NUTRABALANCE

Personalized Health Profile

Licensed to:
Nutrabalance Corporation
Boulder, Colorado

NAME:	**Hyperparathyroid, Mercury Toxicity, Hepatitis C**
REFERRING DOCTOR:	Nutrabalance/NutraCompute
SEX:	Female
AGE:	41
HEIGHT:	5' 8.5"
WEIGHT:	141 lbs
FILE NAME:	demo9 .nbd

This personalized health profile represents the most advanced technology for determining variance from optimal performance.

REPORT SECTIONS

NUTRABALANCE

Summary of Findings

The following is a physiologic analysis which is designed to be educational to the patient and informational to the doctor. It is not to be used for the diagnosis or treatment of disease conditions.

Nutrabalance is a computerized analysis of blood and urine test data which is highly specific and scientifically based. The system uses sophisticated technology to develop an accurate individual nutritional regimen. The Nutrabalance approach enables precise nutrient recommendations to be made. They are designed to clarify biochemical imbalances allowing your health professional to address the causes and not just the effects of your health issues.

The Nutrabalance approach uses fifteen metabolic classifications to analyze a person's condition, and to monitor his/her progress with therapy. These classifications are based on glandular/organ function. Listed below are the metabolic classifications that have the highest variance from optimal health ranges. They are listed by degree of imbalance.

This Laboratory Analysis Indicates that the Patient Has the Following Metabolic Classifications Out of Balance.	**Percent of test results out of optimal health range.**

Primary: **Gonadal** - Under Active **40%**

This metabolic type is characterized by an imbalance in the hormonal regulation of the gonads which can affect reproduction as well as protein metabolism.

Secondary: **Low Gear** - Over Active **40%**

This metabolic type is characterized by an imbalance in the digestion and assimilation of foodstuffs as well as the detoxification/elimination of metabolic acids (toxins) and/or other unwanted substances. The term Low Gear represents a composite of five glandular/organ designations: Pancreas, Liver, Spleen, Gastrointestinal system and the Parathyroid gland.

Tertiary: **Immune** - Under Active **30%**

This metabolic type is characterized by an imbalance of the immunologic defense mechanisms in response to the accumulation of metabolic toxins (acids). This imbalance may also be due to infectious processes and/or allergic phenomena.

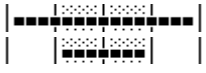
Quaternary: **Adrenal** - Under Active **30%**

This metabolic type is characterized by an imbalance in the sympathetic nervous system as well as the hormonal regulation of electrolytes, acid-alkaline equilibrium and the metabolism of nutrients.

Biochemical: **Acid** **32%**

Acidosis is a physiologic imbalance which is characterized by excessive acid or deficient alkali in the body and/or blood. The term acidosis is not a diagnostic condition and is not meant to represent the absolute measurement of PH in the body, but rather to depict a condition of relative acidosis where the buildup of acidic components has occurred in excess to the alkaline ones.

Basic Lab Tests



Clinical Range
Optimal Range

#	Test	Value 5/31/05	Optimal Range	Clinical Range
1	**GLUCOSE** Blood Sugar Level	83.0	73.5-90.5	65-99
2	**FRUCTOSAMINE** Glycosylation Factor	246.0	210-250	190-270
3	**C-REACTIVE PROTEIN *** Inflammation Marker/Cardiac Risk Factor	0.10	0-0.6	0-0.8
4	**BUN (Nitrogen)** Protein Utilization	11.00	11.5-20.5	7-25
5	**CREATININE** Muscle / Kidney	0.80	0.68-1.02	0.5-1.2
6	**BUN/CREATININE RATIO** Kidney / Body water	13.8	10.8-20.2	6-25
7	**SODIUM** Adrenal / Electrolyte	136	138-143	135-146
8	**POTASSIUM** Heart / Electrolyte	4.6	4-4.8	3.5-5.3
9	**SODIUM/POTASSIUM RATIO** Na/K Retention/ Exchange	29.6	28.8-36.2	25-40
10	**ANION GAP** Anion Concentration	14.6	12-20	8-24
11	**CHLORIDE** Acid-base/ Electrolyte Balance	103.0	101-107	98-110
12	**CO2 (Bicarbonate)** Lung / Acid-Base Indicator	23.0	21.3-27.7	18-31
13	**CHOLESTEROL** Heart / Vascular, Hormone Precursor	180.0	140-180	0-200
14	**TRIGLYCERIDE** Fatty Acid/ Sugar Intake	65.0	60-100	0-150
15	**HDL (GOOD CHOLESTEROL)** Fat Utilization	90.0	48.8-90	35-90
16	**LDL (BAD CHOLESTEROL)** Low Density Lipoprotein, Dietary Factor	85.0	32.5-97.5	0-130
17	**VLDL CALCULATED** Very Low Density Lipoprotein - Calculated	13.0	24-56	8-72
18	**CHOL/HDL RATIO *** Coronary Risk Factor	2.00	0-4.4	0-4.7
19	**HDL/CHOL RATIO** Heart Disease Risk Factor	0.49	0.28-0.52	0.15-0.65
20	**TRIGLYCERIDE/HDL RATIO** Heart Disease Risk Factor	0.59	1.09-3	0.13-3.96
21	**LDL/HDL RATIO *** LDL/Good Cholesterol Ratio *	0.93	0-3.2	0-4
22	**HOMOCYSTEINE (Serum) *** Cardiac Risk Factor	9.7	6.7-9.1	5.4-10.4
23	**CALCIUM** Bone Metabolism	9.70	8.98-9.92	8.5-10.4
24	**PHOSPHOROUS** Bone / Acid-Base Indicator	3.60	3-4	2.5-4.5
25	**CALCIUM / PHOSPHOROUS RATIO** Parathyroid Gland Function Indicator	2.69	2.38-4.12	1.5-5

