (23.0–42.0)	11.6 ± 1.5 *** (6.6–14.3)	300 ± 92 *** (185–849)
pathologic at admission	19 (103) 18.4%	7 (103) 6.8%	7 (103) 6.8%	54 (103) 52.4%	62 (103) 60.2%	54 (103) 52.4%	23 (103) 22.3%	21 (103) 20.4%	21 (103) (20.4%)
pathologic after four weeks	6 (103) 5.8% *	0 (103) 0% *	0 (103) 0% *	12 (103) 11.7% ***	61 (103) 59.2%	37 (101) 36.6% *	22 (101) 21.8%	36 (101) 35.6% *	0 (101) (0%) ***

The T-test was used for normally distributed differences in blood values between two measurement points. A Wilcoxon test was used for non-normally distributed differences. For the percentage change in the proportion of pathological blood values, the two-sided Fischer's exact test was used * $p < 0.05$; ** $p < 0.01$;***. $p < 0.001$; data in parenthesis are ranges. Abbreviations: CK: creatine kinase; Phos: inorganic phosphate; GOT: aspartate aminotransferase; GPT: alanine aminotransferase; Leuco: leukocytes; Hb: hemoglobin; Pla: platelets; SD, standard deviation.

J. Clin. Med. **2020**, *9*, 1535; doi:10.3390/jcm9051535

Exkurs: Refeeding Syndrom

As part of the routine refeeding protocol: weight was monitored daily in underwear (Scale: Seca Model Nr. 6357021004). Height was measured at admission. Blood work was performed at least once a week including blood count (without white blood cell differential), electrolytes (sodium, potassium, phosphate, and chloride), transaminases, gamma-glutamyl-transferase (γ-GT), creatine kinase, creatinine, and urea. Further investigations included continuous monitoring of vital signs (electrocardiogram (ECG), heart rate, and blood pressure), hematocrit (<25%) and weight progression, routine clinical examination including neurological assessment and vital signs, SUSS test, sonography, and bioelectrical impedance analysis to assess edema status. Phosphate (612 mg to 1224 mg/d) and thiamine (200 mg/d) were supplemented routinely for two weeks and phosphate thereafter if needed. In some patients, the administration of diuretics was necessary at times when either the severity of the edema was subjectively too stressful or the heart rate per minute exceeded the systolic blood pressure in mmHg.

All patients received three meals with an average total energy content of 2000 kcal per day from day 1 after admission to the ward, divided into three main meals with a choice between vegetarian and non-vegetarian menus. The caloric intake was adjusted and increased according to weight development to aim for an increase in body weight of 700–1000 g/week. The criterion for sufficient food intake was weight gain, which should be at least 100 g per day. If the agreed amount of food intake for weight gain could not be achieved, the portion size of one or more main meals was increased, and up to three snacks between meals were added. In addition, liquid food was offered to substitute for energy losses in case a part of the meals could not be eaten. All meals were therapeutically accompanied by a nurse or therapist in a 1:6 group supervision. Patient adherence to dietary intake was supported through daily therapeutic contacts and medical rounds. Patients ate their meals in a stable group

