

Practice Name: Vibrant IT4
Practice
Provider Name: Demo Client,
DDD (999994)
Street Address: TEST STREET
City: TEST CITY
State: KY
Zip #: 42437

MICRONUTRIENT

Your guide to customized optimal nutrition.



MK-0074-03



1 (866) 364-0963



www.vibrant-america.com



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1021 Howard Ave. Ste. B
San Carlos, CA 94070

Tests flagged with * were developed by and performance characteristics were determined by Vibrant America. Indicated tests are not FDA-cleared or approved. The laboratory is regulated under CLIA and is CAP certified hence qualified to perform high-complexity testing. This test is used for clinical purposes. It should not be regarded as investigational or for research. Tests flagged with ¹ were performed at Vibrant Genomics. Tests flagged with ² have analytics done at Vibrant Wellness. Laboratory Director: Mervyn Sahud, MD CLIA: 05D2078809 CLF: 00346278 Vibrant America Clinical Laboratory, 1021 Howard Avenue, Suite B, San Carlos, CA 94070. Phone: +1(866)364-0963; FAX: +1(650)508-8260; Email: support@vibrant-america.com

Final Report Date:	12-16-2020 14:30	Specimen Collected:	12-15-2020 14:29
Accession ID:	2012160002	Specimen Received:	12-16-2020 08:29

LAST NAME	FIRST NAME	MIDDLE NAME	DATE OF BIRTH	GENDER	PHYSICIAN ID
REPORT	DEMO		1998-07-05	Male	999994

PATIENT

Name: DEMO REPORT
Date of Birth: 1998-07-05
Gender: Male
Age: 22

Fasting: FASTING

PROVIDER

Practice Name: Vibrant IT4 Practice
Provider Name: Demo Client, DDD (999994)
Phlebotomist: 608
Street Address: TEST STREET
City: TEST CITY
State: KY
Zip #: 42437
Telephone #:
Fax #: 000-000-0000

CRITICAL VALUE FOR Potassium Serum - 2.1 mmol/L
CRITICAL VALUE FOR Potassium Serum - 2.1C mmol/L

Vibrant America is pleased to present to you micronutrient testing that provides a comprehensive extracellular and intracellular assessment of the levels of the most important vitamins, minerals, antioxidants, fatty acids, and amino acids to help you make healthy lifestyle choices in consultation with your healthcare provider.

Testing Methodology: The blood sample is spun down so that the serum can be taken from the top and RBCs from the bottom. The remaining sample is processed to isolate PBMCs (Peripheral Blood Mononuclear cells). All three subsets are processed separately to isolate appropriate micronutrients for injection into mass-spectrometry. Micronutrients measured in RBCs include: folate, omega-3 and omega-6 fatty acids, and magnesium. Serum micronutrient measurements provide extracellular levels. WBC measurements are done and total WBC counts are taken on an automated cell counter. Intracellular WBC levels are normalized to the total WBC count in a patient's sample.

Interpretation of Report: The summary report provided lists the major categories under which the micronutrients are classified and gives a score for each category on a scale of 0-100. Please note that a micronutrient might be essential for more than one category. The contribution to the category score of each micronutrient is based on how important it is for the category based on literature references and the supporting evidence linking it to the respective category. A category score more than 85 is considered optimal, 40-85 considered moderate risk and below 40 is considered high risk. Complete micronutrient testing including serum, WBC and RBC needs to be ordered for the scores to populate.

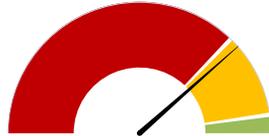
The 'abnormal' section beneath each category score lists the micronutrients which are high/low for the category and the 'normal' section indicates the micronutrients which fall in-range. A suggestion table for suitable foods and space for supplement suggestions by your provider is found at the end of the summary page.

The test results of micronutrient levels are displayed in a graphical format for each Serum, WBC, and RBC levels as applicable. The graph has red and green background color to indicate whether the micronutrient is in-range or out of range. The reference ranges are also provided next to the graphs to help with the interpretation. A trendline of the micronutrient level for the respective patient will be available which will indicate the historical values along with current test results when multiple testing is performed on the patient.

The statements in this report have not been evaluated by the Food and Drug Administration. Please consult your physician/dietitian for medication, treatment, or life style management. This product is not intended to diagnose, treat, or cure any disease.

Please Note - It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your physician before making any changes.

LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
REPORT	DEMO	MALE	1998-07-05	2012160002	12-15-2020 14:29

NEUROLOGICAL, COGNITIVE FUNCTION AND MOOD	LIVER DETOXIFICATION	GASTROINTESTINAL BARRIER
Current Result: 72	Current Result: 72	Current Result: 77
		
Previous Result: 73	Previous Result: 74	Previous Result: 77
ABNORMAL	ABNORMAL	ABNORMAL
Low: Vitamin K1, Magnesium High: Inositol, Iron	Low: Vitamin B1, Vitamin B2	Low: Vitamin D, 25-OH
NORMAL	NORMAL	NORMAL
Glutamine, Coenzyme Q10, Cysteine, Vitamin E, Choline, Zinc, Iron, Magnesium, Vitamin B6, Vitamin B12, Vitamin C, Vitamin D3, Vitamin K1, Folate, Inositol, Glutathione, EPA, DHA	Glutamine, Cysteine, Selenium, Vitamin E, Vitamin A, Vitamin B3, Vitamin B6, Vitamin B5, Vitamin C, Folate, Vitamin B1, Vitamin B2, Glutathione	Glutamine, Cysteine, Vitamin A, Glutathione, EPA, DHA

BONE, JOINT AND MUSCLE HEALTH	CARDIOVASCULAR HEALTH	MITOCHONDRIAL FUNCTION, SKIN AND ANTI AGING
Current Result: 29	Current Result: 58	Current Result: 78
		
Previous Result: 44	Previous Result: 66	Previous Result: 71
ABNORMAL	ABNORMAL	ABNORMAL
Low: Calcium, Vitamin D, 25-OH, Vitamin K2, Magnesium High: Iron	Low: Potassium, Vitamin K2, Magnesium High: Iron	Low: Vitamin D, 25-OH, Magnesium
NORMAL	NORMAL	NORMAL
Glutamine, Zinc, Iron, Magnesium, Vitamin A, Vitamin C, Vitamin K2	Coenzyme Q10, Vitamin E, Carnitine, Iron, Magnesium, Vitamin B3, Vitamin K2, Folate, EPA, DHA, LA	Glutamine, Cysteine, Selenium, Vitamin E, Zinc, Copper, Magnesium, Vitamin A, Vitamin B3, Vitamin B12, Vitamin C, Folate, Glutathione, EPA, DHA

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LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
REPORT	DEMO	MALE	1998-07-05	2012160002	12-15-2020 14:29

ABNORMAL	 CELLULAR SERUM		 COMMON FOOD RESOURCES	 SUGGESTED SUPPLEMENTATION
	Vitamin B1		↓	Pork, salmon, flax seeds, legumes, tofu, acorn squash
Vitamin B2		↓	Beef, fortified tofu, dairy, salmon, mushrooms, pork, spinach	
Vitamin B12	↓			
Vitamin D, 25-OH		↓	Cod liver oil, swordfish, canned salmon, mackerel, sardines	
Vitamin K1	↓		Swiss chard, collards, parsley, broccoli, turnip greens	
Vitamin K2	↓		Natto, cheese, dairy curds	
Calcium	↓	↓	Plain yogurt, tofu, mozzarella cheese, sardines, cheddar cheese, milk	
Manganese	↓		Pecans, brown rice, green tea, black tea, oatmeal, spinach	
Iron	↑		Oysters, spinach, mussels, chicken liver, white beans, dark chocolate	
Magnesium	↓		Oats, mackerel, spinach, almonds, cashews, swiss chard	
Inositol		↑	Oranges, cantaloupe, dried prunes, navy beans, grapefruit, limes	
Potassium		↓C		
Arginine		↓	Sesame seeds, soy protein, peanut, crab, shrimp	
Leucine		↓	Whey protein, soy protein, hemp, beef, hemp	
Total Omega-3	↓			
AA	↓		Meat, poultry, eggs; *safflower oil, sunflower seeds, pine nuts, sunflower oil	
AA/EPA	↓			
Omega-3 Index	↑			

(Your provider will discuss any nutrient deficiencies identified on the report. The suggested supplementation section will be filled by provider.)

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What Do I Do With The Information From This Test?

CELLULAR: Normal SERUM: Deficient	CELLULAR:Deficient SERUM: Normal/Excess	CELLULAR: Deficient SERUM: Deficient
<p>Long term nutrient status is optimal, but short term needs improvement. Recommended interventions:</p> <ul style="list-style-type: none"> * increase dietary intake of nutrient * increase supplementation dosage * medications may have an effect on depletion 	<p>Short term status of micronutrients is optimal, but cellular absorption may be a problem. Recommended interventions:</p> <ul style="list-style-type: none"> *increase dietary intake of nutrient *increase supplementation dosage *consider status of synergistic nutrients for cellular absorption *consider levels of oxidative stress on nutrient depletion *consider follow up testing to identify the source of malabsorption 	<p>Short term and long term status of micronutrients is not optimal, suggesting low dietary intake and both intestinal and cellular malabsorption as possible causes. Recommended interventions:</p> <ul style="list-style-type: none"> * increase dietary intake of nutrient * increase supplementation dosage * medications may have an effect on depletion * consider follow up testing to identify the source of malabsorption

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REPORT	DEMO	MALE	1998-07-05	2012160002	12-15-2020 14:29

Micronutrient	Serum			WBC			RBC		
	Current	Previous	Ref	Current	Previous	Ref	Current	Previous	Ref
Vitamin A	76.2	80.4	43.1~107.0 (mcg/dL)	1.2	0.9	0.9~17.3 (pg/MM WBC)			
Vitamin B1	25.1 ↓	16.7 ↓	188.4~428.9 (nmol/L)	3.10	2.00	0.10~7.00 (pg/MM WBC)			
Vitamin B2	55.2 ↓	110.8	82.6~126.1 (mcg/L)	0.5	1.9	0.2~3.6 (pg/MM WBC)			
Vitamin B3	21.2	17.1	2.6~36.1 (ng/mL)	291.6	146.6	39.6~303.5 (pg/MM WBC)			
Vitamin B6	21.8	24.6	2.8~76.2 (ng/mL)	3.8	3.9	0.5~9.7 (pg/MM WBC)			
Vitamin B12	690	750	232~1245 (pg/mL)	1.40 ↓	1.40 ↓	2.00~11.99			
Vitamin B5	220.3	170.0	22.7~429.2 (mcg/L)	11.3	8.0	2.5~32.8 (pg/MM WBC)			
Vitamin C	0.8	0.7 ↓	0.8~1.7 (mg/dL)	3.4	2.6	0.5~9.7 (ng/MM WBC)			
Vitamin D3	0.9	1.3	0.4~1.8 (ng/mL)	46.3	42.0	25.9~246.6 (pg/MM WBC)			
Vitamin D, 25-OH	19.6 ↓	27.9 ↓	30.0~108.0 (ng/mL)						
Vitamin E	19.4	21.6	7.4~30.6 (mg/L)	157.3	146.1	18.4~1031.1 (pg/MM WBC)			
Vitamin K1	1.30	1.40	0.10~8.10 (ng/mL)	0.09 ↓	0.90 ↑	0.10~0.71 (pg/MM WBC)			
Vitamin K2	1.08	1.10	0.10~5.19 (ng/mL)	0.02 ↓	0.03 ↓	0.10~0.89 (pg/MM WBC)			
Folate	11.6	8.7	≥4.6 (ng/mL)				103.2	101.6	≥95.5 (ng/mL)

Vitamins

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	Micronutrient	Serum			WBC			RBC		
		Current	Previous	Ref	Current	Previous	Ref	Current	Previous	Ref
Minerals	Calcium	8.7 ↓	9.2	8.9~10.6 (mg/dL)	13 ↓	16	15~120 (ng/MM WBC)			
	Manganese	0.8	0.9	0.3~2.0 (ng/mL)	1 ↓	2	2~75 (pg/MM WBC)			
	Zinc	0.8	0.7	0.5~1.0 (mcg/mL)	8	6	4~15 (ng/MM WBC)			
	Copper	1.4	1.3	0.6~1.8 (mcg/mL)	13	11	2~15 (ng/MM WBC)			
	Chromium	0.20	0.20	0.10~0.70 (ng/mL)						
	Iron	135	110	59~158 (ug/dL)				126.3 ↑	116.1	88.9~117.0 (mg/dL)
	Magnesium	2.3	2.2	1.6~2.6 (mg/dL)				1.8 ↓	2.9 ↓	3.6~7.7 (mg/dL)
	Copper to Zinc Ratio	1.7	1.9	0.9~2.6						
Metabolites	Choline	14.6	12.8	6.8~31.0 (nmol/mL)	0.5	0.4	0.2~1.5 (ng/MM WBC)			
	Inositol	76.8 ↑	67.1 ↑	20.5~60.7 (nmol/mL)	1.40	1.50	0.10~2.50 (ng/MM WBC)			
	Carnitine	19.0	20.1	11.6~43.4 (nmol/mL)	0.8	0.9	0.3~1.5 (ng/MM WBC)			
	MMA	0.15	0.11	0.10~0.50 (nmol/mL)						
	Sodium	143	141	136~145 (mmol/L)						
	Potassium	2.1 ↓C	3.0 ↓	3.5~5.1 (mmol/L)						

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	Micronutrient	Serum			WBC			RBC		
		Current	Previous	Ref	Current	Previous	Ref	Current	Previous	Ref
Amino Acids	Asparagine	68.4	72.1	39.2~89.8 (nmol/mL)	0.8	0.9	0.5~2.8 (ng/MM WBC)			
	Glutamine	459.0	504.4	393.5~699.3 (nmol/mL)	4.6	3.1	1.4~7.0 (ng/MM WBC)			
	Serine	114.1	86.8 ↓	94.2~246.8 (nmol/mL)	6.9	8.7	1.8~19.8 (ng/MM WBC)			
	Arginine	59.4 ↓	67.6 ↓	81.6~249.0 (nmol/mL)						
	Citrulline	23.9	38.5	18.7~47.5 (nmol/mL)						
	Isoleucine	106.4	101.9	25.5~158.9 (nmol/mL)						
	Valine	178.5	206.5	155.9~368.0 (nmol/mL)						
	Leucine	56.2 ↓	66.1 ↓	101.2~249.3 (nmol/mL)						
Antioxidants	Coenzyme Q10	0.81	1.12	0.56~2.78 (µg/mL)	89.3	96.8	39.6~225.3 (pg/MM WBC)			
	Cysteine	19.7	23.4	3.4~37.0 (nmol/mL)	97.4	108.3	60.0~565.0 (pg/MM WBC)			
	Glutathione				954.2	866.1	98.7~1163.0 (pg/MM WBC)			
	Selenium	158.3	169.0	109.8~218.4 (ng/mL)	246	256	234~1050 (pg/MM WBC)			