Indicates out of Clinical range

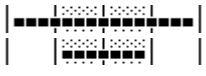
Basic Lab Tests

Clinical Range
Optimal Range

#	Test	Value 5/31/05	Optimal Range	Clinical Range
26	**PTH (Parathyroid Hormone)** Bone Remodeling Metabolism	59.0	23.8-51.2	10-65
27	**ALKALINE PHOSPHATASE** Liver / Bone / Adrenal Indicator	54	46-99	20-125
28	**SGOT (AST)** Amino Acid / Liver Indicator	24	10-27	2-35
29	**SGPT (ALT)** Liver Enzyme-more specific	21	12-30	2-40
30	**GGT (GGTP)** Liver Enzyme-more sensitive	20	17-45	2-60
31	**LDH (LD)** Carbohydrate Utilization	146	138-212	100-250
32	**TOT. BILIRUBIN** Liver Bile/ Spleen Indicator	0.50	0.48-1.02	0.2-1.3
33	**DIR. BILIRUBIN (Conj.)** Liver / Bile Indicator	0.10	0.08-0.22	0-0.3
34	**IND. BILIRUBIN** Spleen / Bile Indicator	0.40	0.4-0.8	0.2-1
35	**URIC ACID** RNA / DNA Metabolism	3.70	3.15-6.05	1.7-7.5
36	**HEPATITIS C ANTIBODY *** Hepatits C Evaluation	60.00	0-0.75	0-1
37	**TOTAL PROTEIN** Serum Protein Level	7.7	6.6-7.7	6-8.3
38	**ALBUMIN** Transport Carrier Protein	4.8	4.4-5.1	4-5.5
39	**CALC/ALB RATIO** Calcium Binding Protein	2.02	1.38-3.12	0.5-4
40	**GLOBULIN** Immune Protein	2.90	2.7-3.7	2.2-4.2
41	**AG RATIO** Blood Viscosity Indicator	1.70	1.1-1.7	0.8-2
42	**THYROID - T3 UPTAKE** Available T3 Receptors	32.60	27.5-32.5	25-35
43	**THYROID - FREE T4 INDEX (T7)** FTI (T7)	2.35	1.89-3.43	1.12-4.2
44	**THYROID - TOTAL T4** Circulating & Bound T4 Hormone	7.20	6.38-10.12	4.5-12
45	**TSH (Highly Sensitive)** Thyroid Stimulating Hormone	0.820	1.675-4.225	0.4-5.5
46	**CORTISOL AM (Serum)** Adrenal Corticoid Steroid Hormone	13.8	8.5-17.5	4-22
47	**DHEA-SULFATE (Serum)** Anti-Aging Sex Steroid Hormone	89.0	143.8-301.2	65-380
48	**CORTISOL / DHEA RATIO (Serum)** Adrenal Stress Index	0.155	0.044-0.121	0.005-0.16
49	**PROGESTERONE (Serum)** Male/ Female Hormone	0.80	8.98-20.32	3.3-26
50	**TOTAL IRON** Oxidation/ Anemia Factor	49.0	70-140	35-175

Indicates out of Clinical range

Basic Lab Tests



Clinical Range
Optimal Range

#	Test	Value 5/31/05	Optimal Range	Clinical Range
51	**TIBC** Total Iron Binding Capacity	375	288-362	250-400
52	**% TRANSFERRIN SAT.** % Transferrin Saturation	13.00	23.75-41.25	15-50
53	**FERRITIN** Iron Tissue Binding Protein	15.0	65.5-176.5	10-232
54	**RBC** Red Blood Cell Count	4.25	4.13-4.77	3.8-5.1
55	**HEMOGLOBIN** Oxygen Carrying Capacity	14.2	12.7-14.5	11.7-15.5
56	**HEMATOCRIT** RBC Concentration	42.30	37.5-42.5	35-45
57	**MCV** Mean Corpuscular Volume	98.4	85-95	80-100
58	**MCH** Mean Corpuscular Hemoglobin	31.0	28.5-31.5	27-33
59	**MCHC** MCH Concentration	34.2	33-35	32-36
60	**RDW** Red Cell Distribution Width	13.3	12-14	11-15
61	**PLATELETS** Blood Clotting Factor	365	205-335	140-400
62	**MPV** Mean Platelet Volume	8.40	8.5-10.5	7.5-11.5
63	**NUCLEATED RBC *** RBC Glycosylation Factor	0.00	0-0.75	0-1
64	**WBC** White Blood Cell Count	6.00	5.55-9.05	3.8-10.8
65	**SEGS/NEUTROPHIL/POLYS %** Acute Immune Response	69.00	51.25-63.75	45-70
66	**ABS. SEGS/NEUTROPHIL/POLY #** Acute Immune Response	4140.00	3075-6225	1500-7800
67	**LYMPHOCYTES %** Chronic Immune Response	27.00	22.5-37.5	15-45
68	**ABSOLUTE LYMPHOCYTE #** Chronic Immune Response	1620.00	1612.5-3137.5	850-3900
69	**BANDS % *** Immune Stimulation	0.00	0-3.75	0-5
70	**ABSOLUTE BANDS #** Immune Stimulation	0.00	0-562.5	0-750
71	**MYELOCYTES %** Immune Stimulation	0.00	0-0.75	0-1
72	**ABSOLUTE MYELOCYTES #** Immune Stimulation	0.00	0-75	0-100
73	**MONOCYTES %** Immune Tissue Response	4.00	3.25-9.75	0-13
74	**ABSOLUTE MONOCYTE #** Immune Tissue Response	240.00	387.5-762.5	200-950
75	**METAMYELOCYTES % *** Immune Stimulation	0.00	0-0.75	0-1

Indicates out of Clinical range

Basic Lab Tests

Clinical Range
Optimal Range

#	Test	Value 5/31/05	Optimal Range	Clinical Range
76	**ABSOLUTE METAMYELOCYTES** * Immune Stimulation	0.00	0-75	0-100
77	**PROMYELOCYTES %** * Immune Stimulation	0.00	0-0.75	0-1
78	**ABSOLUTE PROMYELOCYTES** * Immune Stimulation	0.00	0-75	0-100
79	**BLASTS %** * Immune Stimulation	0.00	0-0.75	0-1
80	**ABSOLUTE BLASTS #** Immune Stimulation	0.00	0-375	0-500
81	**EOSINOPHILS %** Allergic/ Parasitic Factor	0.00	1-3	0-4
82	**ABSOLUTE EOSINOPHIL #** Allergic/ Parasitic Factor	0.00	125-375	0-500
83	**BASOPHILS %** Histamine Containing Cells	0.00	0.5-1.5	0-2
84	**ABSOLUTE BASOPHIL #** Histamine Containing Cells	0.00	50-150	0-200
85	**REACTIVE LYMPHS %** * Atypical (abnormal) Lymphocytes	0.00	0-7.5	0-10
86	**URINE SPECIFIC GRAV.** Urine concentration	1.012	1.01-1.026	1.001-1.035
87	**URINE pH** Acid/Alkaline indicator	5.5	5.8-7.2	5-8
88	**URINE PROTEIN** * Kidney function	0.0	0-0.7	0-1
89	**URINE GLUCOSE** * Diabetic Indicator	0.0	0-0.7	0-1
90	**URINE KETONES** * Fasting/Diabetic Indicator	0.0	0-0.7	0-1
91	**URINE OCCULT BLOOD (HgB)** * Kidney, Bladder, Menses	0.0	0-0.7	0-1
92	**URINE NITRITE** * Ammonia oxidation	0.0	0-0.7	0-1
93	**URINE BILIRUBIN** * Liver/Bile Indicator	0.0	0-0.7	0-1
94	**(U) LEUKOCYTE ESTERASE** * WBC Ester Breakdown	0.0	0-0.7	0-1
95	**URINE WBC** * White Blood Cells	3.0	0-3.7	0-5
96	**(U) SQUAMOUS EPITHEL. CELLS** * Mucosal Erosion	5	0-4	0-5
97	**(U) TRANSITION EPITHEL. CELLS** * Mucosal Erosion	0	0-4	0-5
98	**(U) RENAL EPITHELIAL CELLS** * Mucosal Erosion	0	0-2	0-3
99	**URINE RBC** * Red Blood Cells	3.0	0-3.7	0-5
100	**URINE CASTS #** * Kidney Precipitates	0.0	0-0.7	0-1

Indicates out of Clinical range

Basic Lab Tests

Clinical Range
Optimal Range

#	Test	Value 5/31/05	Optimal Range	Clinical Range
101	**URINE CASTS** * Kidney Precipitates	0.0	0-0.7	0-1
102	**URINE BACTERIA** * Urinary Tract Infection	0.0	0-0.7	0-1
103	**URINE YEAST** * Yeast Discharge	0.0	0-0.7	0-1
104	**URINE CRYSTALS #** * Urine Precipitates	0.0	0-0.7	0-1
105	**URINE CRYSTALS** * Urine Precipitates	0.0	0-0.7	0-1

Indicates out of Clinical range

Tests out of Clinical range

VeryLow Progesterone, **VeryLow % TRNFSat**, **VeryHigh HepatitisCAB**

1) The test results listed above exceed the clinical range and may necessitate further examination and/or testing by a physician or health care professional.