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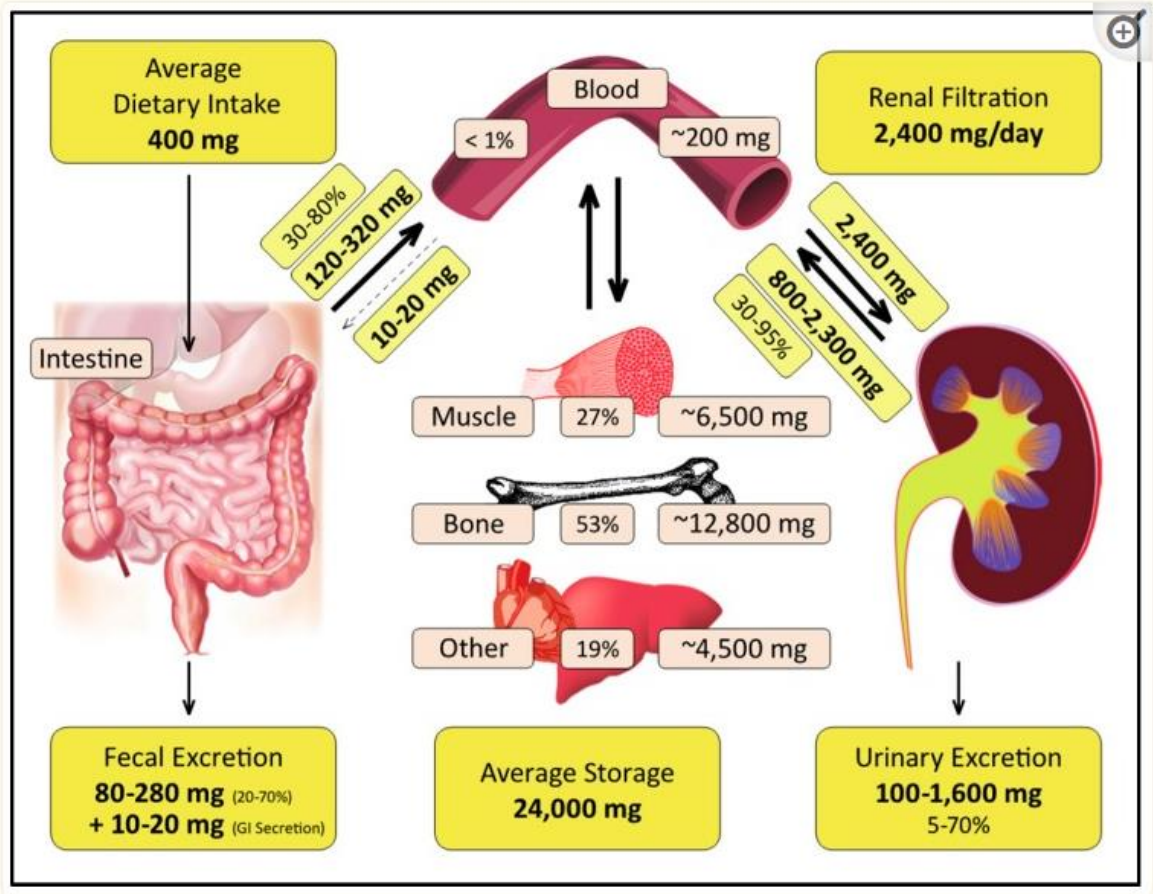
Anorexia Nervosa

- Wichtige Laborparameter:
- Elektrolyte (Na, K, Cl, Ca, P; ggf. Mg und Zn in den Erythrozyten)
- Nüchternglucose
- Nierenwerte (Kreatinin)
- Leberwerte (ALAT, ASAT, GGT)
- Schilddrüsenwerte (TSH, ggf. fT3, fT4)
- Vitamine (Vitamin B12, Vitamin B1)

Binge Eating

- Wichtige Laborparameter:
- Schilddrüsenwerte
- Glucose
- HbA1c
- Leberwerte (ASAT, ALAT, GGT)
- Nierenwerte (Kreatinin, Harnstoff)
- Lipidstatus (LDL, HDL, Gesamtcholesterin, Triglyceride)
- Vitaminstatus (Folsäure, Vitamin B12, Vitamin D, Homocystein)
- Schilddrüsenwerte (TSH, ggf. fT3, fT4)

Exkurs: Magnesium



Nutrients. 2018 Sep; 10(9): 1202.
Published online 2018 Sep 1. doi: 10.3390/nu10091202

Challenges in the Diagnosis of Magnesium Status

Jayme L. Workinger,[1,*] Robert. P. Doyle,[2] and Jonathan Bortz[1]