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	Micronutrient	Serum			WBC			RBC		
		Current	Previous	Ref	Current	Previous	Ref	Current	Previous	Ref
Fatty Acids: Omega-3 & 6	EPA							0.96	1.10	0.15~2.26 (%)
	DPA							1.66	1.73	0.45~1.80 (%)
	DHA							5.17	6.04	2.42~10.52 (%)
	Total Omega-3							2.27 ↓	2.48 ↓	3.25~13.99 (%)
	LA							4.01	3.46	3.22~10.49 (%)
	AA							0.90 ↓	2.20 ↓	5.50~19.01 (%)
	Total Omega-6							27.80	25.10	11.03~34.96 (%)
	AA/EPA							<2.0 ↓	2.0 ↓	2.5~10.9
	Omega-3 Index							14.78 ↑	10.11	8.00~12.65 (%)

WBC Count

WBC Count	Current	Reference Range	Previous
Lymphocyte Count (x 10 ³ /μL)	1.32	1.32~3.57	1.34 (11/16/2020)
Neutrophil Count (x 10 ³ /μL)	2.15	1.78~5.38	2.20 (11/16/2020)
WBC (x 10 ³ /μL)	4.55	4.23~9.07	4.30 (11/16/2020)



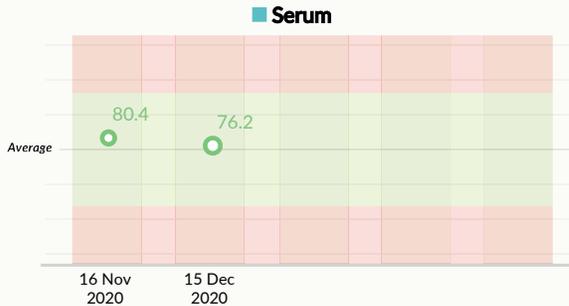
LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
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Vitamin A

Current Result:

76.2
(mcg/dL)

Ref Range:
43.1~107.0
(mcg/dL)

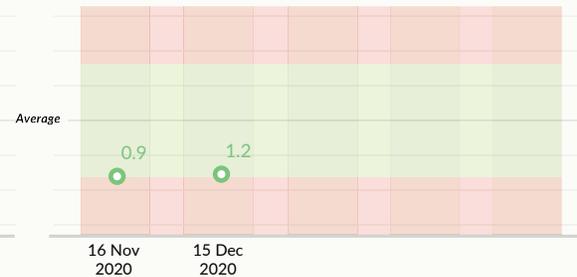


WBC

Current Result:

1.2
(pg/MM WBC)

Ref Range:
0.9~17.3
(pg/MM WBC)

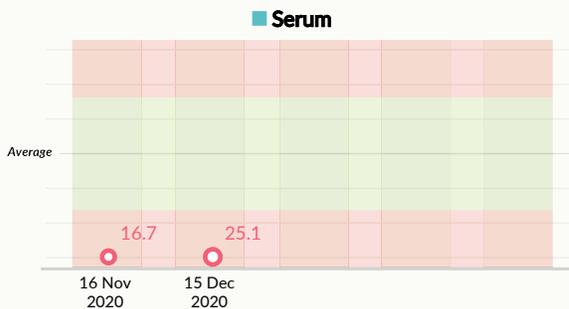


Vitamin B1

Current Result:

25.1 ↓
(nmol/L)

Ref Range:
188.4~428.9
(nmol/L)

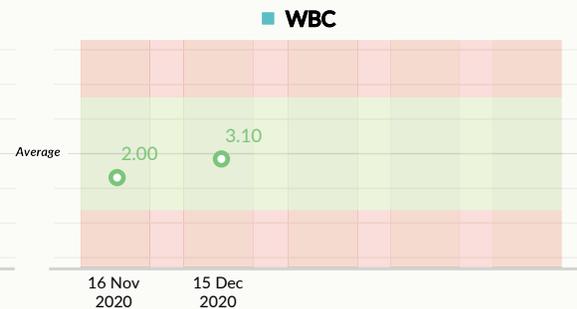


WBC

Current Result:

3.10
(pg/MM WBC)

Ref Range:
0.10~7.00
(pg/MM WBC)

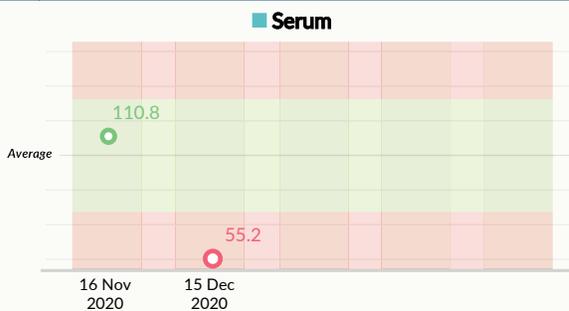


Vitamin B2

Current Result:

55.2 ↓
(mcg/L)

Ref Range:
82.6~126.1
(mcg/L)

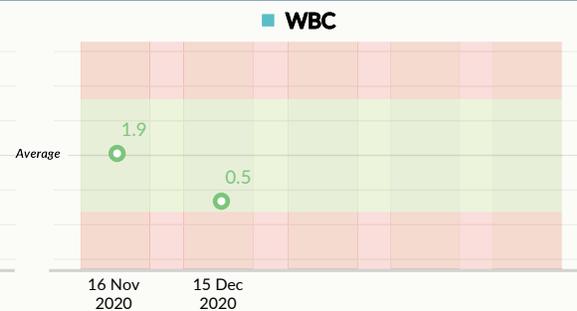


WBC

Current Result:

0.5
(pg/MM WBC)

Ref Range:
0.2~3.6
(pg/MM WBC)

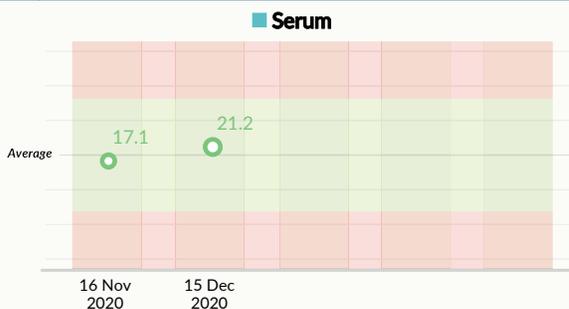


Vitamin B3

Current Result:

21.2
(ng/mL)

Ref Range:
2.6~36.1
(ng/mL)

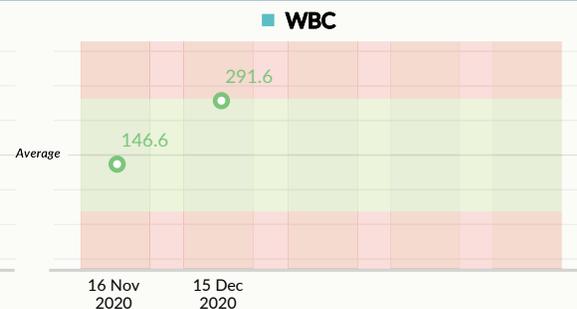


WBC

Current Result:

291.6
(pg/MM WBC)

Ref Range:
39.6~303.5
(pg/MM WBC)





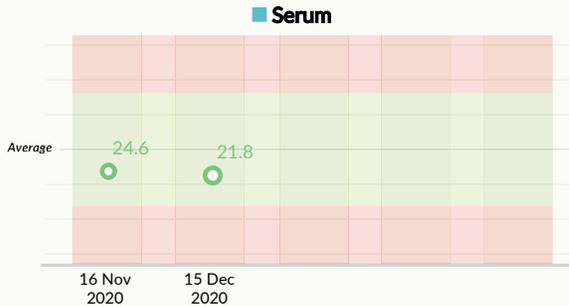
LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
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Vitamin B6

Current Result:

21.8
(ng/mL)

Ref Range:
2.8~76.2
(ng/mL)

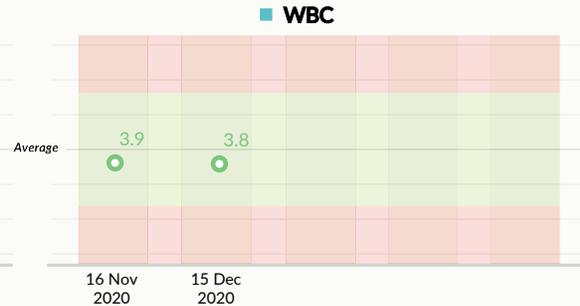


WBC

Current Result:

3.8
(pg/MM WBC)

Ref Range:
0.5~9.7
(pg/MM WBC)

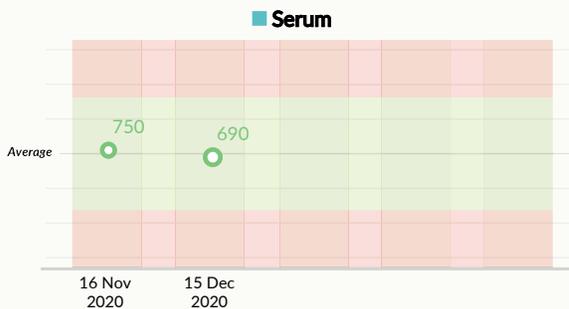


Vitamin B12

Current Result:

690
(pg/mL)

Ref Range:
232~1245
(pg/mL)

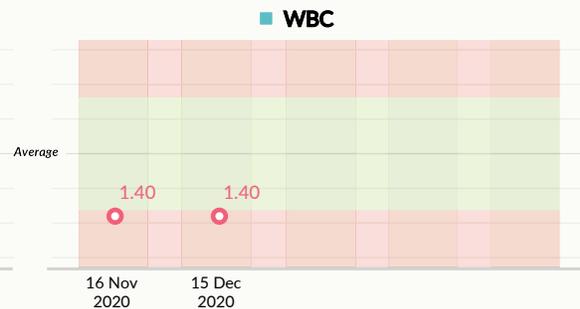


WBC

Current Result:

1.40 ↓

Ref Range:
2.00~11.99



Vitamin B5

Current Result:

220.3
(mcg/L)

Ref Range:
22.7~429.2
(mcg/L)

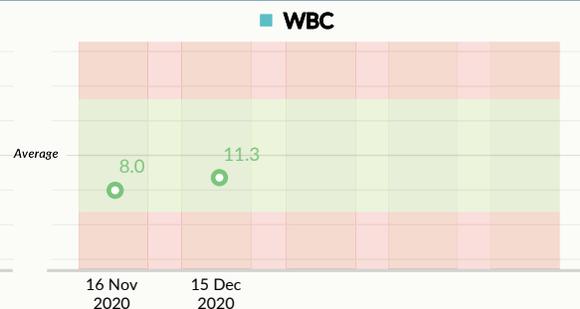


WBC

Current Result:

11.3
(pg/MM WBC)

Ref Range:
2.5~32.8
(pg/MM WBC)

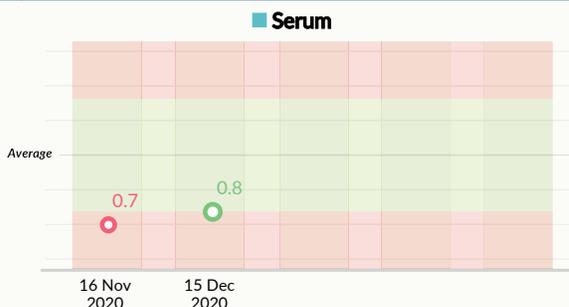


Vitamin C

Current Result:

0.8
(mg/dL)

Ref Range:
0.8~1.7
(mg/dL)

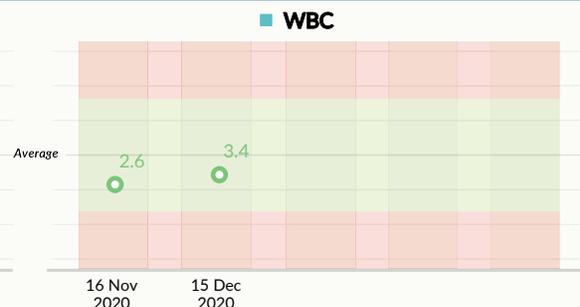


WBC

Current Result:

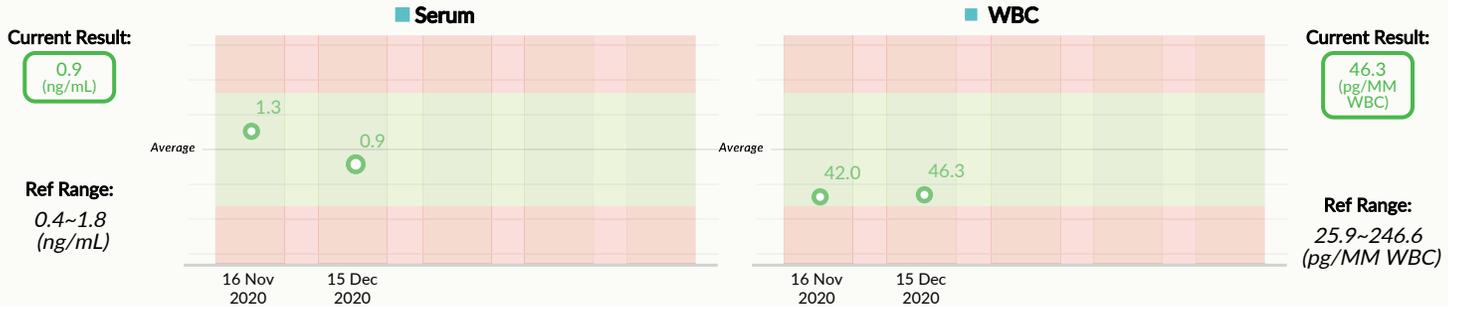
3.4
(ng/MM WBC)

Ref Range:
0.5~9.7
(ng/MM WBC)