2) Optimal Ranges for laboratory parameters represent a more precise criteria for optimizing one's health state. The ranges have been determined by scientific and clinical analyses and by the expertise of physicians specializing in Nutritional Medicine.

3) The Clinical Ranges used on this report may in some cases slightly deviate from those of your laboratory.

4) An asterisk (*) after the test indicates that this is a zero based test or one with a fixed optimal range.

Symptomatic Profile

High Priority
Low Priority

#	Test	Value 5/31/05	Low Prio Range	High Prio Range
1	**Hypothalmus (IA)**	20	0-10	0-20
2	**Anterior Pituitary (IB)**	13	0-15	0-30
3	**Pineal (IC)**	31	0-10	0-20
4	**General Neurological (ID)**	30	0-10	0-20
5	**Posterior Pituitary (IIA)**	16	0-15	0-30
6	**Kidney (IIB)**	29	0-15	0-30
7	**Thyroid (III)**	25	0-10	0-20
8	**Adrenal (IVA)**	37	0-10	0-20
9	**Heart / Circulation (IVB)**	5	0-8	0-16
10	**Heart / Blood Pressure (IVC)**	9	0-10	0-20
11	**Pre Menstrual Syndrome (VIA)**	26	0-10	0-20
12	**Reproduction (VIB)**	10	0-12	0-24
13	**Dysmennorhea (VIC)**	32	0-10	0-20
14	**Genital Disorders (VID)**	11	0-10	0-20
15	**Menopause (VIE)**	15	0-10	0-20
16	**Bone Integrity (VIIA)**	7	0-10	0-20
17	**Musculoskeletal (VIIB)**	23	0-10	0-20
18	**Connective Tissue (VIIC)**	25	0-10	0-20
19	**Low Blood Sugar (VIIIA)**	18	0-10	0-20
20	**Hi Blood Sugar (VIIIB)**	14	0-10	0-20
21	**Pancreatic Digestion (VIIIC)**	14	0-10	0-20
22	**Digestion (IXA)**	21	0-8	0-16
23	**Colon (IXB)**	25	0-10	0-20
24	**Upper GI (IXC)**	14	0-10	0-20
25	**GI Infection / Inflammation (IXD)**	23	0-15	0-30

Exceeds High Priority range

Symptomatic Profile

High Priority
Low Priority

#	Test	Value 5/31/05	Low Prio Range	High Prio Range
26	**GI Systemic Effects (IXE)**	15	0-10	0-20
27	**Liver (XA)**	34	0-10	0-20
28	**Spleen (XB)**	5	0-15	0-30
29	**Immune (XI)**	58	0-15	0-30
30	**Physical Stress Factors (XII)**	46	0-10	0-20
31	**Exercise Risk Factors (XIII)**	9	0-10	0-20
32	**Motivational Behaviors (XIV)**	26	0-10	0-20
33	**Relationships (XV)**	33	0-10	0-20
34	**Emotional (XVI)**	40	0-10	0-20
35	**Rest & Relaxation (XVII)**	20	0-10	0-20
36	**Mood Effects (XVIII)**	5	0-10	0-20
37	**Adjustment Skills (XIX)**	7	0-10	0-20

Exceeds High Priority range

Categories exceeding High Priority range

VeryHigh Pineal Symptoms, **VeryHigh General Neurological Symptoms**, **VeryHigh Thyroid Symptoms**, **VeryHigh Adrenal Symptoms**, **VeryHigh Musculoskeletal Symptoms**, **VeryHigh Connective Tissue Symptoms**, **VeryHigh Digestion Symptoms**, **VeryHigh Colon Symptoms**, **VeryHigh Liver Symptoms**, **VeryHigh Immune Symptoms**, **VeryHigh Physical Stress Factor**, **VeryHigh Motivational Behaviours**, **VeryHigh Relationship**, **VeryHigh Emotional**, **VeryHigh Pre Menstrual Syndrome Symptoms**, **VeryHigh Dysmennorhea Symptoms**

The symptomatic categories listed above reflect the individual's subjective report of symptoms as they pertain to the metabolic categories of glandular/organ dysfunction. In addition, any significant past history of disease or medical treatment will be included in this section. All of this information is then factored into the diagnostic evaluation which then produces the classifications listed in the Summary of Findings section.

The symptomatic categories, in distinction to the laboratory tests, begin at a zero point and progress upward through an optimal range to a clinical upper limit. If the subjective symptoms pertaining to a metabolic category exceed an acceptable number, the dotted line will penetrate into the optimal range. This then implicates that particular glandular/organ category as a possible source of the symptoms. Scores above the clinical limit indicate more of a severe concern which may require added attention with a focus on symptomatic relief.

The final categories of the symptomatic profile pertain to behavior and psychological issues. Categories relating to exercise, relaxation and physical stress factors point to daily behavior patterns which can affect overall health. Other categories including emotional and mood effects, motivational behaviors, relationships and adjustment skills serve to identify psychological areas of concern which can have an effect on well-being.

Trace and Toxic Blood Tests

Clinical Range
Optimal Range

#	Test	Value 6/12/05	Optimal Range	Clinical Range
1	**BORON (BLOOD)** Trace - Bone Strength	0.0280	0.0298-0.0852	0.002-0.113
2	**CHROMIUM (BLOOD)** Trace - Blood Sugar Metabolism	0.0860	0.08-0.16	0.04-0.2
3	**IODINE (BLOOD)** Trace - Thyroid Gland	0.0390	0.1148-0.3142	0.015-0.414
4	**MAGNESIUM (BLOOD)** Trace - Enzyme Activator	26.6100	30.875-42.625	25-48.5
5	**MANGANESE (BLOOD)** Trace - Tissue/ Bone Strength	0.0210	0.0415-0.1105	0.007-0.145
6	**MOLYBDENUM (BLOOD)** Trace - Bone Strength	0.0020	0.045-0.135	0-0.18
7	**SELENIUM (BLOOD)** Trace - Mineral AntiOxidant	0.2830	0.155-0.385	0.04-0.5
8	**VANADIUM (BLOOD)** Trace - Blood Sugar Metabolism	0.0310	0.0233-0.0497	0.01-0.063
9	**ZINC (BLOOD)** Trace - Pancreas/ Gonadal Factor	5.5500	4.475-6.025	3.7-6.8
10	**ARSENIC (BLOOD)** Trace/Toxic	0.0110	0.0125-0.0375	0-0.05
11	**COBALT (BLOOD)** Trace/Toxic	0.0000	0.0075-0.0225	0-0.03
12	**COPPER (BLOOD)** Immune/ Psychosis Factor	0.6030	0.71-1.17	0.48-1.4
13	**GERMANIUM (BLOOD)** Immune Lymphocyte Stimulation	0.0030	0.0135-0.0405	0-0.054
14	**LITHIUM (BLOOD)** Mood Stabalizer Factor	0.0010	0.0333-0.0997	0-0.133
15	**NICKEL (BLOOD)** Trace/Toxic	0.0040	0.0025-0.0075	0-0.01
16	**SILVER (BLOOD)** Immune Stimulation Factor	0.0000	0.0008-0.0022	0-0.003
17	**STRONTIUM (BLOOD)** Trace/Toxic	0.0190	0.0178-0.0392	0.007-0.05
18	**TIN (BLOOD)** Trace/Toxic	0.0450	0.0425-0.1075	0.01-0.14
19	**ALUMINUM (BLOOD) *** Toxic	0.0400	0-0.2032	0-0.271
20	**ANTIMONY (BLOOD) *** Toxic	0.0000	0-0.003	0-0.004
21	**BARIUM (BLOOD) *** Toxic	0.1240	0-0.135	0-0.18
22	**BERYLLIUM (BLOOD) *** Toxic	0.0000	0-0.003	0-0.004
23	**CADMIUM (BLOOD) *** Toxic	0.0020	0-0.0075	0-0.01
24	**LEAD (BLOOD) *** Toxic	0.0120	0-0.075	0-0.1
25	**MERCURY (BLOOD) *** Toxic	0.0120	0-0.0037	0-0.005