Exkurs: Magnesium

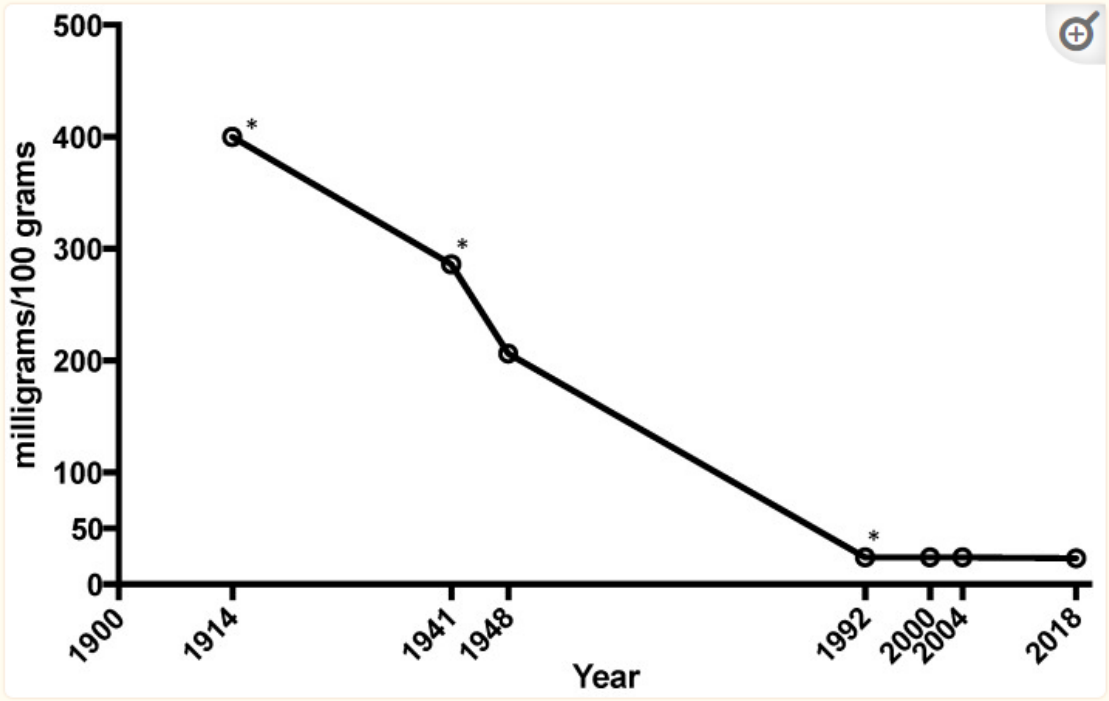


Figure 2

The average mineral content of calcium, magnesium, and iron in cabbage, lettuce, tomatoes, and spinach has dropped 80–90% between 1914 and 2018 [30,34,35,36,37]. Asterisks indicate numbers could not be independently verified.

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Jayme L. Workinger,[1,*] Robert. P. Doyle,[2] and Jonathan Bortz[1]

Exkurs: Magnesium

The tight control of magnesium serum levels, representing only 0.8% of total body stores (see Section 2.4), therefore serves as a poor proxy for the 99.2% of magnesium in other tissues that constitutes the body's true magnesium status. Furthermore, this narrow serum range feeds the common perception of clinicians that magnesium levels rarely fluctuate, and therefore, are not indicative of the condition for which the blood tests are ordered. Therefore, practitioners are apt to order blood tests for magnesium infrequently, if at all, and if a magnesium level is in the patient chart, it is more often as part of a blood test panel and not purposely ordered to determine the magnesium status [89,124,125,126]. This contributes significantly to magnesium deficiency not being recognized as a modifiable nutritional intervention, and magnesium in general, being the neglected mineral that it is.

Red blood cells' (RBC; erythrocyte and monocyte) magnesium levels are often cited as preferable to serum or plasma levels due to their higher magnesium content (0.5% vs. 0.3%, respectively). Some RBC studies report correlation to magnesium status particularly when subjects are placed on long-term (~3 months) magnesium replete or deplete diets. However, most studies using RBC magnesium endpoints do not satisfy this long-term design and have not been performed in nearly enough randomized clinical studies to be considered sufficiently robust or reliable (Table 1) [127,128,129]. In addition, the majority of RBC studies do not validate the method through inter-compartmental sampling (e.g., urine and muscle), challenging the claim that this test is a reliable representation of the large magnesium pool.