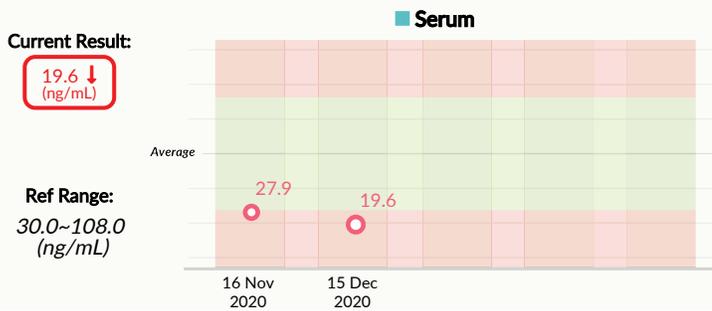


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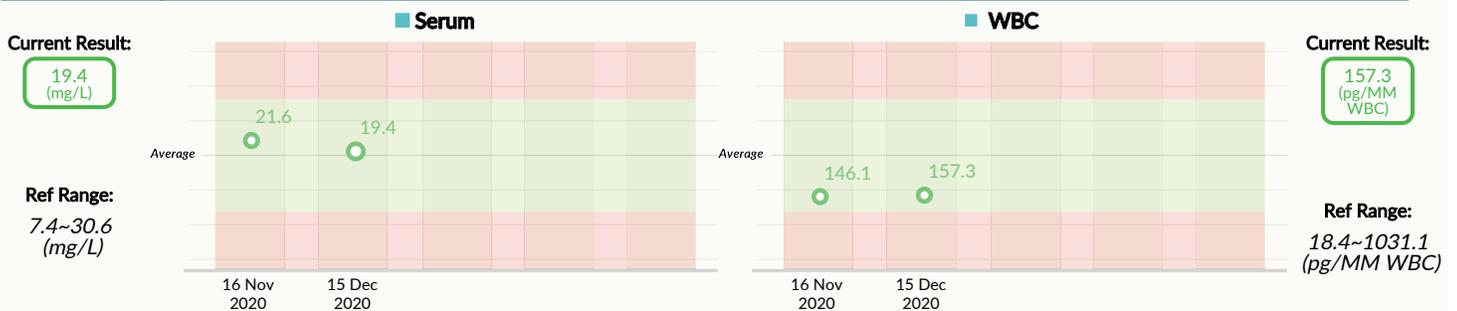
Vitamin D3



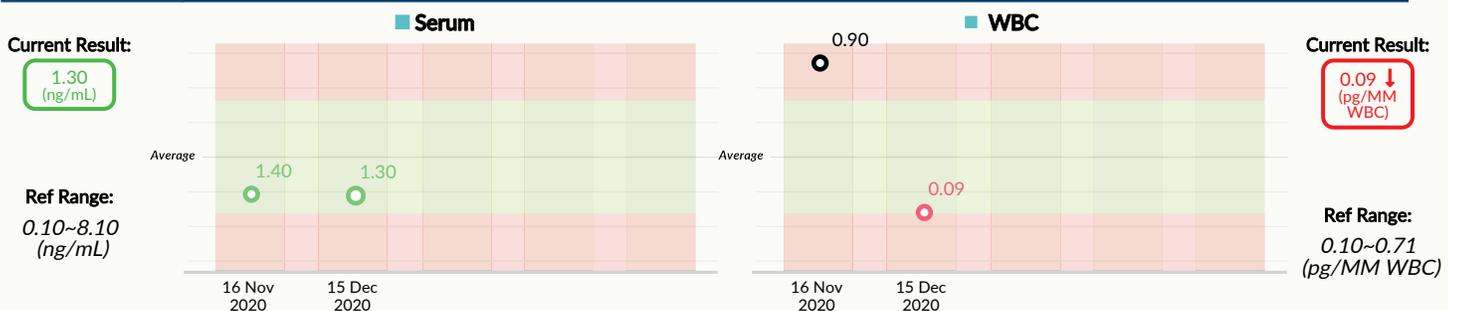
Vitamin D, 25-OH



Vitamin E

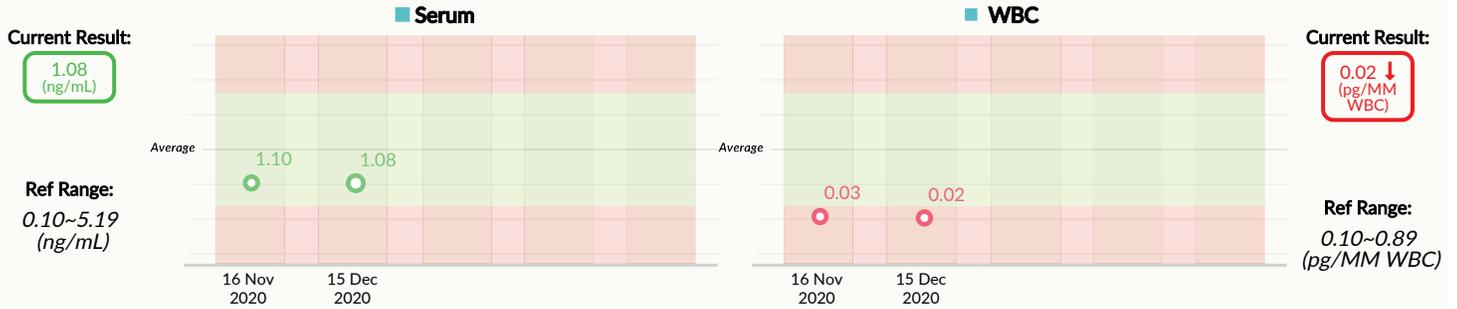


Vitamin K1

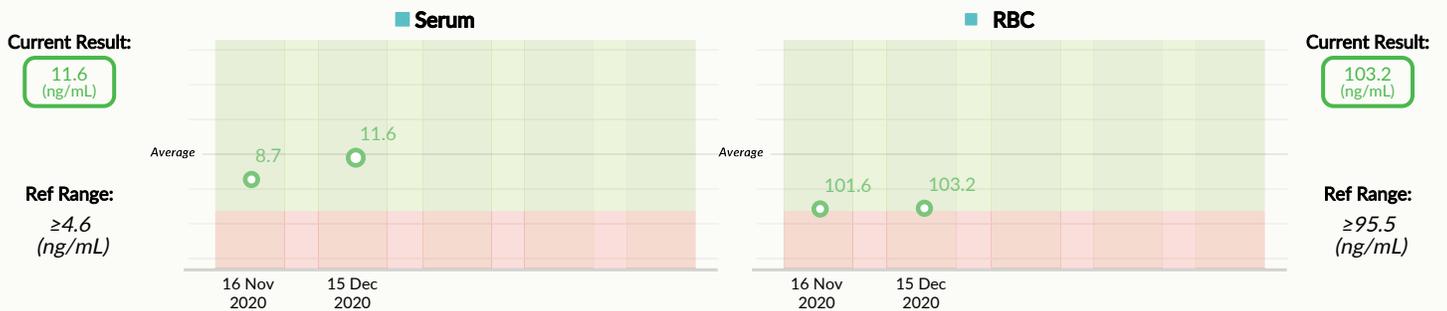


LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
REPORT	DEMO	MALE	1998-07-05	2012160002	12-15-2020 14:29

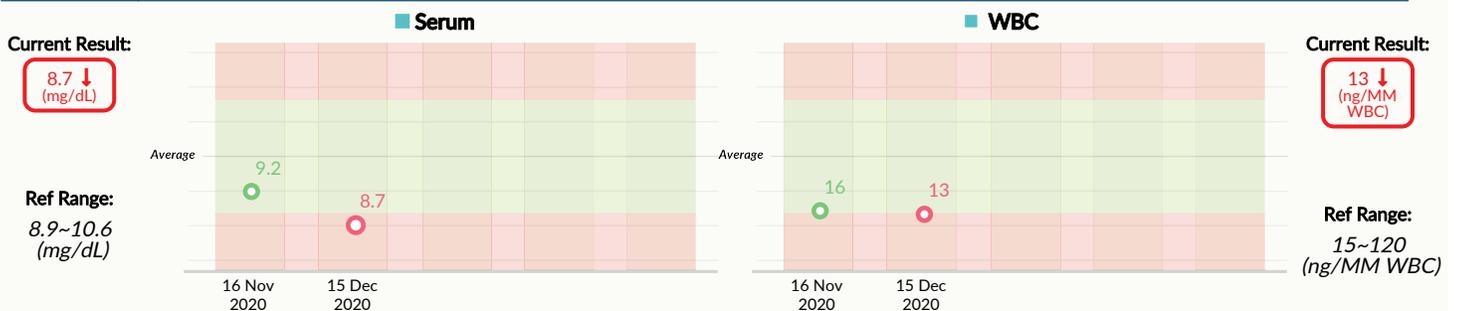
Vitamin K2



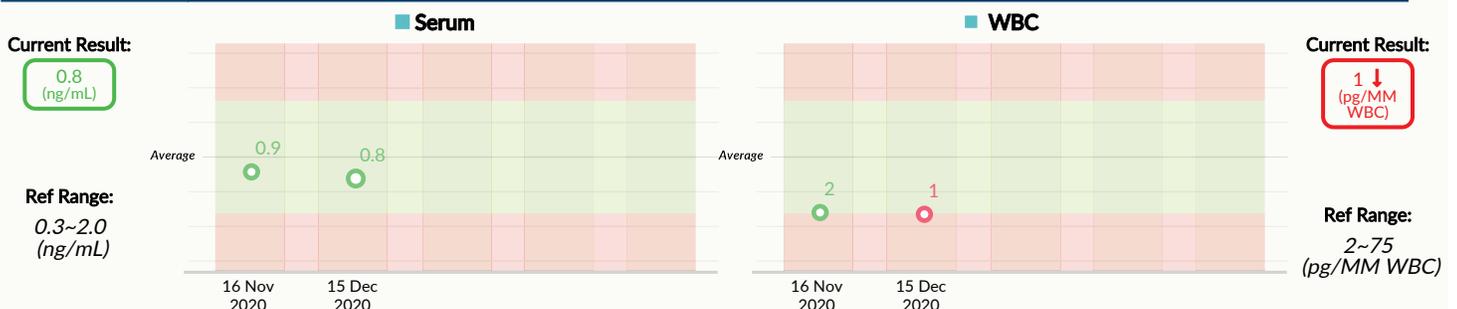
Folate



Calcium



Manganese



MICRONUTRIENT

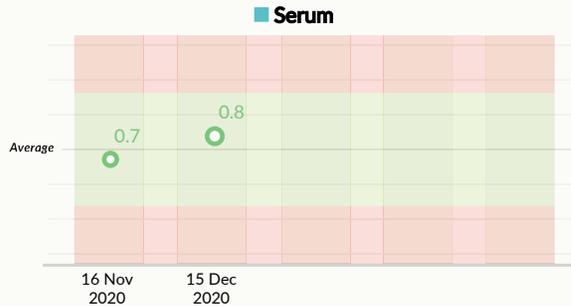
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Zinc

Current Result:

0.8
(mcg/mL)

Ref Range:
0.5~1.0
(mcg/mL)

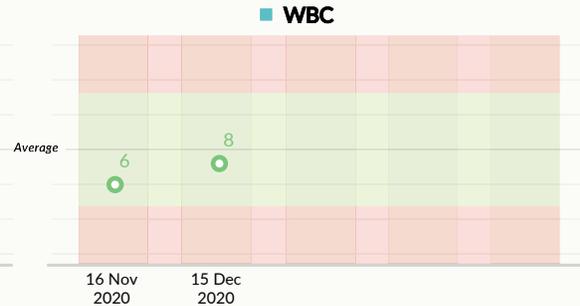


WBC

Current Result:

8
(ng/MM WBC)

Ref Range:
4~15
(ng/MM WBC)

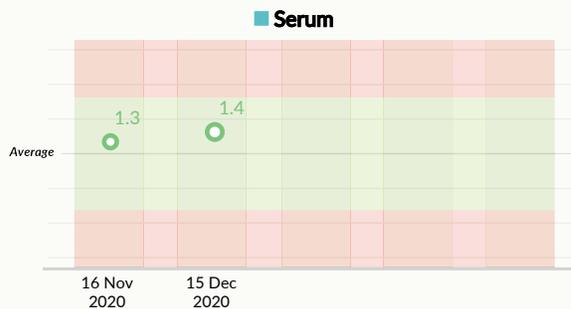


Copper

Current Result:

1.4
(mcg/mL)

Ref Range:
0.6~1.8
(mcg/mL)

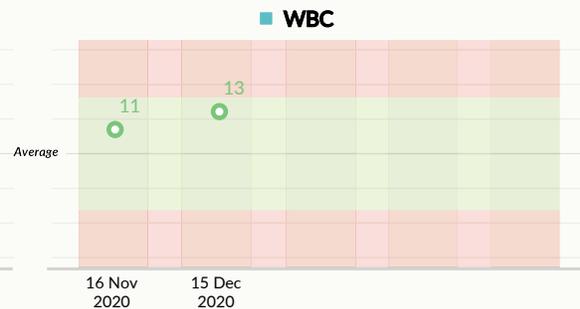


WBC

Current Result:

13
(ng/MM WBC)

Ref Range:
2~15
(ng/MM WBC)



Chromium

Current Result:

0.20
(ng/mL)

Ref Range:
0.10~0.70
(ng/mL)

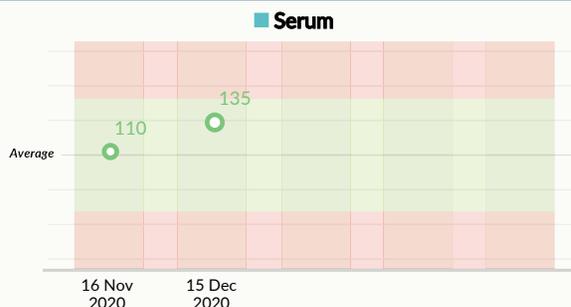


Iron

Current Result:

135
(ug/dL)

Ref Range:
59~158
(ug/dL)

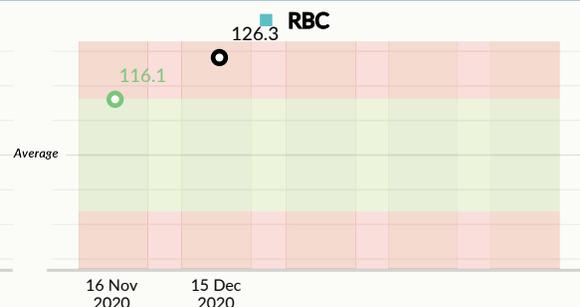


RBC

Current Result:

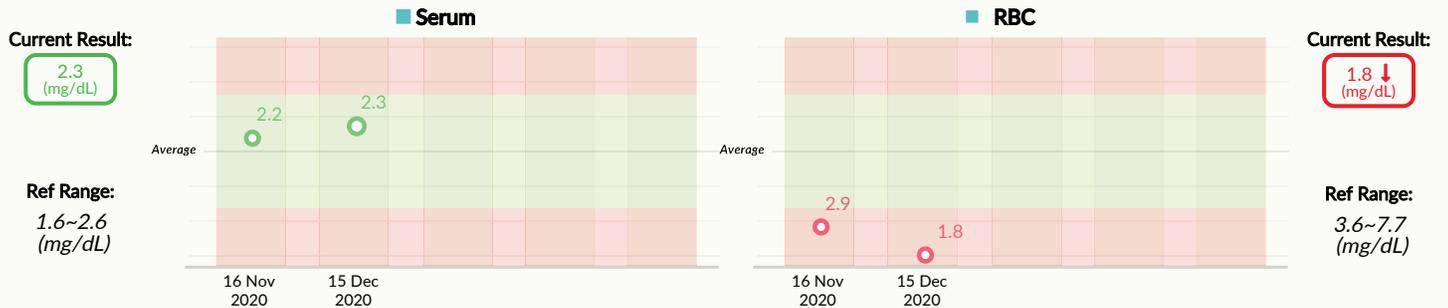
126.3 ↑
(mg/dL)

Ref Range:
88.9~117.0
(mg/dL)

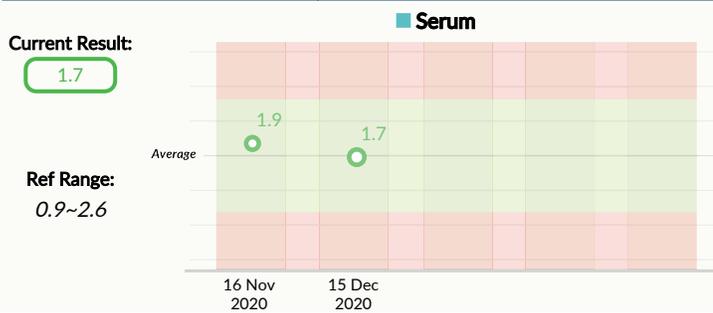


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Magnesium



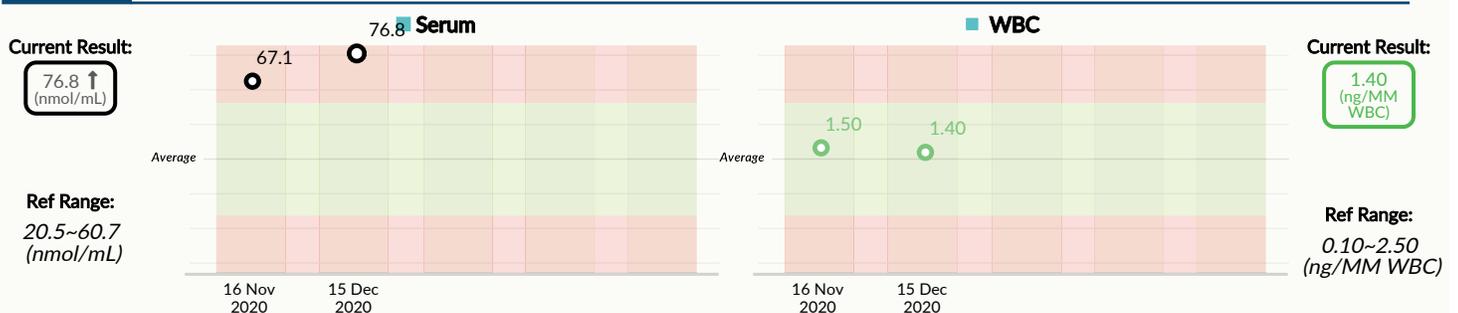
Copper to Zinc Ratio



Choline



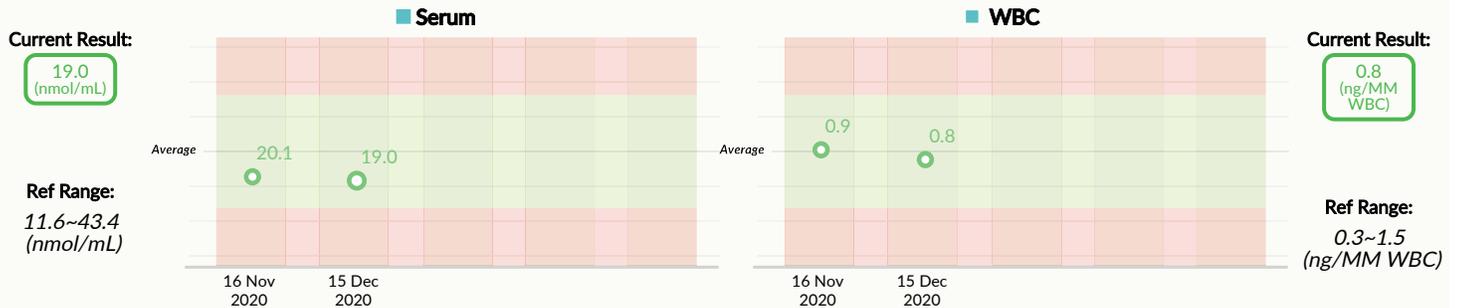
Inositol



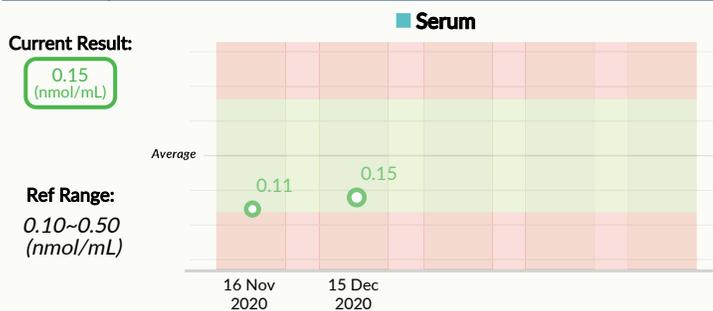


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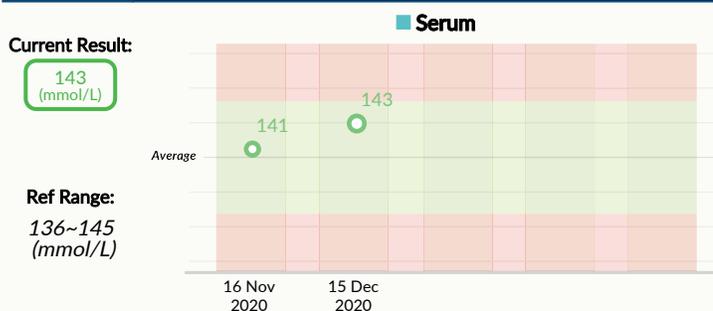
Carnitine



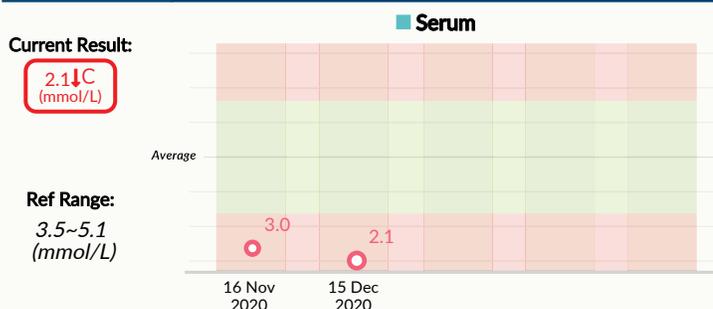
MMA



Sodium

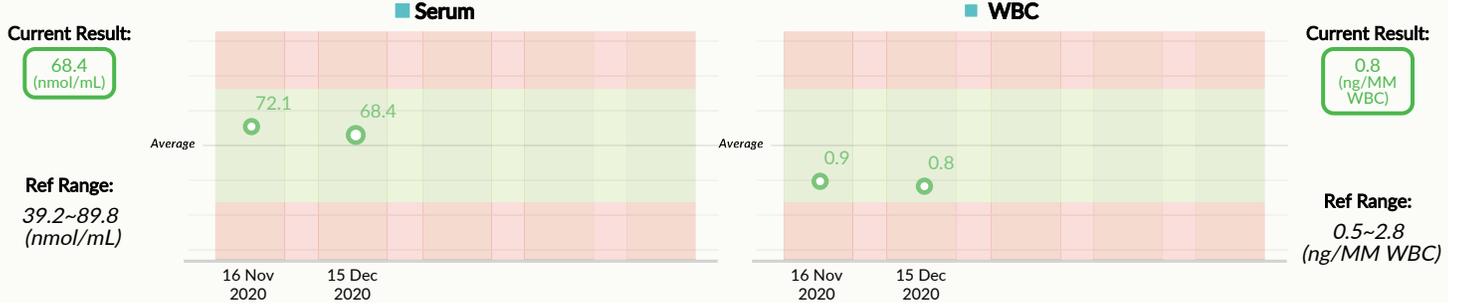


Potassium

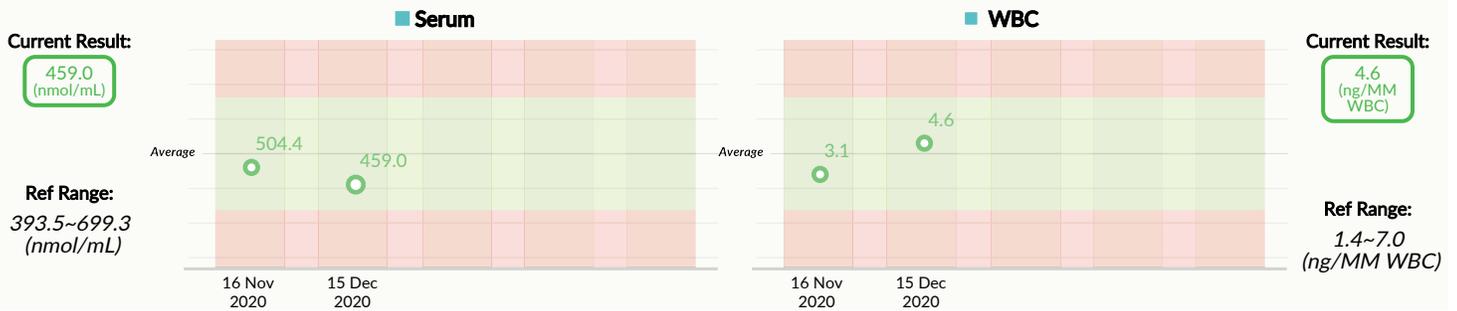


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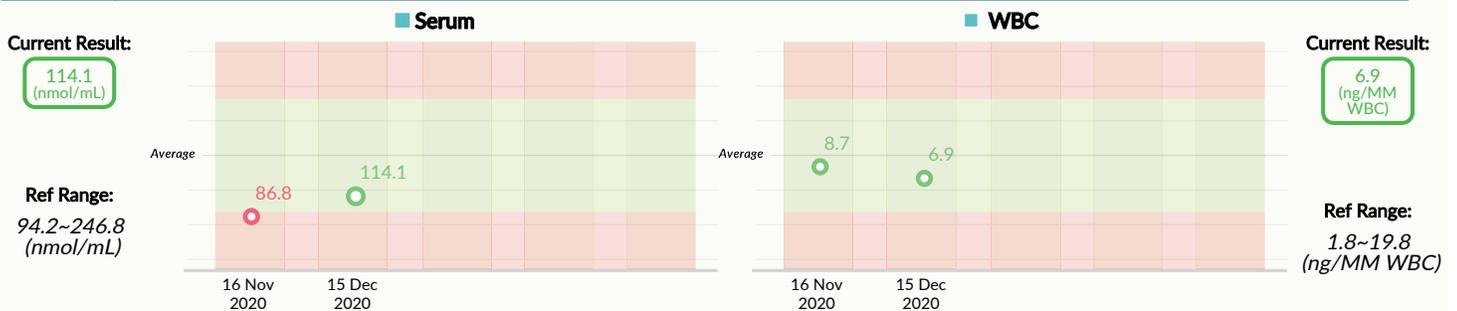
Asparagine



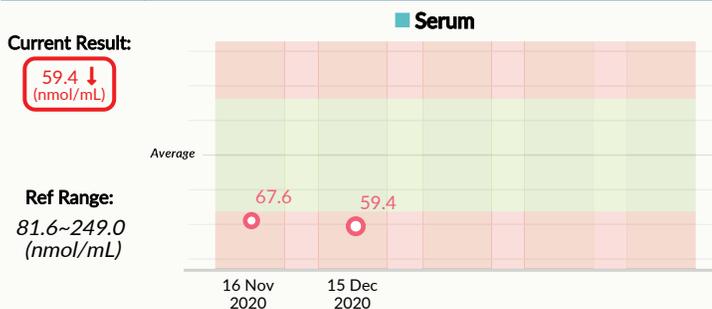
Glutamine



Serine

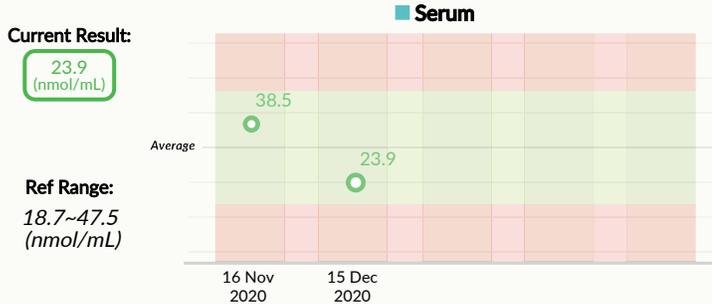


Arginine

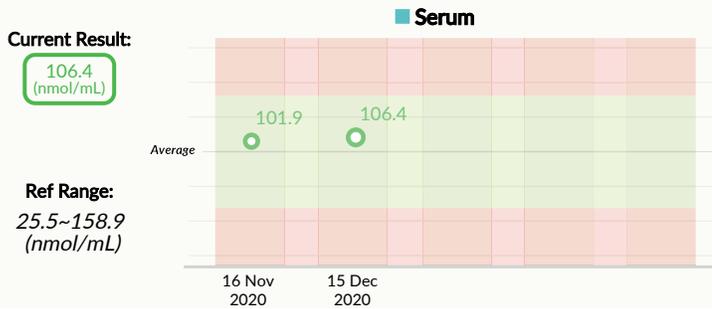


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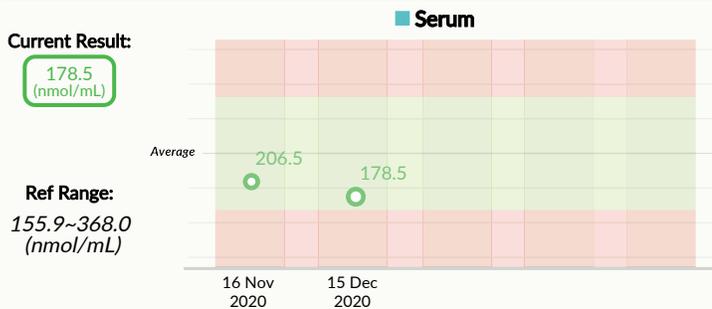
Citrulline