Indicates out of Clinical range

Trace and Toxic Blood Tests

Clinical Range
Optimal Range

#	Test	Value 6/12/05	Optimal Range	Clinical Range
26	**PLATINUM (BLOOD) *** Toxic	0.0000	0-0.0052	0-0.007
27	**THALLIUM (BLOOD) *** Toxic	0.0000	0-0.0037	0-0.005
28	**URANIUM (BLOOD) *** Toxic	0.0000	0-0.0015	0-0.002

Indicates out of Clinical range

Tests out of Clinical range

VeryHigh BMercury

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Dietary Recommendations

Blood Type: O Positive

Acid Food Plan

The following dietary recommendations are general guidelines to proper nutrition for your present metabolic condition. Due to individual biochemical variations, your health care professional may suggest additional or alternative recommendations.

The blood and urine analysis indicates that you have an acid metabolic condition. This evaluation reflects a relative acidosis because the blood is normally maintained in an alkaline state. A person with an acid metabolic condition should emphasize the consumption of alkaline forming foods in his/her diet. Recommended portion, by volume: **60-70% alkaline forming foods; 30-40% acid forming.**
It is important to realize that **some acid formation is necessary** to generate metabolic activity.

Listed below are some of the acid and alkaline forming foods (both healthy and unhealthy variations.) These categories are based on the body's reaction to a moderate amount of each. Excess amounts may produce the opposite effect. **These lists are not intended to provide dietary recommendations**, but to clarify the chemical effect of the food consumed. An actual meal plan, including menus and recipes, can be found further along in this dietary section of the report.

Acid Producing Foods: (from the least potent to the most potent)

grains	pasta	eggs	most chemicals and drugs
beans	fish	dairy	sugar, saccharin, sweet
tofu	fowl	red meat	tobacco, soft drinks, pop
tempeh	nuts	vinegar	most alcoholic beverages

Alkaline Producing Foods: (from the least potent to the most potent)

miso	natural yeast	fruits and fruit juices (quantity dependant)
seeds	sea salt	mineral and soda water
sprouts	soy sauce	natural wine and beer (in moderation)
seaweed; sea vegetables	most spices and herbs	coffee, tea (in moderation)

Alkaline Producing Vegetables:

-leafy green vegetables (watercress, spinach, kale, etc.)
-root vegetables (carrots, turnips, onions etc.)
-ground vegetables (cauliflower, squash, cucumbers etc.)

General Dietary Guidelines: (non-specific, recommended by the Am. Council on Nutrition)

-Increase consumption of fruits and vegetables.
-Decrease consumption of refined and other processed sugars.
-Decrease consumption of foods high in saturated fat (eggs, meat and dairy products.)
-Decrease consumption of butterfat, eggs, and other high-cholesterol sources.
-Carefully watch consumption of salt and foods high in salt content.
-Avoid artificially colored, sprayed, chemically preserved foods.
-Limit consumption of coffee, black tea, or other stimulating beverages.
-Moderate alcohol intake.
-Use natural or whole food products whenever possible.
-Consume between 6-8 glasses of water per day

Blood Type: O Positive

General Blood Type Dietary Guidelines:
ABO Blood Types can be used to recommend dietary guidelines, and these are based on genetic tendencies developed over centuries in time. Since blood types have been shown to correlate to specific metabolic performance criteria, certain food combinations can be more helpful for one type vs. another.

It is important to remember that genetic tendencies, as far as digestion is concerned, are often highly influenced by environmental factors as well as lifestyle patterns. Fluctuations in digestive activity occur on a daily basis and, in addition, chronic impairments develop which become ingrained in our physiology as sort of "bad habits" which we are subjected to on-goingly. Such factors can alter our genetic requirements to some degree.

Specific Blood Type Dietary Guidelines:
Blood Type O, can be referred to as **natural meat eaters** who readily metabolize proteins and fats into ketones, which keep glucose levels steady and energy levels high. Generally this type has a **hearty GI tract,** as long as it has not been compromised by years of faulty living habits and dietary improprieties. Should this occur, a 30 day detoxification program is highly recommended to assist in the restoration process.

The O Type requires an efficient metabolism to stay lean, healthy and energetic. They require **vigorous regular exercise** and intense physical activity to maintain optimal performance. Without this stimulation, O Types fall prey to sluggish metabolisms and toxic buildup, which can lead to poor performance and fatigue. In general, the O Type has an overactive immune system, and is intolerant to dietary and environmental adaptations.

The following are highly **beneficial foods** for optimizing metabolic efficiency of the O Type:
-kale, beat tops, kelp, dulse, sea vegetables
-pumpkin seeds, walnut, carob, ginseng
-cold processed flax seed oil, soy bean oil
-red meat, liver, heart, veal, venison, lamb, beef, fish
-butter, Feta cheese, Farmers cheese, Goat Cheese, Soya cheese, Mozzarella cheese
-Essene bread, Ezekiel bread
-artichoke, horseradish, escarole, dandelion, collard greens, Romaine, okra, chicory, onion, broccoli, pumpkin, red peppers, parsnips, parsley, sweet potato, spinach, Kohlrabi
-figs, prunes, plums, pineapple, papaya, black cherry, apricot

The following are **avoid foods** for optimizing metabolic efficiency of the O Type:
-pork products, shell/scavenger type fish
-hydrogenated oils, heat processed oils, heated oils, pasteurized cheese products
-peanuts, peanut butter, cashews, processed heated nuts
-lentils, beans, wheat, oatmeal, bread, pasta, corn products, cold cereals, pastries
-eggplant, white potato, olives, plantains
-melons, cantaloupe, oranges, strawberry, tangerines
-corn syrup, sugar, honey, maple syrup, sweets
-coffee, black tea, black pepper
-in the event of an overweight condition, detoxification followed by vigorous exercise, smaller meals, leaner cuts of meat and strict adherence to the avoid list is recommended.