Nutrients. 2018 Sep; 10(9): 1202.
Published online 2018 Sep 1. doi: 10.3390/nu10091202
Challenges in the Diagnosis of Magnesium Status
Jayme L. Workinger,[1,*] Robert. P. Doyle,[2] and Jonathan Bortz[1]

Exkurs: Zink

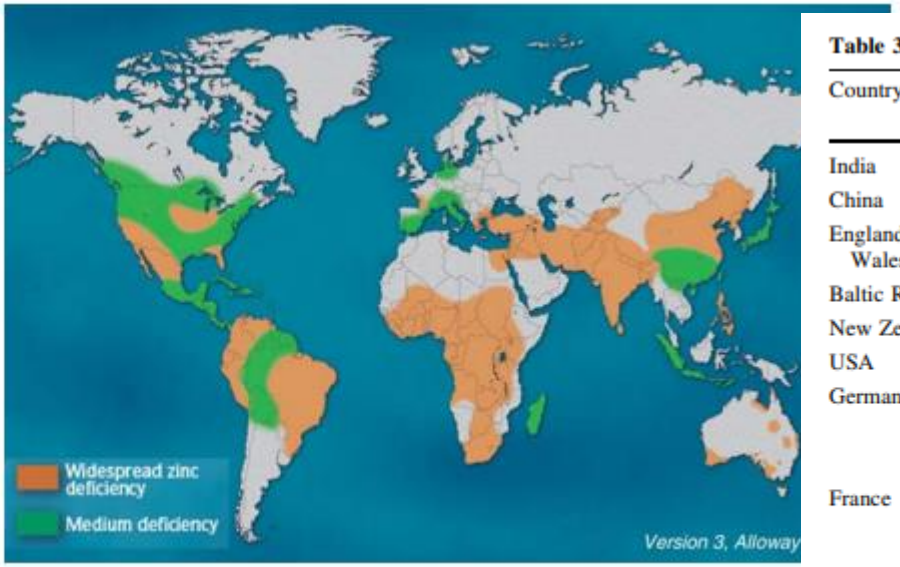

Fig. 1 Global distribution of reported cases of zinc deficiency in crops (Alloway 2008b). Reproduced with permission from the International Zinc Association (IZA) and International Fertilizer Industry Association (IFA)

Table 3 Representative total zinc concentrations in soils

Country	Soil type	Total Zn (mg kg^{-1})
India	Various	2–1205
China	Various	3–790
England & Wales	Various	5–3548
Baltic Region	Various	33 (5–125)
New Zealand	Pasture soils	42–91
USA	Various	43 (3–264)
Germany	Sandy soils	27 (median)
	Loams/silts	59 (median)
	Clay soils	76 (median)
France	Sandy soils	17 (median)
	Silty soils (<20% clay)	40 (median)
	Loamy (20–30% clay)	64 (median)
	Clayey (30–50% clay)	98 (median)
	Very clayey (>50% clay)	132 (median)
World	Various	64 (mean)

Sources: Baize (2000), Gorny et al. (2000), Reimann et al. (2003), Kabata-Pendias (2001), Longhurst et al. (2004)

Soil factors associated with zinc deficiency in crops and humans

B. J. Alloway

Die andere Perspektive: Malnutrition bei Kindern in Indien

- Wie sieht hier das Profil der Mikronährstoffmängel aus?
- Gibt es Unterschiede zu den anorektischen PatientInnen?

Indian Journal of Pediatrics
https://doi.org/10.1007/s12098-023-04520-0

ORIGINAL ARTICLE

Check for updates

Status of Vitamin B12, Zinc, Copper, Selenium, Manganese, Molybdenum and Cobalt in Severe Acute Malnutrition

Laxmi Kamath[1] · Vinod H. Ratageri[1] · Apurva S. Kanthi[1] · S. R. Fattepur[1] · R. H. Desai[1]