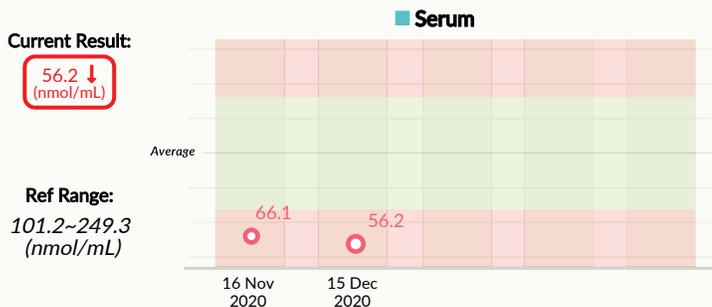
Isoleucine



Valine



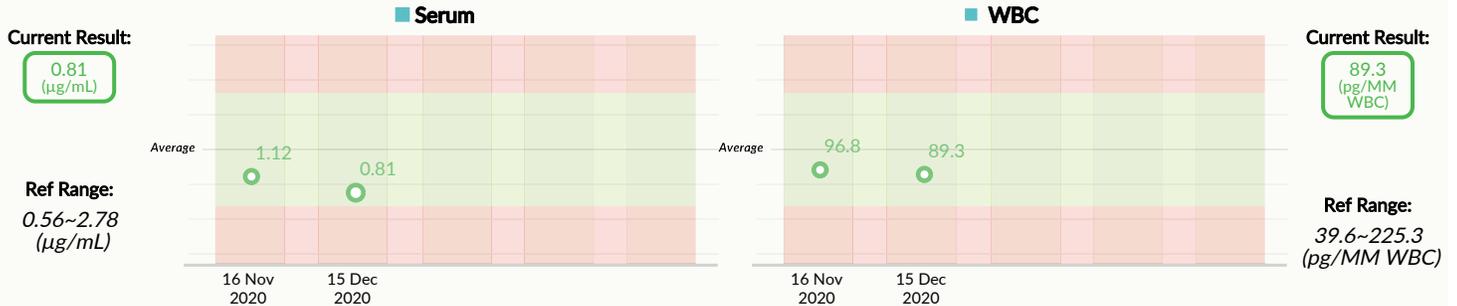
Leucine



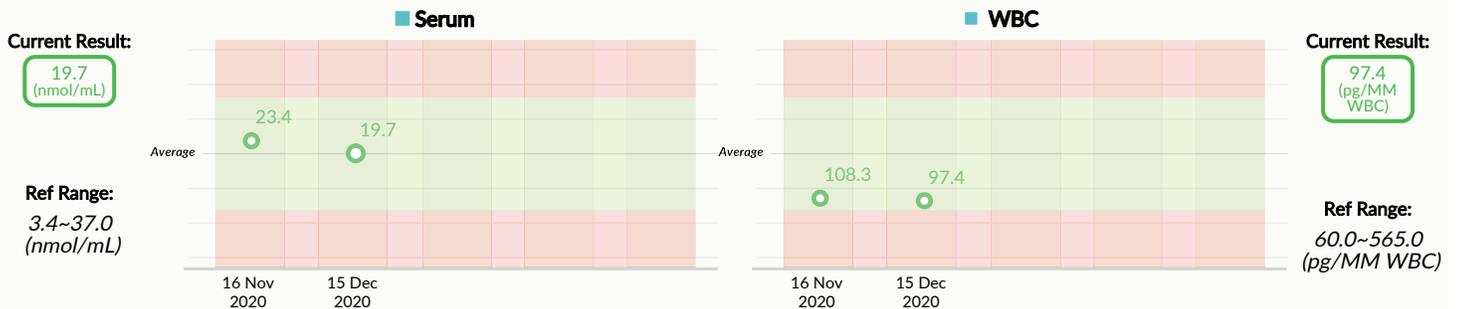


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Coenzyme Q10



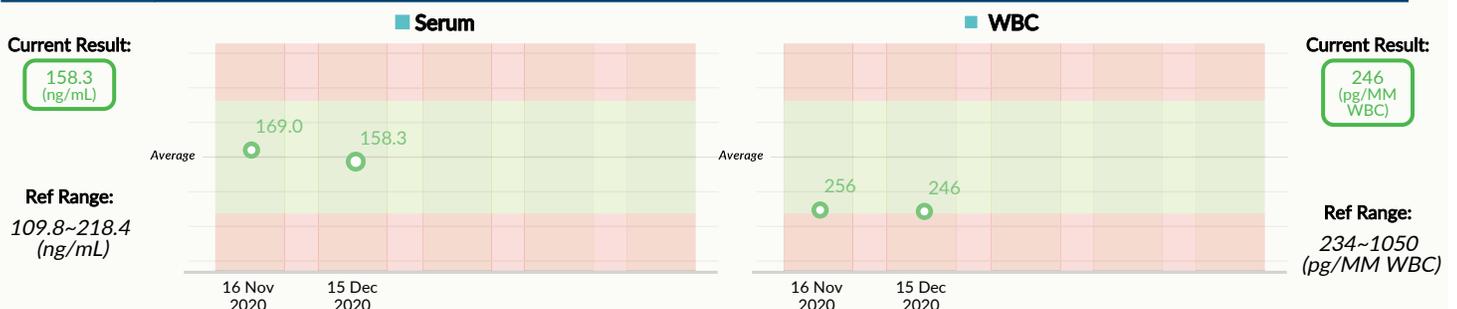
Cysteine



Glutathione



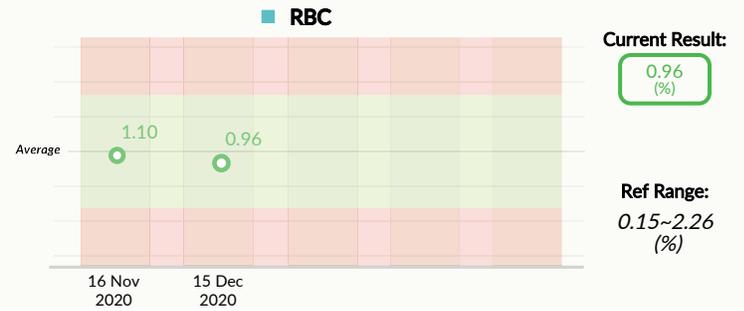
Selenium



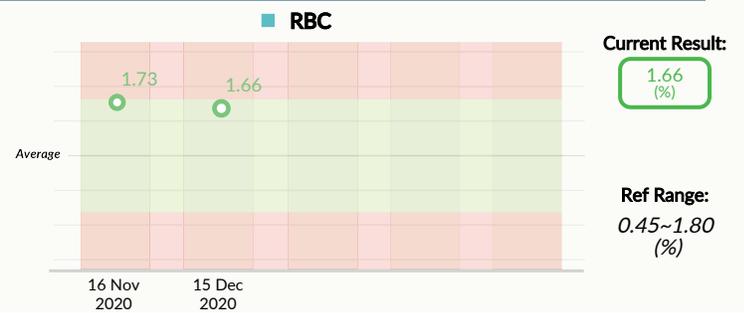


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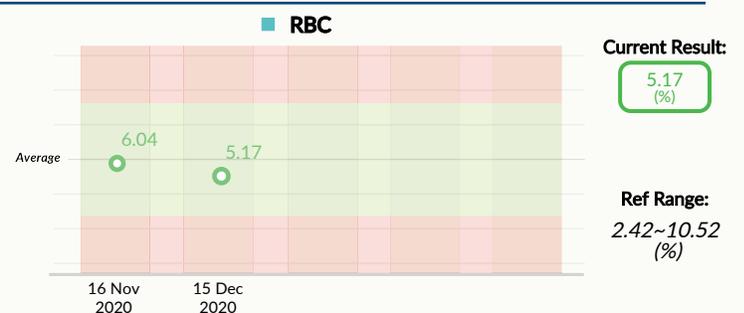
EPA



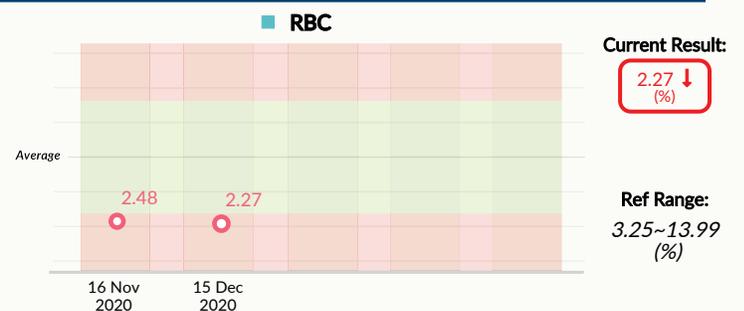
DPA



DHA



Total Omega-3



MICRONUTRIENT

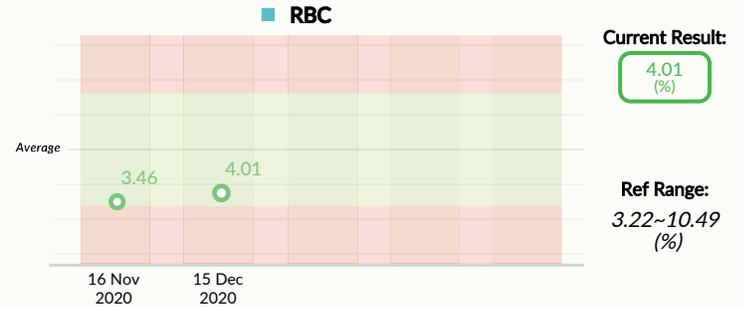


VibrantAmerica

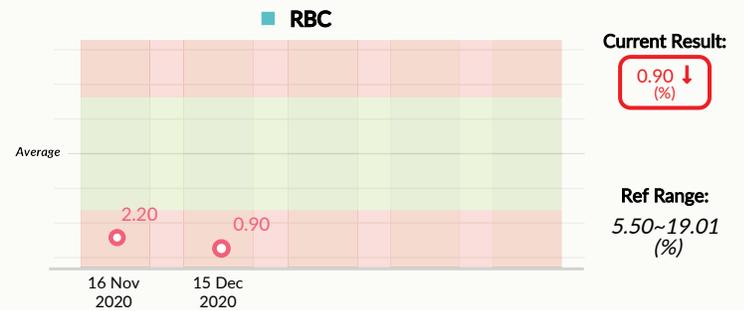
Vibrant America | 1021 Howard Ave, Ste B, San Carlos, CA 94070
1(866) 364-0963 | support@vibrant-america.com | www.vibrant-america.com

LAST NAME	FIRST NAME	GENDER	DATE OF BIRTH	ACCESSION ID	DATE OF SERVICE
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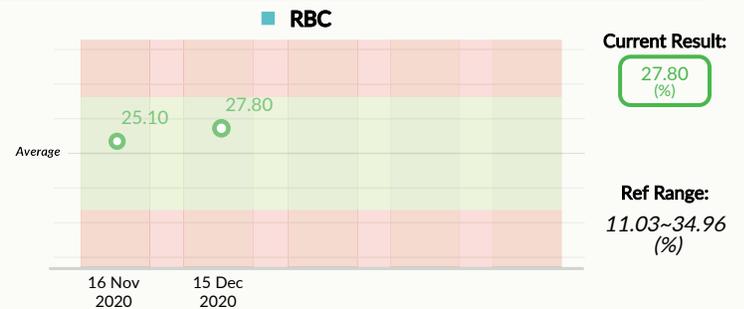
LA



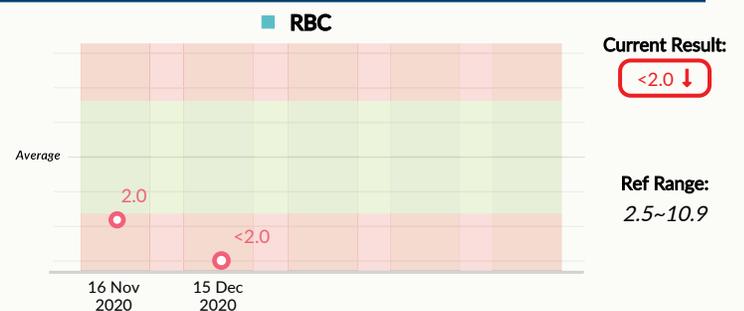
AA



Total Omega-6

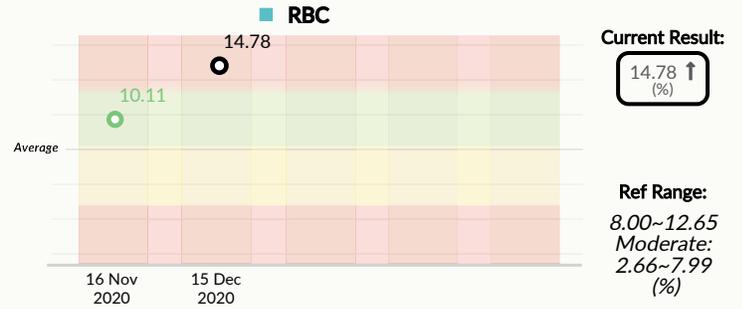


AA/EPA Ratio



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Omega-3 Index



Labnotes

Omega-3 Index RBC :- Omega-3 Index is the sum of EPA % and DHA % as measured in red blood cells, and derived by validated calculations to yield the equivalent sum of EPA % and DHA % in red blood cell membranes. Please note this value is a percentage, with the denominator being the sum of all Fatty Acids measured in the red blood cells and thus the index can vary based on fatty acid composition of the diet.