For **Rh Positive blood**, follow above recommendations with emphasis on the beneficial list of foods.

HIGH PROTEIN FOR AMERICAN FEMALE

SEVEN DAY MENU PLAN

(* = Recipe to follow menus)

DAY 1 MENU
BREAKFAST:
Scrambled eggs*
LUNCH:
Seafood salad sandwich*
AFTERNOON SNACK:
Cheese slice
DINNER:
Chili*
LATE NIGHT SNACK
Turkey slice

DAY 2 MENU
BREAKFAST:
Old Fashion Oatmeal* and Bacon
LUNCH:
Cheeseburger*
AFTERNOON SNACK:
Tofu Veggie Dip*
DINNER
Barbecued Chicken*
LATE NIGHT SNACK
Cheese & fruit

DAY 3 MENU
BREAKFAST:
Fruit Salad*
LUNCH:
Chef Salad*
AFTERNOON SNACK:
Cottage cheese & pineapple
DINNER:
Foiled Fish*
LATE NIGHT SNACK:
Sliced turkey

DAY 4 MENU
BREAKFAST:
Yogurt & Fruit
LUNCH:
Grilled Chicken Sandwich*
AFTERNOON SNACK:
Cheese & fruit
DINNER:
Pork Medallions*
LATE NIGHT SNACK:
Soft cheese & wine

DAY 5 MENU
BREAKFAST:
French Toast* Sticks
LUNCH:
Chicken Salad Sandwich*
AFTERNOON SNACK:
Turkey Guacamole*
DINNER:
Meatloaf*
LATE NIGHT SNACK:
Sliced turkey

DAY 6 MENU
BREAKFAST:
Skillet Hash*
LUNCH:
BLT Sandwich*
AFTERNOON SNACK:
Cottage cheese & pineapple
DINNER:
Quick Turkey Dinner*
LATE NIGHT SNACK:
Cheese & fruit

DAY 7 MENU
BREAKFAST:
Scrambled Eggs Benedict*
LUNCH:
Turkey in a Pocket*
AFTERNOON SNACK:
Egg whites
DINNER:
Broiled Salmon*
LATE NIGHT SNACK:
Turkey breast sliced.

SEVEN DAY RECIPES*

DAY 1 RECIPES
BREAKFAST:
Scrambled eggs
4 egg whites or 1/2 cup egg mix
1 ounce nonfat cheese, shredded
1 cup grapes
1/2 piece rye toast
2/3 tsp. Olive oil
1/2 tsp. Fresh ground or natural peanut butter
Spray nonstick pan with vegetable spray.
Beat eggs and shredded nonfat cheese with olive oil and add a little milk if desired, then scramble
LUNCH:
Seafood Salad Sandwich
4 1/2 ounces seafood (shrimp, crabmeat or lobster)
1 tbs. light mayonnaise
1/2 mini pita pocket
1 small side salad
1 apple
Mixed seafood with mayonnaise. Stuff into mini pita pocket
AFTERNOON SNACK:
1 ounce low-fat cheese
1/2 orange
DINNER:
Chili
4 1/2 ounces lean ground meat (beef or turkey)
non-fat cheese
sprinkling of shredded nonfat cheese
minced onion, chopped mushrooms and chopped green bell pepper
chili powder, oregano and pepper to taste
1/4 cup kidney beans
1 cup tomatoes crushed
1 tsp. Olive oil
1 peach
Brown meat in the olive oil with onions, green pepper, mushrooms and spices, stirring often
add kidney bean and tomatoes. Simmer 30 minutes or until beans are tender, stirring occasionally
top with shredded cheese. Have the peach for dessert
LATE-NIGHT SNACK:
1 ounce turkey breast, sliced
1/2 cup grapes
1 macadamia nut.

DAY 2 RECIPES
BREAKFAST:
Old Fashion Oatmeal and Bacon
1 cup dry oatmeal plus 2 cups water
1 ounce Canadian bacon
2 tbs. Protein powder
nutmeg and cinnamon to taste
1 tbs. Slivered almonds
Cook oatmeal according to package directions. After cooling, stir in protein powder and spices
and top with slivered almonds. Cook Canadian bacon separately
LUNCH:

Cheeseburger
4 1/2 ounces lean hamburger meat (less than 10% fat)
1 slice reduced fat cheese
tomato slices, lettuce leaf and onion slice
1 piece rye bread
1/2 apple
6 peanuts
Broil hamburger to preferred degree of doneness (about 5 minutes per side for medium) place cheese on top and broil hamburger until cheese is melted. Put cheeseburger together with tomato, lettuce and onion have the apple and peanuts for dessert

AFTERNOON SNACK:

Tofu Veggie Dip
3 ounces firm tofu
mix with 1/3 tsp. Olive oil and sprinkling of onion soup mix
1 1/2 cups broccoli and green peppers. Cut for dipping

DINNER:

Barbecued Chicken
3 ounces grilled chicken
2 cups romaine lettuce
1/4 cup mushrooms, sliced
1/4 cup tomatoes, sliced
1/4 cup onions, chopped
lemon juice to taste, dash Worcestershire sauce, pepper to taste
sprinkling of Parmesan cheese
1 orange
1 tbs. Olive oil and vinegar dressing
Preheat oven to 450 degrees. Cover the chicken breast with slices of lemon and onion.
Baste with barbecue sauce. Cook for 10-15 minutes or until done.
Serve with vegetable and have strawberries for dessert

LATE-NIGHT SNACK:

1 ounce reduced-fat cheese
1 peach
3 olives.

DAY 3 RECIPES

BREAKFAST:

Fruit Salad
3/4 cup low-fat cottage cheese
cup strawberries, 1/2 cup grapes
3/4 cup cantaloupe, cubed
3 macadamia nuts, crushed
mix together and enjoy!

LUNCH:

Chef Salad
1 1/2 ounces deli-style ham
1 1/2 ounces deli-style turkey breast
1 ounce reduced-fat cheese
1 large tossed green salad
1 tbs. Olive and vinegar for salad dressing
1 nectarine for dessert
toss together all ingredients and enjoy

AFTERNOON SNACK:

1/4 cup low-fat cottage cheese
mix with 1/2 cup diced pineapple

DINNER:

Foiled Fish

4 1/2 ounces fish fillet of your choice (flounder is suggested)
Freshly ground pepper to taste
Squirt of lemon juice
Onion to taste, chopped
1 cup asparagus cooked
1 tossed salad w/ olive oil & vinegar dressing
Sprinkling of Parmesan cheese
1 plum for dessert

Tear off a good-sized piece of foil. Spray the center lightly with vegetable spray. Put the fish in the center of the foil with the onion, pepper, lemon juice and cheese. Fold foil over fish, leaving space around the fish. Carefully turn up and seal the sides and the middle so that juices don't leak out. Bake in a 425 ° oven for 18 minutes. When done, carefully open the foil to prevent steam burn

LATENIGHT SNACK:

1 ounce turkey breast, sliced
1/2 cup grapes
1 macadamia nut.