Malnutrition bei Kindern in Indien

Table 3 Proportion of SAM children deficient in various micronutrients

		0–6 mo	7–12 mo	13–59 mo	Total	Fischer exact test
Vitamin B12 (pg/ml)	Deficiency (<203)	3 (33.3)	8 (32)	6 (37.5)	17 (34)	*p* 0.924
	No deficiency (≥203)	6 (66.7)	17 (68)	10 (62.5)	33 (66)	
	Total	9 (100)	25 (100)	16 (100)	50 (100)	
Zinc (µg/dl)	Deficiency (<65)	0 (0)	2 (10.5)	2 (13.3)	4 (9.5)	*p* 0.826
	No deficiency (≥65)	8 (100)	17 (89.5)	13 (86.7)	38 (90.5)	
	Total	8 (100)	19 (100)	15 (100)	42 (100)	
S. Copper (µg/dl)	Deficiency (<63.5)	0 (0)	3 (15)	2 (14.3)	5 (11.9)	*p* 0.686
	No deficiency (≥63.5)	8 (100)	17 (85)	12 (85.7)	37 (88.1)	
	Total	8 (100)	20 (100)	14 (100)	42 (100)	
Cobalt (µg/L)	Deficiency (<1.5)	3 (100)	13 (100)	8 (100)	24 (100)	NA
	No deficiency (≥1.5)	0 (0)	0 (0)	0 (0)	0 (0)	
	Total	3 (100)	13 (100)	8 (100)	24 (100)	
Molybdenum (µg/L)	Deficiency (<0.7)	0 (0)	2 (15.4)	1 (12.5)	3 (12.5)	*p* 0.99
	No deficiency (≥0.7)	3 (100)	11 (84.6)	7 (87.5)	21 (87.5)	
	Total	3 (100)	13 (100)	8 (100)	24 (100)	
Selenium (µg/L)	Deficiency (<60)	0 (0)	0 (0)	0 (0)	0 (0)	NA
	No deficiency (≥60)	3 (100)	13 (100)	8 (100)	24 (100)	
	Total	3 (100)	13 (100)	8 (100)	24 (100)	
Manganese (µg/L)	Deficiency (<7.1)	0 (0)	0 (0)	0 (0)	0 (0)	NA
	No deficiency (≥7.1)	3 (100)	13 (100)	8 (100)	24 (100)	
	Total	3 (100)	13 (100)	8 (100)	24 (100)	

Status of Vitamin B12, Zinc, Copper, Selenium, Manganese, Molybdenum and Cobalt in Severe Acute Malnutrition

Wie sieht es insgesamt aus bei Frauen?

Micronutrient deficiencies among preschool-aged children and women of reproductive age worldwide: a pooled analysis of individual-level data from population-representative surveys

Gretchen A Stevens, Ty Beal*, Mduduzi N N Mbuya, Hanqi Luo, Lynnette M Neufeld, on behalf of the Global Micronutrient Deficiencies Research Group†*

Status of Vitamin B12, Zinc, Copper, Selenium, Manganese, Molybdenum and Cobalt in Severe Acute Malnutrition

Die weltweite Prävalenz im Vergleich

	Iron deficiency	Zinc deficiency	Folate deficiency	Any core deficiency*	Sample size (any core deficiency)†	Vitamin A deficiency	Vitamin B_{12} deficiency	Vitamin D deficiency	Any sentinel deficiency‡
Afghanistan, 2013	33% (28–39)	23% (18–29)	..	69% (52–87)	1044	10% (8–13)	..	78% (74–82)	..
Azerbaijan, 2013	43% (40–45)	..	35% (31–39)	76% (66–85)	2551	0% (0–1)	20% (16–24)	..	..
Bangladesh, 2011	9% (6–13)	41% (35–48)	84% (79–87)	91% (86–94)§	699	6% (4–9)	7% (3–13)	..	..
Cambodia, 2014	4% (2–6)	63% (57–69)	61% (55–67)	84% (80–88)§	689	3% (2–4)	1% (0–3)	4% (3–7)	85% (80–88)
Cameroon, 2009	18% (13–24)	84% (78–89)	35% (28–44)	92% (88–94)§	332	1% (0–2)	14% (10–20)	..	..
Côte d'Ivoire, 2007	22% (19–26)	..	91% (88–94)	96% (93–99)	792	1% (0–1)	18% (12–26)	..	..
Ecuador, 2012	17% (16–19)	57% (55–59)	10% (9–11)	68% (66–69)§	7230	3% (2–4)	1% (1–2)	..	..
Ethiopia, 2015	9% (7–11)	21% (17–24)	32% (28–36)	49% (44–53)§	1607	5% (3–7)	14% (11–16)	..	..
Georgia, 2009	2% (1–2)	..	52% (44–59)	72% (60–83)	407	..	..	..	..
Ghana, 2017	20% (16–24)	..	59% (53–65)	81% (74–89)	466	2% (1–3)	7% (5–10)	..	..
Guatemala, 2013–16	16% (15–17)	25% (18–34)	..	48% (37–59)	209	0% (0–0)	15% (12–17)	0% (0–3)¶	..
India, 2016–18	37% (34–41)	31% (28–35)	58% (54–61)	81% (77–84)§	2348	12% (9–15)	29% (26–33)	24% (21–28)	89% (86–92)
Malawi, 2015–16	15% (12–19)	58% (52–64)	23% (18–29)	72% (67–77)§	746	3% (2–5)	13% (9–17)	..	..
Mexico, 2006	35% (32–39)	29% (25–34)	..	61% (54–68)	1813	..	..	..	..
Mexico, 2012	43% (39–46)	..	3% (2–4)	67% (55–79)	3603	..	2% (1–2)	..	..
Nepal, 2016	19% (16–21)	25% (22–28)	16% (13–19)	49% (46–52)§	2125	1% (1–2)	..	..	..
Pakistan, 2011	42% (41–44)	46% (44–48)	..	78% (72–84)	7390	42% (40–44)	52% (50–55)	31% (29–33)	..
UK, 2008–19	21% (18–24)	10% (8–12)	19% (16–22)	43% (39–46)§	1310	1% (0–2)	7% (5–9)	22% (19–25)	55% (51–59)
USA, 2015–16	22% (17–27)	14% (10–19)	0% (0–1)	32% (26–39)§	551	..	..	3% (2–6)	..
Viet Nam, 2010	18% (16–20)	67% (63–71)	22% (19–25)	78% (74–81)§	1348	1% (1–2)	12% (9–15)	9% (7–13)	78% (73–83)