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VITAMIN B1

Physiological Function

Vitamin B1 aids in energy transformation and production of ATP. It acts as a coenzyme in the breakdown of carbohydrates, fats and proteins to produce energy.

How it gets depleted

Thiamin can become depleted or deficient from frequent consumption of thiaminases present in higher amounts in raw fish and tannins/tannic acid (tea and coffee).

Thiamin is vulnerable to loss during cooking. Can be depleted with excessive or chronic alcohol intake. There may be higher risk of depletion with gastric bypass surgery.



Clinical Manifestations of Depletion

Thiamin deficiency can lead to nervous system and cardiac abnormalities.

The most severe form of thiamin deficiency is called beri beri, a condition commonly resulting in weakness, fatigue, confusion, irritability, weight loss, muscle wasting, and peripheral neuropathy.

Food Sources

Food sources of thiamin include: pork, organ meats, legumes, sweet potato, brown rice, brewer's yeast, pine nuts, sunflower seeds, enriched grains*

Supplement Options

- The RDA for thiamin is 1.0 mg/day for females and 1.2 mg/day for males.
- The RDA for thiamin during pregnancy/lactation is 1.4 mg/day.
- Therapeutic intake of thiamin is commonly 25-100mg/day.
- No UL for thiamin has been set.
- Thiamin is commonly found in multi-B vitamin products.

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VITAMIN B2

Physiological Function

Two very important coenzymes involved in energy metabolism are derived from riboflavin to participate in oxidation/reduction reactions.

Riboflavin is also essential for NOS enzyme (nitric oxide synthase) and glutathione reductase which regenerates glutathione, and which is very important for antioxidation/detoxification.

How it gets depleted

Riboflavin is commonly depleted by excessive or chronic alcohol consumption. Need for riboflavin is increased in the elderly.



Clinical Manifestations of Depletion

Frank deficiency of riboflavin is rare, however, marginal deficiency is common.

Deficiency of riboflavin is associated with fatigue/weakness.

Food Sources

Food sources high in riboflavin include: organ meats, dairy foods, eggs, leafy greens (spinach), broccoli, and liver.

*Enriched grains include riboflavin

Supplement Options

- The RDA for riboflavin is 1.7 mg/day.
- Common levels of therapeutic intake of riboflavin are 25-50 mg/day.
- No UL for riboflavin has been set.

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VITAMIN B12

Physiological Function

Vitamin B12 is an important coenzyme when in its active form of methylcobalamin.

B12 facilitates the metabolism of folic acid through its primary role as a methyl donor.

B12 requires intrinsic factor for absorption, which is calcium dependent.

The role of vitamin B12 in the production of some neurotransmitters may also be evidenced by mood imbalance in susceptible individuals.

How it gets depleted

Age is a risk factor for deficiency of B12 due to a natural decline in intrinsic factor. Chronic use of PPIs may reduce HCl and lead to sub-clinical deficiencies.

Some genetic SNPs (such as MTHFR) may lead to deficiencies in active B12 (methylcobalamin).



Clinical Manifestations of Depletion

- Deficiency of B12 can appear as pernicious anemia, usually due to lack of intrinsic factor.
- Another form of anemia associated with B12 deficiency is megaloblastic anemia, when folate is in excess and insufficient B12 is present, which creates a 'folate trap.'
- Another symptom of B12 deficiency is dementia due to degeneration of myelin.
- In B12 deficiency, methylmalonyl CoA will be metabolized to methylmalonic acid (MMA), which is why MMA is considered the definitive marker for B12 deficiency.
- Achlorhydria (insufficient stomach acid) can lead to B12 deficiency because HCl is required to cleave B12 from intrinsic factor.

Food Sources

Vitamin B12 is synthesized by bacteria and exists in all animal foods.

Vitamin B12 is only available from animal sources.

The B12 synthesized by gut bacteria may not be a significant source for humans, as it is not absorbed in the colon.

Supplement Options

- The RDA for B12 is 6 mcg/day.
- Consider the upper limit of folate supplementation as a factor for the supplementation of B12, due to potential for folate trap.
- Vitamin B12 is extremely safe. No toxicity from high doses of vitamin B12 has ever been reported.
- Intramuscular injections are often used, particularly in the elderly to bypass intrinsic factor.
- Humans store large amounts of B12 in the liver so larger doses can be given at 6 month intervals.
- Supplementation is highly encouraged on a vegan diet. Due to high storage capacity in the liver, it may take years to deplete the body of B12 after adopting a vegan diet.
- Consider MTHFR genetic, and methyl cobalamin supplementation, particularly with hyperhomocysteinemia.
- Methylcobalamin is the recommended form of supplementation, but may be poorly absorbed in people taking antacids or those with very poor absorption (celiac, intestinal permeability, etc).
- Cyanocobalamin is not recommended for patients with MTHFR mutations.
- Hydroxocobalamin is recommended for patients with autoimmune diseases and elevated nitric oxide levels.
- Glutathione is also required for methylcobalamin to be bound for transport adequately.
- Vitamin B12 supplementation may help manage anemia, asthma, fatigue, hepatitis, dementia, epilepsy, depression, psychosis, irritability, ataxia, numbness, tingling, neuropathy, AIDS, multiple sclerosis, tinnitus, and infertility.
- Supplemental B12 is commonly given in 1000 to 5000 mcg doses.

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VITAMIN D, 25-OH

Physiological Function

25-hydroxyvitamin D is a standard lab test which measures the inactive precursor to 1,25-OHD, which is a combination of two forms of vitamin D in the body: vitamin D2 and vitamin D3.

25-OHD has a longer half-life in the blood than 1,25-OHD, and, therefore, levels may differ from levels of active 1,25-OHD3.

Because 25-OHD is a precursor to active forms of vitamin D, it is important to note that it is not reflective of overall active D3 levels, but rather what is available for conversion if cofactors are sufficient.

The conversion of 25-OHD to 1,25-OHD is performed in the kidneys and regulated by parathyroid hormone (PTH). When blood calcium levels fall, PTH signals the kidneys to convert more 25-OHD to 1,25-OHD, which increases intestinal absorption of calcium, and reduces bone demineralization of calcium.

Upon conversion to 1,25-OHD, it also regulates the function of hundreds of genes, supports the immune system, supports production and function of endocrine hormones, is important for normal growth and development of bones and teeth, tightly regulates the levels of calcium and phosphorus being absorbed intestinally as well as released from bone, regulates cell differentiation and growth, and may play an important role in regulating mood.

Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer from unregulated conversion of 25-OH-VitD to 1,25-OHD.

How it gets depleted

Vitamin D deficiency is very common in the U.S.

The most common reasons for vitamin D deficiency include: lack of sun exposure and regular use of sunscreen. Individuals with darker pigmented skin are at greater risk for vitamin D deficiency.

Chronic liver disease and kidney failure are risk factors for vitamin D deficiency.

Patients who present with hypercalcemia, hyperphosphatemia, and low PTH may suffer from unregulated conversion of 25-OH-VitD to 1,25-OHD.

Some medications can deplete vitamin D: anti-inflammatory medications, antibiotics, anticonvulsant medications, cholesterol lowering medications, laxatives and anti-ulcer medications.

Clinical Manifestations of Depletion

Conditions that have been associated with low vitamin D status include: Alzheimer's disease, asthma, autism, cancer, cavities, colds and flus, cystic fibrosis, dementia, depression, diabetes 1 and 2, eczema and psoriasis, hearing loss, heart disease, hypertension, infertility, inflammatory bowel disease, insomnia, macular degeneration, migraines, multiple sclerosis, Crohn's disease, muscle pain, obesity, osteomalacia, osteoporosis, periodontal disease, preeclampsia, rheumatoid arthritis, schizophrenia, seizures, septicemia, and tuberculosis.

Reasons for suboptimal 25-OHD levels, specifically, include lack of sun exposure (particularly in northern latitudes and during the winter season), malabsorption (due to Celiac disease, or other inflammatory digestive disorders), inadequate hepatic vitamin D 25-hydroxylase enzyme activity, and some prescription medications such as antiepileptic drugs, including phenytoin, phenobarbital, and carbamazepine, that increase 25-OHD metabolism.

Levels of PTH may be high-normal or elevated in sub-clinical and frank vitamin D deficiency.

Food Sources

Food sources of vitamin D include: dairy products, such as fortified milk and yogurt, fortified orange juice, egg yolks, liver, fatty fish, such as salmon, tuna, mackerel, sardines, shrimp, mushrooms grown in adequate sunlight, baker's yeast.

Naturally occurring sources will contain vitamin D3, whereas fortified sources (baker's yeast) will contain D2.

Supplement Options

- The previously established RDA of 400IU/day has been found to be insufficient for therapeutic needs. Common doses are used between 1000 and 10,000 IU/day.
- Vitamin D comes in two forms: D2 (ergocalciferol) and D3 (cholecalciferol); both forms can be converted to active vitamin D in the body (25-hydroxyvitamin D).
- Vitamin D is produced when skin is exposed to ultraviolet light from the sun.
- Supplementation with Vitamin D is almost always necessary, as it is extremely difficult to meet needs through diet and sun exposure alone. Consult with your practitioner for supplement recommendations and target goal for serum levels.
- Because vitamin D can be stored or trapped in adipose tissue (fat cells) obese individuals and pregnant women have higher vitamin D requirements.
- Obtaining too much vitamin D from sun exposure is not possible, but it is possible to obtain too much from supplementation.
- Taking too much vitamin D in supplement form can also cause an increase in blood levels of calcium, or hypercalcemia, due to increased intestinal absorption of calcium when serum vitamin D levels are high.
- Vitamin D toxicity has been observed in individuals taking greater than 50,000 IU/day, but intake levels less than 10,000 IU/day are unlikely to cause toxicity.



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VITAMIN K1

Physiological Function

Vitamin K is a group of fat-soluble vitamins. This group of vitamins includes two natural vitamins: vitamin K1 and vitamin K2. These Vitamins are structurally similar and their name comes from the German word “klotting”.

Vitamin K1, is also known as phyloquinone.

Vitamin K assists with blood clotting, supports the formation of bone and bone matrix, and aids in glucose to glycogen conversion for storage in the liver.

How it gets depleted

Dietary deficiency of vitamin K is extremely rare unless there has been significant damage to the intestinal lining, such as in inflammatory bowel disorders (Crohn’s, ulcerative colitis, etc), liver disease, cystic fibrosis, and fat malabsorption disorders.

Taking broad-spectrum antibiotics can reduce vitamin K production in the gut.

Individuals with chronic kidney disease are at risk for vitamin K deficiency. Individuals with ApoE4 genotype may be at greater risk for low vitamin K.

Since Vitamin K is a fat soluble vitamin, following a chronically low-fat diet can inhibit absorption.



Clinical Manifestations of Depletion

Symptoms of vitamin K depletion or deficiency include: excessive bleeding, menorrhagia, bruises that form easily, or appearance of ruptured capillaries.

Food Sources

The best sources of Vitamin K1 are plant foods, especially dark green leafy vegetables.

Note: the absorption of vitamin K1 from food is extremely low. Only 10 percent of the vitamin K, which is found in green leafy vegetables, is absorbed in your body. There’s no variable or modification of the consumption that will significantly increase the absorption

Supplement Options

The AI for vitamin K is set at 90 µg/day for women and 120 µg/day for men.

Individuals who are on certain anti-clotting medications should consult with their medical provider about their dietary vitamin K intake.

Individuals suffering from blood clotting disorders, osteoporosis, coronary artery disease, cancer, liver disease, celiac disease, Crohn’s disease, ulcerative colitis or cystic fibrosis should discuss dietary intake of vitamin K with their healthcare provider.

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VITAMIN K2

Physiological Function

Vitamin K is a group of fat-soluble vitamins. This group of vitamins includes two natural vitamins: vitamin K1 and vitamin K2.

Vitamin K2 is the main storage form of Vitamin K in animals. It has several forms, referred to as menaquinones.

The nomenclature denoting vitamin K2 types will include an 'MK' to specify this is a menaquinone and the number following this denotes how many isoprenyl units are on the side chain of the molecule. The most common forms are MK-4 and MK-7.

Bacteria in the colon can convert K1 (from plant-based foods) into vitamin K2.

Vitamin K2 is necessary to prevent arterial calcification, which it does by activating matrix GLA protein (MGP). This matrix GLA protein is present in blood vessels and inhibits soft tissue calcification. Matrix GLA protein needs to be carboxylated to work properly and Vitamin K2-MK7 plays a major role in this carboxylation.

How it gets depleted

Dietary deficiency of vitamin K1 is extremely rare unless there has been significant damage to the intestinal lining, such as in inflammatory bowel disorders (Crohn's, ulcerative colitis, etc), liver disease, cystic fibrosis, and fat malabsorption disorders.

In addition, the use of oral blood-thinning medications and some antibiotics can interfere with vitamin K.

Individuals with chronic kidney disease are at risk for vitamin K deficiency. Individuals with ApoE4 genotype may be at greater risk for low vitamin K.

Since Vitamin K is a fat soluble vitamin, following a chronically low-fat diet can inhibit absorption.

Clinical Manifestations of Depletion

Inadequate levels of both Vitamin K1 and K2 will radically increase risk for heart disease and stroke.

Chronically low vitamin K levels can lead to uncontrolled bleeding and chronic marginally low vitamin K levels are correlated in some studies with osteoporosis.

Because vitamin K2 also assists in calcium homeostasis, low or deficient levels of vitamin K2 can lead to unregulated calcium release from bone tissue sources in the presence of vitamin D3 supplementation. Supplementation of vitamin D2 does not tend to lead to this, however. It is recommended that vitamin K2 be supplemented when vitamin D3 is supplemented.

Levels of K2 are inversely related to cardiovascular disease and coronary calcification.

Food Sources

The best sources of vitamin K2 include some fermented foods predominantly natto and some rare fermented cheeses, and liver. There are minor amounts present in egg yolk and butter.

Supplement Options

Studies suggest daily therapeutic doses of about 360-500 micrograms (mcg) of vitamin K2

Fermented foods contain a wide variety of different bacteria, and only certain ones—such as *Bacillus subtilis*—actually make vitamin K2. Dietary vitamin K2 intake is enhanced with regular consumption of fermented foods. You can make fermented foods yourself, by using a starter culture specifically designed to optimize K2.