DAY 4 RECIPES

BREAKFAST:

Yogurt and Fruit
1 ounce lean Canadian Bacon
1 cup strawberries
1 1/2 cups plain low-fat yogurt
1 tbs. Slivered almonds

Cook Canadian bacon separately.Mix fruit with yogurt and top with slivered almond

LUNCH:

Grilled Chicken Salad
3 ounces grilled chicken
2 cups romaine lettuce
1/4 cup mushrooms, sliced
1/4 cup tomatoes, sliced
1/4 cup onions, chopped
lemon juice and pepper to taste
1tsp. Olive oil and vinegar for dressing
1 apple for dessert
1 orange for dessert

Prepare the salad, drizzle with olive oil, vinegar. Add lemon juice, season with garlic powder and Worcestershire sauce and grind in fresh pepper. Toss until well combined. Place grilled chicken on top and sprinkle cheese to taste. Serve

AFTERNOON SNACK:

1 ounce cheese
1/2 apple

DINNER:

Pork Medallions
3 ounces pork medallion or thinly sliced pork chops
1 1/2 cups steamed broccoli
1 spinach salad w/ olive oil and vinegar dressing
1/2 apple sliced
1 tbs. White wine
1/4 cup water
Rosemary and Dijon mustard to taste

Put pork into baking dish in a singer layer. Top with apple slices, rosemary and mustard.
Pour wine and water around the pork. Bake at 450 degrees for 15 minutes. Baste the pork with pan juices.
Reduce heat to 350 degrees and continue cooking for 10-15 minutes or until pork is white, not pink inside

LATENIGHT SNACK:

1 ounce soft cheese

4 ounces red wine.

DAY 5 RECIPES
BREAKFAST:
French Toast Sticks
4 egg whites or 1/2 cup egg mix
1 ounce extra-lean Canadian bacon
1 slice whole grain bread
1 cup strawberries, sliced
1 tsp. Slivered almonds
Cook Canadian bacon separately. Cut bread into sticks and soak into beaten egg whites (scramble any egg mixture that remains). Spray a nonstick pan with vegetable spray. over medium-low heat, cook breadsticks, turning often, until done. Top with sliced strawberries and slivered almonds
LUNCH:
Chicken Salad Sandwich
3 ounces cooked chicken breast, shredded celery, chopped
1/2 cup grapes
lettuce
tomato slice
1 piece rye bread or 1 mini pita pocket
1 tbs. light mayonnaise
Mix shredded chicken with mayonnaise, celery and grapes. Put into mini pita pocket, add lettuce and tomato slice
AFTERNOON SNACK:
3 ounces firm tofu mix with 1/3 tsp. Olive oil and sprinkling of onion soup mix
1 1/2 cups broccoli and green peppers cut for dipping
DINNER:
Meatloaf
3 ounces lean ground meat or turkey
2 tbs. egg whites
1 tbs. ketchup
1/2 onions chopped
1 tsp. bread crumbs
pepper to taste
dash of Worcestershire sauce
1 1/2 cups cooked zucchini
1 apple for dessert
1 tossed salad with 4 tsp. Olive oil and vinegar dressing
Mix ground meat, egg whites, ketchup, onions, bread crumbs, pepper and Worcestershire sauce. Form into a shallow loaf and place in microwave-safe dish. Cover with waxed paper. Microwave on medium for 10-15 minutes or until done. Have the zucchini as a side dish
LATENIGHT SNACK:
1 ounce turkey breast, sliced
1/2 cup grapes
1 macadamia nut.

DAY 6 RECIPES
BREAKFAST:
Skillet Hash
3 ounces cooked lean ham, chicken or beef
1/3 cup cooked potato, diced
1 cup tomato, chopped
green bell pepper, onions and mushroom, chopped
dash of Worcestershire sauce
1/2 cantaloupe
1 1/3 tsp. Olive oil
In a non-stick pan, sauté green pepper, onions and mushrooms in olive oil until tender.

Add cooked meat, potato and vegetables, spices and Worcestershire sauce. Cook, stirring until heated through.
Serve. Have cantaloupe as a side dish

LUNCH:

BLT Sandwich
3 ounces cooked extra lean Canadian bacon
1 ounce non-fat cheese
1 slice rye bread
lettuce
tomato, sliced
1 tsp. light mayonnaise
6 macadamia nuts
1 orange

Cook bacon, serve with rye bread topped with lettuce sliced tomatoes, cheese and mayonnaise.
Have the orange and nuts with the sandwich

AFTERNOON-SNACK:

2 ounces low-fat cottage cheese
1/2 cup pineapple diced
3 olives chopped

DINNER:

Quick Turkey dinner
6 ounces deli-style turkey breast or 4 ounces cooked skinless turkey breast
1 1/2 cups steamed broccoli
1/2 cup cranberries
4 tsp. slivered almonds

Serve cooked broccoli and cranberries as a side dish. Sprinkle turkey with slivered almonds

LATE-NIGHT SNACK:

1 ounce reduced-fat cheese
1 peach
3 olives.

DAY 7 RECIPES

BREAKFAST:

Scrambled Eggs Benedict
2 ounces lean Canadian bacon
4 large egg whites or 1/2 cup egg mix
1/2 English muffin
1/2 grapefruit
1 1/3 tsp. olive oil
Beat egg whites and olive oil with a little milk if desired.
Spray a nonstick pan with vegetable spray and then scramble the eggs. Toast the English muffin.
Cook Canadian bacon, place on the toasted muffin and top with the egg

LUNCH:

Turkey in a Pocket
4 ounces deli-style turkey breast or 4 ounces cooked turkey breast
1/2 mini pita pocket
1 green bell pepper chopped
1 tomato, sliced
1 plum for desert
1/2 tbs. Guacamole

Stuff pita pocket with turkey*, top with green bell pepper, tomato slices and guacamole

AFTERNOON SNACK:

two hard-boiled egg whites
1/2 apple
1 celery stick

DINNER:

Broiled Salmon

4 ounces salmon fillet with rosemary, tarragon, and dill to taste, lemon (optional)
1 cup cooked (steamed) zucchini
2 tomatoes
1 apple
1 tsp. olive oil

Rub the fillet with herbs and then brush with olive oil. Broil for 10 minutes per inch of thickness, turning and basting once. Cut tomatoes in halves, sprinkle with parmesan cheese and broil. Have the apple for dessert

LATENIGHT SNACK:

1 ounce turkey breast, sliced
1/2 cup grapes
1 macadamia nut
1 cup strawberries
1 teaspoon slivered almonds.

REFERENCES:

1. Zone Diet, Barry Sears, Ph.D.
2. Mastering the Zone, Barry Sears, Ph.D.
3. Protein Power, Michael R. Eades, M.D., Mary Dan Eades, M.D.

ZONE DIET RATIOS

By Barry Sears, Ph. D.

WEIGHT IN GRAMS / DAILY DIETARY CALORIE COUNT

% TOTAL CALORIES	FOODSTUFFS	1300	1500	1800	2000
40 %	**carbohydrate (gms./day)**	**102**	**120**	**135**	**150**
30 %	**protein (gms./day)**	**80**	**90**	**100**	**110**
30 %	**fat (gms./day)**	**30**	**35**	**40**	**45**

1.33 x protein gms = carbohydrates gms
0.4 x protein gms = fat gms

Dietary Zone Ratio Guidelines offer several benefits:

1) Permanent weight control through active fat loss.
2) Easier maintenance of your ideal weight, along with higher allowable daily calorie consumption.
3) A more palatable maintenance diet plan, with a bedtime snack!
4) Higher endurance and maximum physical performance, without lingering muscle pain.
5) The proper hormonal balancing effect of food, resulting in symptomatic relief and enhanced mental productivity.