Data are % (95% uncertainty interval) or n. The age range for non-pregnant women of reproductive age was 15–19 years for India. Other age ranges varied slightly by dataset and are specified in the appendix (pp 21–22). Definitions of deficiencies are specified in table 1. Empty cells indicate missing data or exclusion of the micronutrient in the dataset. *Core micronutrients are iron, zinc, and folate. †Sample size varies by deficiency; sample size for any included core deficiency is shown in this column. ‡Sentinel micronutrients are iron, zinc, vitamin A, folate, vitamin B_{12}, and vitamin D; prevalence is only shown for datasets containing all six sentinel micronutrients. §These values are for datasets containing all three core micronutrients; the other values are predicted as described in the appendix (pp 26–28). ¶No individuals identified with deficiency in the sample; uncertainty interval does not take into account complex survey design.

***Table 3*: Prevalence of deficiency by type for each dataset included in the analysis for non-pregnant women aged 15–49 years**

langanese,
trition

Wie sieht es in der Schweiz aus?

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Published online 2021 Mar 24. doi: 10.1016/j.clnesp.2021.03.012

PMCID: PMC7987506
PMID: 34024545

Strengthening the immunity of the Swiss population with micronutrients: A narrative review and call for action

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Die Ernährungspyramide – Theorie und Praxis