Vitamin K2 supplements come in 'MK' varieties and MK-4 is what all forms of vitamin K2 are converted in vivo. If one takes an MK-7 variety, the body will convert to MK-4, however, MK-4 supplements can be found commercially to bypass activation after absorption



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CALCIUM

Clinical Manifestations of Excess/ Risk for Toxicity

Symptoms and conditions that are associated with excess calcium include: Calcification of soft tissues (including heart and arteries); parathyroid disorders; kidney stones.

Causes of excess calcium in the blood include: low levels of PTH; high or excess intake of vitamin D2 or D3 supplements (unlikely with D2, however); hyperparathyroidism; reduced conversion of 25-OHD to 1,25-OHD in the kidneys; renal failure; parathyroid cancer.

Caution with excess calcium Supplements:
Calcium supplements may cause an excess of calcium in the blood if one has parathyroid dysfunction or renal failure. It is not recommended to take calcium supplements if those conditions exist, unless under the direction of a doctor.

Calcium supplementation should almost always be accompanied with supplementation of Vitamin D and possibly Vitamin K2 to ensure calcium is assimilated into bone and not ectopically deposited into soft tissue.

Physiological Function

Calcium is a mineral that is a major component of bones and teeth, is required for muscle contraction, nerve transmission, cellular metabolism, and aids in blood clotting.

How it gets depleted

Calcium stores in the blood are not depleted metabolically, however, calcium stores elsewhere in the body may become depleted, conditionally, due to increased demand.

Low dietary intake of calcium during times of growth or stress may result in low stores of calcium. Evaluate vitamin D and magnesium levels alongside calcium status.

Iron supplementation may interfere with calcium absorption, and it is recommended to take iron supplements at least 2 hours apart from a meal containing calcium-rich foods.

Clinical Manifestations of Depletion

A deficiency of calcium causes osteoporosis. Some research connects low calcium intake to increased risks of high blood pressure, colon cancer and preeclampsia (high blood pressure and excess protein in the urine of a woman more than 20 weeks pregnant).

Food Sources

Good sources of calcium are: dairy foods, salmon, turnip greens, *Chinese cabbage, kale, bok choy and broccoli. Sardines and other canned fish with bones are additional sources. Some foods such as orange juice and bread are fortified with calcium.

*Chinese cabbage, kale and turnip greens contain absorbable calcium. Spinach and some other vegetables contain calcium that is poorly absorbed.

Supplement Options

- The AI for adults aged 19 to 50 is 1000 mg/day. Because calcium is so critical to preventing bone disease later in life, the AI is higher for adolescents.
- The AI for males and females aged nine to 18 is 1300 mg/day. For those aged 51 and older, the AI is 1200mg/day.
- The UL for calcium is 2,500 milligrams. Excess calcium may cause mineral imbalances because it interferes with the absorption of iron, magnesium, zinc and other minerals.
- Forms of calcium supplementation available include calcium carbonate, calcium citrate, calcium citrate malate, calcium gluconate, and calcium lactate.
- Calcium citrate is the preferred form of calcium for individuals with hypo- or achlorhydria (low or insufficient stomach acid).
- In order to maximize absorption of calcium supplements, limit doses to no more than 500mg/dose.
- Supplementation of calcium should be accompanied by concurrent adequate vitamin D supplementation due to insufficient vitamin D levels impairing cellular calcium absorption, which can lead to atopic calcium deposits in epidermal tissue.
- Iron supplementation may interfere with calcium absorption, and it is recommended to take iron supplements at least 2 hours apart from a meal containing calcium-rich foods.

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MANGANESE

Physiological Function

Manganese is important in many enzyme-mediated chemical reactions including enzymes involved in antioxidant actions in mitochondria and enzymes involved in the synthesis of cartilage in skin and bone.

Manganese also activates enzymes that participate in metabolism of carbohydrates, amino acids, and cholesterol.

In addition, enzymes that incorporate manganese convert the neuro-excitatory glutamate to glutamine.

How it gets depleted

- Iron supplementation may decrease absorption of dietary manganese.
- Intestinal absorption of manganese is reduced when iron stores (ferritin levels) are higher, and tends to be lower in men than women.
- Magnesium supplementation has been shown to decrease manganese levels through reduced intestinal absorption or increased urinary excretion.



Clinical Manifestations of Depletion

- Manganese deficiency is rare.
- Symptoms of manganese deficiency are impaired growth, particularly skeletal abnormalities, and possibly glucose tolerance abnormalities.
- Toxicity is also uncommon and is most frequently the result of exposure to airborne manganese dust.
- Symptoms of toxicity include multiple neurological problems that resemble Parkinson's disease. In children, exposure to elevated levels of manganese in drinking water has been associated with increased rates of attention deficit hyperactivity disorder, cognitive decline, and behavioral problems.
- Individuals with liver failure are at risk for manganese toxicity-associated neurological symptoms.

Food Sources

Tea and coffee are significant sources of manganese in the American diet. Additional sources are nuts, whole grains, legumes and some fruits and vegetables, such as leafy greens.

Supplement Options

- The AI for manganese is 1.8 mg/day.
- The UL for manganese is 11 mg per day.
- Supplementation of manganese is not generally necessary, and may result in toxicity.

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IRON

Physiological Function

- Iron is required for the production of red blood cells (a process known as hematopoiesis), but it's also part of hemoglobin (that is the pigment of the red blood cells) binding to the oxygen and thus facilitating its transport from the lungs via the arteries to all cells throughout the body. Once the oxygen is delivered, the iron (as part of hemoglobin) binds the carbon dioxide which is then transported back to the lung, from where it gets exhaled. Iron is also involved in the conversion of blood sugar to energy.
- The production of enzymes (which play a vital role in the production of new cells, amino acids, hormones and neurotransmitters) also depends on iron, this aspect becomes crucial during the recovery process from illnesses or following strenuous exercise.
- The immune system is dependent on iron for its efficient functioning. Physical and mental growth require sufficient iron levels, particularly important in childhood and pregnancy, where the developing baby solely depends on its mother's iron supplies.

Clinical Manifestations of Excess/ Risk for Toxicity

Iron levels are typically evaluated in conjunction with other iron tests or a full anemia panel. High levels of serum iron can occur as the result of multiple blood transfusions, excessive iron supplementation or injections, lead poisoning, liver or kidney disease. Elevated iron levels can also be due to the genetic disease hemochromatosis-when too much iron accumulates in the body and can damage organs.

High iron levels from dietary or supplementation are more likely in men, and women after menopause because they do not lose iron in blood.

How it gets depleted

Iron is lost by the body through a variety of ways including urination, defecation, sweating, and exfoliating of old skin cells. Bleeding contributes to further loss of iron which is why women have a higher demand for iron than men. If iron stores are low, normal hemoglobin production slows down, which means the transport of oxygen is diminished, resulting in symptoms such as fatigue, dizziness, lowered immunity or reduced ability for athletes to keep up with their training programs. Since our bodies can't produce iron itself, we need to make sure we consume sufficient amounts of iron as part of our daily diet.

Clinical Manifestations of Depletion

Mild iron deficiency can be prevented or corrected by eating iron-rich foods and by cooking in an iron skillet. Because iron is a requirement for most plants and animals, a wide range of foods provide iron. Good sources of dietary iron have heme-iron as this is most easily absorbed and is not inhibited by medication or other dietary components. Two examples are red meat, and poultry.

Non-heme sources do contain iron, though it has reduced bioavailability. Examples are lentils, beans, leafy vegetables, pistachios, tofu, fortified bread, and fortified breakfast cereals. Iron from different foods is absorbed and processed differently by the body; for instance, iron in meat (heme iron source) is more easily absorbed than iron in grains and vegetables (non-heme iron source) but heme/hemoglobin from red meat has effects which may increase the likelihood of colorectal cancer. Minerals and chemicals in one type of food may also inhibit absorption of iron from another type of food eaten at the same time. For example, oxalates and phytic acid form insoluble complexes which bind iron in the gut before it can be absorbed.

Because iron from plant sources is less easily absorbed than the heme bound iron of animal sources, vegetarians and vegans should have a somewhat higher total daily iron intake than those who eat meat, fish or poultry. Legumes and dark-green leafy vegetables like broccoli, kale and oriental greens are especially good sources of iron for vegetarians and vegans. However, spinach and Swiss chard contain oxalates which bind iron making it almost entirely unavailable for absorption. Iron from nonheme sources is more readily absorbed if consumed with foods that contain either heme- bound iron or vitamin C.

Food Sources

Symptoms of iron deficiency can occur even before the condition has progressed to iron deficiency anemia. Symptoms of iron deficiency are not unique to iron deficiency.

Iron is needed for many enzymes to function normally, so a wide range of symptoms may eventually emerge, either as the secondary result of the anemia, or as other primary results of iron deficiency. Symptoms of iron deficiency include: fatigue, dizziness, pallor, hair loss, twitches, irritability, weakness, pica, brittle or grooved nails.

Supplement Options

Frequently used forms of iron in supplements include ferrous and ferric iron salts, such as ferrous sulfate, ferrous gluconate, ferric citrate, and ferric sulfate. Because of its higher solubility, ferrous iron in dietary supplements is more bioavailable than ferric iron. High doses of supplemental iron (45 mg/day or more) may cause gastrointestinal side effects, such as nausea and constipation. Other forms of supplemental iron, such as heme iron polypeptides, carbonyl iron, iron amino-acid chelates, and polysaccharide-iron complexes, might have fewer gastrointestinal side effects than ferrous or ferric salts. Many medicinal herbs can offer iron boosting properties to those who suffer from iron deficiency. These medicinal properties can easily be assimilated into the bloodstream as a hot water infusion (tea). Iron enhancing herbs include yellow dock, red raspberry leaf, gentian, yellow root, turmeric, mullein, nettle, parsley, ginseng, watercress, and dandelion.

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MAGNESIUM

Clinical Manifestations of Excess/ Risk for Toxicity

Too much magnesium from food does not pose a health risk in healthy individuals because the kidneys eliminate excess in the urine. However, high doses of magnesium from dietary supplements or medications often result in diarrhea, nausea and abdominal cramping. Forms of magnesium most commonly causing diarrhea include magnesium carbonate, chloride, gluconate, and oxide. The laxative effects of magnesium salts are due to the osmotic activity of unabsorbed salts in the intestine and colon and the stimulation of gastric motility. ULs for magnesium only apply to supplemental magnesium (1-3 years 65 mg, 4-8 years 110mg, and 9-18 years and adults 350mg).

Very large doses of magnesium-containing laxatives and antacids (typically providing more than 5,000 mg/day magnesium) have been associated with magnesium toxicity, including fatal hypermagnesemia. Symptoms of magnesium toxicity can include hypotension, nausea, vomiting, facial flushing, retention of urine, ileus, depression, and lethargy before progressing to muscle weakness, difficulty breathing, extreme hypotension, irregular heartbeat, and cardiac arrest. The risk of magnesium toxicity increases with impaired renal function or kidney failure because the ability to remove excess magnesium is impaired.

Physiological Function

Important functions of magnesium include: assisting enzymes in more than 300 chemical reactions in the body, supporting cellular activity, participating in muscle contraction, aiding in blood clotting, and as a critical component of bone/skeletal tissue.

How it gets depleted

- Alcohol will lead to increased excretion in urine
- Prolonged use of diuretics will lead to increased urinary excretion.
- Excessive sweating/long bouts of endurance exercise.
- Hyper parathyroidism, Chronic renal failure, malabsorptive conditions (celiac disease, Crohn's disease, partial bowel resection) diabetes (30% show signs of depletion)
- Age is a risk factor for magnesium depletion because intestinal absorption of magnesium declines with age.
- High doses of zinc in supplemental form can interfere with absorption of Magnesium.

Clinical Manifestations of Depletion

- Primary magnesium deficiency is rare.
- Deficiency is usually secondary to another condition.
- Signs and symptoms of deficiency include weakness, heart irregularities, muscle cramps/twitches, insomnia, mental confusion, fatigue, irritability.
- Magnesium deficiency can impede Vitamin D and calcium absorption; increasing risk for bone mineral density disorders. Magnesium depletion is commonly associated with other disease states including both type 1 and type 2 diabetes, hypertension, endothelial dysfunction, asthma, and migraine headaches.

Food Sources

Magnesium is part of chlorophyll so leafy greens are rich in magnesium. Best food sources include: oats, brown rice, spinach, swiss chard, almonds, cashews, hazelnuts, potatoes, bananas, milk, raisins, halibut, avocado, black strap molasses, and chocolate.

Supplement Options

- The UL for magnesium is 350 milligrams from supplements or medicines because it may cause diarrhea. Severe toxicity may cause confusion, loss of kidney function, difficulty breathing and cardiac arrest individuals with kidney disease are at higher risk for magnesium toxicity.
- The use of supraphysiological doses of magnesium can be used therapeutically. Supplemental magnesium is available in several different salts/chelations including: magnesium oxide, magnesium glycinate, magnesium chloride, magnesium citrate, and magnesium threonate. These compounds have different absorption, bioavailability and therapeutic values.
- Magnesium oxide and magnesium citrate are typically recommended for their ability to draw water into the gastrointestinal tract and hitave a laxative effect to produce a bowel movement. Also, it can help alleviate acid- reflux.
- Magnesium citrate has better bioavailability and is typically preferred over magnesium oxide. Citrates have also been shown to bind oxalates and may be the best form for those following a low oxalate diet.
- Magnesium glycinate has good bioavailability and is recommended to help increase magnesium levels without the bowel side effects.
- Magnesium malate is a form of magnesium that has been studied for its positive effects on depression, chronic fatigue, diabetes, and cardiovascular disease
- Magnesium Threonate has recently been studies to cross the blood brain barrier and improve memory and brain function and potentially relieve headaches and migraines. Many studies have shown that supplemental Mg at doses ~400 mg/day reduces blood pressure in mildly hypertensive patients and pregnant women with preeclampsia.

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INOSITOL

Physiological Function

Inositol derivatives are used in the cellular signaling process after the insulin receptor is activated; it is crucial for the development of peripheral nerves, helps move fats out of the liver, promotes the production of lecithin, and is anti-arteriosclerotic, and anti-atherogenic.