TOTAL CARBOHYDRATE SOURCES -- generally need to be reduced for most people

1) **Glucose** - highest glycemic index
   a) sweets, sugar, honey --- very high insulin response.
   b) pasta, rice, potato, bread, grains --- moderately high insulin response.
   c) vegetables - good low calorie options --- low insulin response.

2) **Galactose** - moderate glycemic index
   a) milk and dairy --- moderately high insulin response.
   b) yogurt - good zone snack --- low insulin response.
   c) low fat cottage cheese - good high protein zone snack --- low insulin response.

3) **Fructose** - lowest glycemic index
   a) fruit --- generally produce a moderate to low insulin response.
   b) fruit juice --- should be diluted to reduce insulin secretion

Nutritional Supplement Recommendations Company: NutraCompute LLC
Order # Local: (303) 818-1278, www.nutracompute.com

List of Supplements to be Taken i.e. condensed list	Dosage	Times Per Day
CardioCysteine (NutraCompute,$10.40,120ct.)	1	2
Daily Defense 120 (NutraCompute,$34.00,60ct.)	2	2
DHEA-5 Sublingual (NutraCompute,$8.40,100ct.)	3	am
DIM (Douglas,$35.80,60ct.)	1 cap	2
Essential-4-Nutrition Pak (NutraC.,$83.00,30ct.)	1	1
Iodine (NutraCompute,$22.00,60ct.)	1	1
Magnesium Complex (NutraCompute,$15.50,120ct.)	2	1
NutraPro-Gest Cream (NutraCompute,$22.00,2oz.)	Apply	2
SAMe 200 (NutraCompute,$39.00,30ct.)	2 tabs	2
Toxic Metal Eliminator (NutraCompute,$16.00,90ct)	1	2

Recommended Supplements per Classification i.e. expanded list

Gonadal		
Aqua-E (Douglas,$25.00,8oz.)	1 tsp	2
Ant-E Oxidant 400 (NutraCompute,$21.30,60ct.)	2	1
NutraFem (NutraCompute,$24.00,120ct.)	1	2
Pregnenolone 25 (NutraCompute,$18.20,60ct.)	2	pm
NutraPro-Gest Cream (NutraCompute,$22.00,2oz.)	Apply	2
Low Gear		
Friendly Bacteria (NutraCompute,$23.90,100ct.)	1 cap	2
Ultra Green Food Caps (NutraC.,$23.90,90ct.)	2 caps am	2 caps
DIM (Douglas,$35.80,60ct.)	2 caps am	2 caps
Ultra Green Food Caps (NutraC.,$23.90,90ct.)	2 caps am	2 caps
DIM (Douglas,$35.80,60ct.)	2 caps am	2 caps pm
L-Glutamine (Douglas,$8.40,60ct.)	2	1
Immune		
SAMe 200 (NutraCompute,$39.00,30ct.)	2 tabs	2
Daily Defense 120 (NutraCompute,$34.00,60ct.)	2	2
Adrenal		
Ultra Anti-Oxidant (Douglas,$44.30,90ct.)	1	2
CardioCysteine (NutraCompute,$10.40,120ct.)	1	2
Soy-Lecithin (NutraCompute,$9.40,100ct.)	1	2
DHEA-5 Sublingual (NutraCompute,$8.40,100ct.)	3	am
Gin-Ergy (NutraCompute,$13.00,60ct.)	1	2
DIM (Douglas,$35.80,60ct.)	1 cap	2
Nutra Lipoic Acid (NutraCompute,$18.70,60ct.)	1	2
Magnesium Complex (NutraCompute,$15.50,120ct.)	1	2
Activated B-2 (NutraCompute,$10.40,100ct.)	1	2
DIM (Douglas,$35.80,60ct.)	1 cap	1
Vitamin/Mineral Balance		
Phos Free Calcium (NutraCompute,$16.00,120ct.)	2	2
Essential-4-Nutrition Pak (NutraC.,$83.00,30ct.)	1	1
Ant-E Oxidant 400 (NutraCompute,$21.30,60ct.)	2	1

NutrOmega 3 (NutraCompute,$14.00,100ct.)	2	2
Magnesium Complex (NutraCompute,$15.50,120ct.)	2	1
Single Lab Tests Out of Range		
CardioCysteine (NutraCompute,$10.40,120ct.)	1	2
Nutra B-12 Plus (NutraCompute,$9.40,90ct.)	1	2
Soy-Lecithin (NutraCompute,$9.40,100ct.)	1	2
DHEA-5 Sublingual (NutraCompute,$8.40,100ct.)	3	am
Immune Protect (Botanicals,$59.80,60ct.)	1	2
NutraPro-Gest Cream (NutraCompute,$22.00,2oz.)	Apply	2
Single Lab Tests Out of Range - Minerals		
CalMag Plus (NutraCompute,$9.40,90ct.)	2	1
pH Balance (Botanicals,$15.00,90ct.)	1	1
Ultra Green Food Caps (NutraC.,$23.90,90ct.)	1 cap	2
Copper 2mg (Douglas,$5.20,100ct.)	1	1
T-Cell Factor (NutraCompute,$45.70,30ct.)	1	1
Daily Defense 120 (NutraCompute,$34.00,60ct.)	1	2
Iron + C (NutraCompute,$9.40,60ct.)	1	2
Lithium Plus (NutraCompute,$19.20,100ct.)	1	2
Mineral Ascorbate C (NutraCompute,$17.20,90ct)	1	1
Manganese Chelate (Douglas,$6.80,90ct.)	1	2
Molybdenum (NutraCompute,$9.40,60ct.)	1	2
NutraPlex I (NutraCompute,$16.00,60ct)	1	2
Colloidal Silver (Progressive,$22.00,4oz.)	1 tsp	2
Biochemical Balance		
CalMag Plus (NutraCompute,$9.40,90ct.)	1	2
pH Balance (Botanicals,$15.00,90ct.)	1	2
Ester C (NutraCompute,$19.20,100ct.)	1	2
Ester C (NutraCompute,$19.20,100ct.)	1	2
Toxic Minerals		
DIM (Douglas,$35.80,60ct.)	2 caps am	2 caps
Ant-E Oxidant 400 (NutraCompute,$21.30,60ct.)	2	1
Aqua-E (Douglas,$25.00,8oz.)	1 tsp	2
Lymph Detox (Botanicals,$26.00,120ct.)	1	2
Toxic Metal Eliminator (NutraCompute,$16.00,90ct)	1	2
Digestants		
Pan Enzyme Plus (NutraCompute,$17.20,90ct.)	1	2

Take all nutrients with meals unless otherwise advised.

The nutrient recommendations above are designed to support you in your present metabolic condition. We strongly advise your health professional to carefully review and evaluate these recommendations to determine their usefulness to you.

CAUTION: If any of these nutrients cause unusual side effects discontinue at once and contact your physician or health professional.

The above nutritional recommendations are applicable for a 30 to 45 day period unless otherwise directed by your health professional. A repeat NutraBalance program to evaluate your level of improvement is advised at that time. For most people, maintainance recommendations can be instituted by following the results of this second analysis.