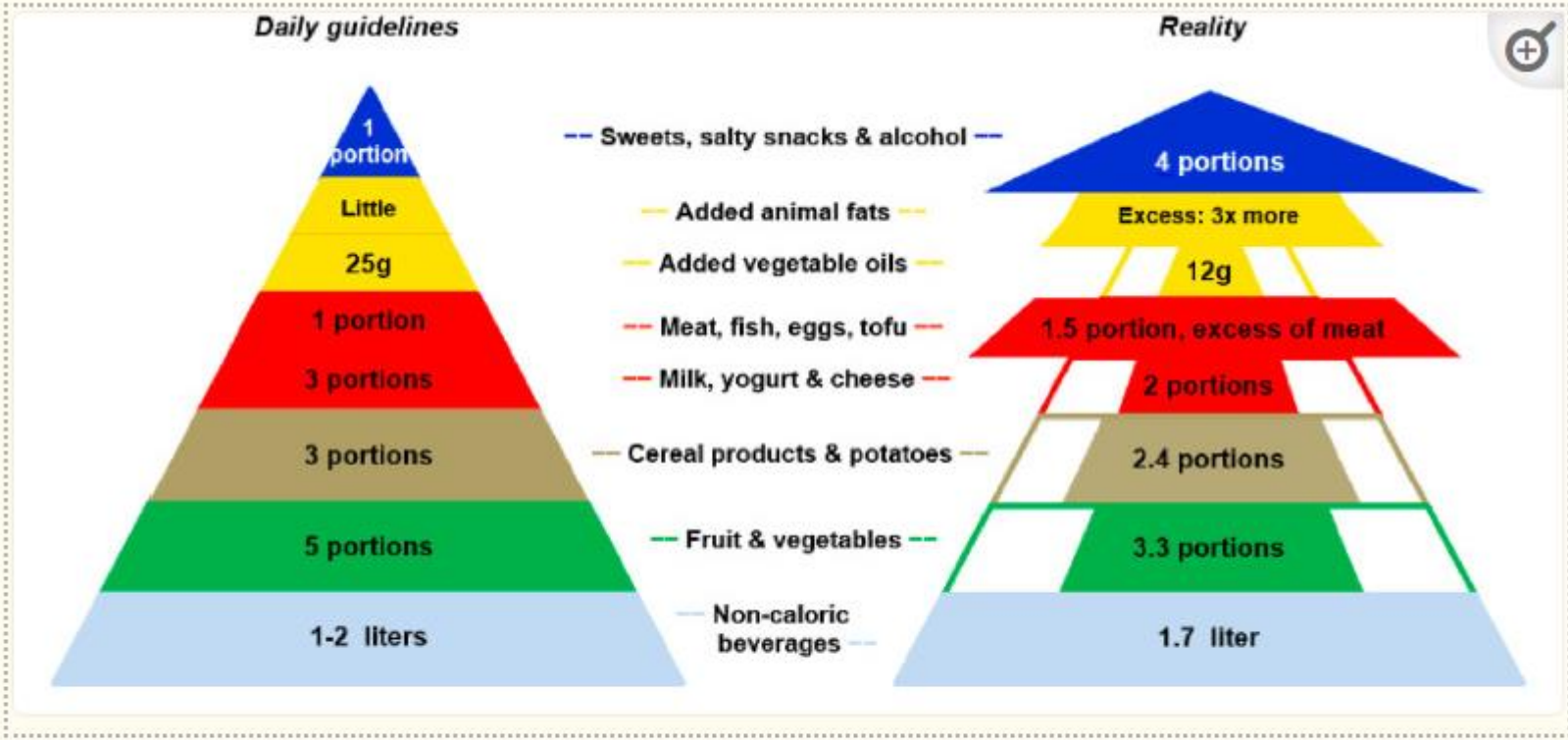

Fig. 1

Comparison of Swiss Dietary recommendations (left) with the actual daily food consumption (right) [107].

Strengthening the immunity of the Swiss population with micronutrients: A narrative review and call for action

Versorgung in der Schweiz

Table 3
Likelihood of deficiency in the Swiss population.

Nutrient	Adults	Older adults 65+
Vitamin C	+ Men	+++
Vitamin D	+	+++
Fe	++ Women	++
Se	+	+++
Zn	+	++
n-3 PUFA	+	++

Table 4
Composition of the proposed nutritional supplements. Correction of iron deficiency requires individualized therapy and is therefore not mentioned.

Nutrient	Dosage/day	Upper intake limits/day
Vitamin C	200 mg	2000 mg
Vitamin D	800–1600 IU (20–40 µg)	4000 IU(100 µg)
Se	100 µg	400 µg
Zn	20 mg	40 mg
n-3 PUFA	500 mg	5000 mg

Population representative dietary intake data for zinc are not available for Switzerland. However, a cross-sectional study reported a plasma zinc concentration of 85 ± 12 µg/dl for omnivorous Swiss adults, with 11% of subjects at risk for deficiency. The percentage of adults at risk of zinc deficiency was even higher for vegetarians (19%) and vegans (47%) [116].

Selenium intake and status may be suboptimal in the European population, including the Swiss population [118–120]. A selenium concentration of 98.7 µg/L in plasma or serum are required to optimize glutathione peroxidase (GPX) activity, a level required for optimal antioxidant defense. A study in the Swiss healthy blood donors from different regions reported a mean plasma selenium 90 µg/L [121], and the data suggested that a substantial portion of the population has a suboptimal selenium status [120]. This may be due to the low selenium content of the Swiss soil which translates in a low selenium content of locally produced foods. These data could be considered for public health strategies [15], although such measures are slow and require several years to yield results [122].

A report by the Federal Office of Public Health on vitamin D status estimated that about 50% of the Swiss population has inadequate serum 25(OH)D concentrations (<50 nmol/L) and that only 30% has optimal levels (>75 nmol/L). A population-based study showed that low serum 25(OH)D levels are common among Swiss adults, in particular during winter months [110]. Also, a recent

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