How it gets depleted

Inositol can be released from phytate compounds via intestinal bacteria breaking phytate-degrading enzymes (*Lactobacillus plantarum*, *Lactobacillus brevis*, *Lactobacillus curvatus*, *L. gasseri* *B. subtilis* and *Saccharomyces cerevisiae*).

If many courses of antibiotics are used, there may be some depletion of inositol from microbiome conversion.

Inositol is also stored in the liver, spinal cord nerves, and in the brain and cerebral spinal fluid.



Clinical Manifestations of Depletion

- There do not appear to be any clinical manifestations of depletion of inositol. Inositol can be synthesized in the human body from glucose-6-phosphate, a derivative of glucose, therefore, deficiency would be rare.
- Urinary levels of inositol derivatives (D-chiro-inositols and myo-inositols) are seen as a biomarker for insulin resistance.
- Conditions associated with depletion of inositol, however, are depression, anxiety, PCOS, diabetes, CVD, and obesity.

Food Sources

Good dietary sources of inositol include: oranges, cantaloupe, prunes, navy beans, grapefruit, limes, blackberries, kiwis, rutabagas, fresh green beans, unrefined molasses, stone ground wheat, bran flakes, and pumperknickel.

Supplement Options

- There is currently no established RDA, AI, or UL for inositol. Myo-inositol is noted for its benefits to female fertility and insulin sensitivity, and is used often in treatment for PCOS in dosages of 2-4g/day.
- Higher doses of inositol are used to treat psychiatric conditions like depression and anxiety/OCD in much higher doses of 12-18 g/day; some mild gastrointestinal distress is noted with the higher doses and may need to be consumed in split doses.
- Lowering blood glucose can be seen with doses of inositol around 2-4 g/day.
- Currently supplementation of inositol has shown some promise in treating Alzheimer's to reduce progression of fibril formation.
- Inositol may decrease LDL-C and ApoB in persons with metabolic syndrome with doses of 5-10 g/day.
- Doses of inositol of 4 g/day have been associated with improvement of all markers of glycemic control and insulin resistance in gestational diabetes.

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POTASSIUM

Physiological Function

Potassium is one of the main bodily electrolytes (a substance that carries and electrical charge). Potassium helps regulate blood pressure and heart contractions, and is needed for muscle contractions. Also helps control intracellular and extracellular fluid balance in an appropriate ratio with sodium.

How it gets depleted

- Sodium is excreted through the body by any significant loss of body fluid such as diarrhea, vomiting, excessive sweating.
- Magnesium deficiency can contribute to potassium loss and potassium repletion is more difficult if there are inadequate levels of magnesium.
- Diabetes can cause excess potassium loss through urine.
- Excessive alcohol use, excessive laxative use, excessive sweating, diabetic ketoacidosis, folic acid deficiency, and some antibiotic use can cause potassium deficiency. Potassium wasting diuretics are a drug category known to cause potassium depletion.
- In general, losses of body fluid should be considered before dietary intake if potassium levels are low.

Clinical Manifestations of Depletion

- The condition of low blood levels of potassium is called hypokalemia. It can result in hypotension (low blood pressure), muscle weakness, altered heart rate (bradycardia).
- Potassium can also modulate insulin secretion and there is evidence to suggest that "carbohydrate tolerance" is reduced when levels of potassium are low.
- Blood electrolyte levels are not typically regarded as markers of nutritional status. Deviations from normal range are typically not caused by nutritional factors.

Food Sources

Recommended dietary intake for Potassium is 4.7g/day, but the average American intake falls far short of this recommendation.

Food sources rich in Potassium include:
Almonds, artichokes, avocados, banana, bone broth, Brazil nuts, coconut water, dates, dried figs, dulse, flounder, grass-fed beef has 3-5 times more potassium than grain-fed, kelp, legumes, orange juice, parsley, peanuts, pecans, prunes (dried plums), potatoes (sweet), potato skin, raisins, salmon, sardine, sunflower seeds, tomatoes, wheat germ, yams

Potassium can be depleted from food sources through cooking, so raw foods are typically better sources than cooked.

Supplement Options

- Potassium supplements should not be used by anyone with diabetes, insulin resistance, impaired kidney function, or anyone using ACE inhibitors or NSAIDs.
- There is a risk for refeeding syndrome if severe potassium depletion is corrected rapidly with supplementation.

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ARGININE

Physiological Function

L-Arginine is a conditionally essential amino acid found in the diet. It is a dietary supplement used mostly by athletes because it is the amino acid that directly produces nitric oxide via the nitric oxide synthase enzymes.

Arginine helps heal injuries, aids kidneys in removing waste, and boosts immune system function.

How it gets depleted

Arginine is important during periods of illness and chronic conditions like hypertension and type II diabetes, as these states tend to be characterized by an increase in the enzyme that degrades L-arginine (known as arginase) resulting in a transient deficiency; this precedes an increase in blood pressure in these states, and can be partially remedied by an increase in L-arginine intake or resolution of the illness/disease state.



Clinical Manifestations of Depletion

Arginine is one of the three substrates to form creatine which is a vital nutrient (deficiency induces mental retardation) and is also used to form agmatine, a signalling molecule in the body. Arginine is an intermediate in both the urea cycle (with L-ornithine, L-citrulline, and arginosuccinate) and the nitric oxide cycle (with ornithine and arginosuccinate), and vicariously through ornithine it produces polyamine structures which can regulate cellular function. In some individuals with viral infections such as shingles, arginine supplementation may exacerbate symptoms and consultation with a healthcare provider is recommended.

Food Sources

Dietary arginine accounts for 40-60% of serum arginine. Food sources include: turkey, pork, chicken, pumpkin seeds, soybeans, peanuts, spirulina, dairy, chickpeas, lentils.

Supplement Options

- To maintain elevated arginine levels throughout the day, arginine can be taken up to three times a day, with a combined dose total of 15-18g. Note: L-citrulline supplementation is more effective at maintaining elevated arginine levels for long periods of time.
- Taking more than 10g of arginine at once can result in gastrointestinal distress and diarrhea.

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LEUCINE

Physiological Function

Leucine is one of nine essential amino acids in humans (provided by food). Leucine is important for protein synthesis and many metabolic functions. Leucine contributes to regulation of blood-sugar levels, growth and repair of muscle and bone tissue, growth hormone production, and wound healing. Leucine also prevents breakdown of muscle proteins after trauma or severe stress and may be beneficial for individuals with phenylketonuria.

How it gets depleted

Leucine is available in many foods and deficiency is rare.



Clinical Manifestations of Depletion

Leucine supplementation alone exacerbates pellagra and can cause psychosis in pellagra patients by increasing excretion of niacin in the urine. Leucine may lower brain serotonin and dopamine.

Food Sources

Leucine is more highly concentrated in foods than other amino acids. A cup of milk contains 800 mg of leucine and only 500 mg of isoleucine and valine. A cup of wheat germ has about 1.6 g of leucine and 1 g of isoleucine and valine. The ratio evens out in eggs and cheese. One egg and an ounce of most cheeses each contain about 400 mg of leucine and 400 mg of valine and isoleucine. The ratio of leucine to other BCAA is greatest in pork, where leucine is 7 to 8 g and the other BCAA together are only 3-4 grams.

Supplement Options

BCAAs, and particularly leucine, are among the amino acids most essential for muscle health. Supplementation is typically not necessary if total protein intake from a variety of sources is optimal.

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AA

Physiological Function

Arachidonic acid is considered a conditional essential fatty acid and is a structural component of cell membranes-particular cell membranes of the central nervous system (nerve and brain cells).

AA is also a metabolic precursor for proinflammatory signaling molecules (eicosanoid) synthesis.

How it gets depleted

A low AA level with a high or normal LA level likely indicates a delta-6-desaturase deficiency. Activity of this enzyme can be impaired with increased age, alcohol use, certain genetic defects or nutrient deficiency or excess.



Clinical Manifestations of Depletion

Low levels of AA are somewhat rare but can lead to an impairment to cell membrane functions of the central nervous system. Children with attention deficient or hyperactivity disorders have been shown to have low levels. Low levels could also lead to an inappropriate or insufficient immune response or delayed wound healing.

In western cultures, high levels of AA tend to be more problematic as they are associated with many proinflammatory conditions including heart disease, diabetes, arthritis and other autoimmune conditions. High levels of AA stimulate the production of proinflammatory cytokines.

Food Sources

AA can be made endogenously inside the body from the parent compound Linolenic Acid. The rate of conversion is largely dependent on the activity of the delta-6-desaturase.

Supplement Options

It is rarely necessary to supplement with arachidonic acid. If levels are deficient consider linolenic acid levels and factors that could influence delta-6-desaturase enzyme.

To reduce endogenous AA production, reduce dietary intake of vegetable oils high in LA (corn, soy, canola, safflower oil).

Fish oil supplementation or increased intake of EPA fatty acids in the diet can also lower AA.

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OMEGA-3 INDEX

Physiological Function

Omega-3 Index is the sum of EPA % and DHA % as measured in whole blood, and derived by validated calculations to yield the equivalent sum of EPA % and DHA % in red blood cell membranes. Please note this value is a percentage, with the denominator being the sum of all Fatty Acids measured in the blood and thus the index can vary based on fatty acid composition of the diet.

The index can be used as an indicator of risk for sudden cardiac death and nonfatal cardiovascular events and as a therapeutic target. It can also be used to assess adherence to omega-3 therapy and/or success or failure of such therapy. Optimal omega-3 index positively impacts heart rate, blood pressure, triglyceride levels, myocardial efficiency, inflammatory responses, and endothelial function while also improving cognitive function.

How it gets depleted

The Omega-3 Index is a validated biomarker of tissue membrane omega-3 (n-3) polyunsaturated fatty acid (PUFA) status. The ratio is expressed as a percentage where the denominator is the sum of all fatty acids measured in the blood. Thus, a decrease in the ratio can be caused by a low intake of omega-3 fatty acids and incorporation of those fatty acids into cell membranes; or due to a proportionally high intake of other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's poly unsaturated fatty acids)



Clinical Manifestations of Depletion

Low levels of omega-3 index are associated with increased risk for cardiac death.

Food Sources

If omega-3 index is <8.0% it is advised to increase dietary sources of omega-3's (EPA and DHA) from both plant and animal sources. Because the omega-3 index is a relative ratio of omega-3 compared to all other fatty acids in the blood, it is also important to evaluate intake of all other dietary fatty acids (saturated fatty acids, mono-unsaturated fatty acids and omega-6's poly

Supplement Options

- Currently, no official dietary intake recommendations have been established.
- Several official health organizations have proposed a minimum dietary intake level of 500 mg/day of EPA+DHA.
- Because the efficiency of conversion of ALA to DHA is so low, supplementing DHA is generally recommended to meet therapeutic doses.
- The recommended minimum level of DHA supplementation in adults is 250 mg per day.
- Pregnant and lactating women are recommended to consume at least 200 mg DHA per day.
- Diabetic individuals may benefit from supplementing DHA (along with EPA) due to its triglyceride-lowering effects.
- High dose supplementation of omega-3 fatty acids (including DHA) has been shown to reduce the need for non-steroidal anti-inflammatory drugs (NSAIDS).
- Persons suffering from ulcerative colitis have been shown to need fewer corticosteroids when supplementing with high dose omega-3 fatty acids.
- Adverse side effects observed with high dose omega-3 fatty acids from supplement form include gastrointestinal upset and loose stools.
- Omega-3 supplements including EPA and DHA should be used with caution in persons with clotting disorders or on anti-clotting medication.

Key Terms/Glossary

AI

Adequate Intake. A nutrient measure used when RDA cannot be determined due to insufficient data. AIs are approximations of nutrient needs and based on average intake in a healthy population.

Antioxidant

A chemical compound that serves to quench free radicals and other reactive species produced by the process of oxidation, thereby reducing cellular protein damage, as well as inflammation.

Cofactor

A substance that is required for the activity of an enzyme or another protein in a biochemical reaction.

Conditionally Essential

Nutrients that become essential only in certain situations: stress, drug interactions, illness, aging, etc.

Enriched

Refers to refined cereal grains that have had nutrients added back after processing removes the bran and the germ layers. In the United States, enriched grains have the B vitamins (thiamin, riboflavin, niacin, folic acid) and iron added in. Fiber is not added back to enriched grains.

Essential

Refers to a nutrient that is required for life and body function that the body cannot synthesize (produce) on its own. For dietary vitamins, minerals, fatty acids, and amino acids, many, but not all, are essential.

RDA

Recommended Daily Allowance. The estimated amount of a nutrient or calories per day set by the Food and Nutrition Board of the National Research Council. RDA intake level for a particular nutrient that will meet the needs for healthy individuals. RDAs are usually determined for different groups (male, female, children, elderly, pregnant, lactating, etc.) RDAs were originally developed during World War II for soldiers' meal ratios with the intention to prevent frank nutrient deficiencies. They do not take into consideration interactions/depletions from medications or lifestyle factors.

Citations/Sources

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RISK AND LIMITATIONS

This test has been laboratory developed and its performance characteristics determined by Vibrant America LLC, a CLIA and CAP certified laboratory performing the test. The test has not been cleared or approved by the U.S. Food and Drug Administration (FDA). Although FDA does not currently clear or approve laboratory-developed tests in the U.S., certification of the laboratory is required under CLIA to ensure the quality and validity of the tests.

However, laboratory error can occur, which might lead to incorrect results. Some of them may include sample mislabeling or contamination, operational error, or failure to obtain data for certain micronutrients. Vibrant's laboratory may need a second sample to complete the testing. Vibrant America has effective procedures in place to protect against technical and operational problems; however, such problems may still occur. Examples include failure to obtain the result for a specific micronutrient due to circumstances beyond Vibrant's control. Vibrant may re-test a sample in order to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

All supplement and dietary suggestions for specific micronutrients must be evaluated and approved by your provider. Suggested Supplementation is based off references provided at the end of this report. Please see detailed explanation for each micronutrient and follow your ordering providers' recommendation before using this as a therapeutic intake.

A limitation of this testing is that most scientific studies have been performed in Caucasian populations only. The interpretations and recommendations are done in the context of Caucasian studies, but the results may or may not be relevant to tested individuals of different or mixed ethnicities. Please note that pediatric ranges have not been established for these tests. Interference studies have not been established for individuals on immunosuppressive drugs. Based on test results and other medical knowledge of the tested individual, health care providers might consider additional independent testing, or consult another health care provider.