The results of following this program may be variable for each individual and may also be influenced by other factors and conditions. The recommendations in this program are meant to be followed under the supervision of a physician or other health professional. They are not to be used as a prescription or a cure for disease and are not meant to be a substitute for necessary medical care.

Lifestyle Recommendations

Category IA	21st Century Health/Therapeutic Environmental Center
Category IIA	Magnetic Therapy
Category IIB	Skin Brushing
Category III	Detoxification Therapy
Category IVA	Living Air Ion/Ozone Therapy
Category VIA	Female Hormone Booster Therapy
Category VID	Immune Enhancement Therapy
Category VIE	Anti-Aging Therapy
Category VIIB	Massage Therapy
Category VIIC	Physical Medicine
Category VIIIA	Body/Mind Medicine
Category IXA	Raw Juice/Enzyme Therapy

Nutritional Supplement Recommendations Company: Metagenics

List of Supplements to be Taken i.e. condensed list	Dosage	Times Per Day
Andrographis Plus	2	3
Bio-Scorb	1	2
Endurabolic	1	2
Fem Endocrine	1	2
Fem Premenstrual	2	2
GLA Forte	1	2
Metagest	1	3
Multigenics Intensive Care	1	2
Phyto Complete	1 scoop	1
Botanigest	6 plts	2
Toxi-Cleanse	1	2
UltraClear Plus	2 scoops	1

Recommended Supplements per Classification i.e. expanded list

Gonadal		
E-400 Selenium	1	2
Fem Endocrine	1	2
Fem Premenstrual	2	2
Mycelized Vitamin E	1 ml (20 drops)	2
Low Gear		
Chlorotene	1	2
Endurabolic	1	2
Ultra Dophilus	1/2 tsp	2
U.G.I.	1	2
Immune		
Andrographis Plus	2	3
VascuTone	1	2
Adrenal		
Cortico-B5B6	1	1
Endura	1	2
Intrinsic B12/Folate	2	1
VascuTone	1	2
Vitamin/Mineral Balance		
Magnesium Citrate	1 am	2 pm
Multigenics	2	3
Single Lab Tests Out of Range		
Fibroplex	1	2
Germanium Sesquioxide Formula	1	1
GLA Forte	1	2
Hemagenics	1	2
Intrinsic B12/Folate	1	2
Mag Glycinate	1	1
Multigenics Intensive Care	1	2
Osteo-Citrate	1	2
Phyto Complete	1 scoop	1
UltraBalance Herbulk	1 scoop	2

UltraClear	2 scoops	2
UltraClear Plus	2 scoops	1
UltraClear Sustain	2 scoops	2
UltraMeal	2 scoops	1
Biochemical Balance		
Bio-Scorb	1	2
Ultra Potent-C 500	1	2
Toxic Minerals		
E-400 Selenium	1	2
Botanigest	6 plts	2
HepataPlex	8 plts	2
Toxi-Cleanse	1	2
Digestants		
Metagest	1	3

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Nutritional Supplement Recommendations Company: Progressive Labs

List of Supplements to be Taken i.e. condensed list	Dosage	Times Per Day
B-12 Lingual	2	1
Coppomin	1	2
Dhea-10 Sublingual	2	am
Ex-Tox	1	2
Gonado-F	1	2
Acida-Zyme (after meals)	1	3
Thymotrate	2 tabs	2
MI-T Cell	1	2
Mega Once-A-Day	1	1
Pro-Caps	1 cap	1
Calcium Citrate Plus Vit. D	1	1
Pro-Gest Cream	Apply	2
Magnezyme	1	2
Pyridoxal 5 Phosphate	1	1

Recommended Supplements per Classification i.e. expanded list

Gonadal		
E 400 Sesame	1	2
Gonado-F	1	2
Pro-Gest Cream	Apply	2
Low Gear		
Siberian Ginseng	1	2
MI-T Cell	1	2
Immune		
Thymotrate	2 tabs	2
Sinus-Allergy Capsules	2 caps	2
Adrenal		
B-12 Lingual	2	1
Dhea-10 Sublingual	2	am
Pyridoxal 5 Phosphate	1	1
Vitamin/Mineral Balance		
Mega Once-A-Day	1	1
Single Lab Tests Out of Range		
B-12 Lingual	2	1
B-12 Lingual	1	2
C Aspa Scorb	1/2 tsp	1
C Aspa Scorb	1/2 tsp	1
Coppomin	1	2
Hemaplex	1	2
Germanium Sesquioxide	1	1
Thymotrate	1 tabs	2
Molymin	1	2
Bio-Guard	1	2
Pro-Caps	1 cap	1

Lithinase	2 caps	2
Calcium Citrate Plus Vit. D	1	1
Pro-Gest Cream	Apply	2
Magnezyme	1	2
Toxic Minerals		
Green Tea Polyphenols	2	2
E 400 Sesame	1	2
Ex-Tox	1	2
Digestants		
Acida-Zyme (after meals)	1	3

Take all nutrients with meals unless otherwise advised.

The nutrient recommendations above are designed to support you in your present metabolic condition. We strongly advise your health professional to carefully review and evaluate these recommendations to determine their usefulness to you.

CAUTION: If any of these nutrients cause unusual side effects discontinue at once and contact your physician or health professional.

The above nutritional recommendations are applicable for a 30 to 45 day period unless otherwise directed by your health professional. A repeat NutraBalance program to evaluate your level of improvement is advised at that time. For most people, maintainance recommendations can be instituted by following the results of this second analysis.

The results of following this program may be variable for each individual and may also be influenced by other factors and conditions. The recommendations in this program are meant to be followed under the supervision of a physician or other health professional. They are not to be used as a prescription or a cure for disease and are not meant to be a substitute for necessary medical care.

Metabolic Classification Discussion

The strength of this Nutrabalance report lies in the process of clustering and summarizing the tests into metabolic classifications. The most significant classifications for this individual are listed on the Summary of Findings page. The following pages discuss in more detail the relationship between individual test or symptomatic categories, and their respective metabolic or biochemical class. You may notice that any one test can be listed in more than one classification. The classifications are listed in order of severity: first primary, then secondary, tertiary and if applicable quaternary.

PRIMARY TYPE: GONADAL

VeryLow Progesterone, **VeryHigh Pre Menstrual Syndrome Symptoms**, **VeryHigh Dysmennorhea Symptoms**, Low BArsenic, Low BBoron, Low BCopper, Low BGermanium, Low BLithium, Low BManganese, Low BUN, Low DHEAS, Low BMagnesium, High Genital Disorders Symptoms, High Menopause Symptoms, Low Sodium

This type indicates an imbalance of the reproductive system including the effect of gonadal hormones (estrogen, progesterone, and testosterone) on metabolism. The reproductive system mainly consists of the uterus/prostate and ovaries/testicles. The system is primarily regulated by the hypothalamus, anterior pituitary, and pineal glands.

The reproductive system's function is the maintenance and development of male and female sexual characteristics, reproduction and sexual arousal. The gonadal hormones also have effects on protein, fat and water/electrolyte metabolism including protein synthesis from amino acids and sodium/potassium regulation.

An imbalance in gonadal function can be associated with a number of metabolic effects:

-deficient levels of Progesterone (LOW PROGESTERONE), if tested during the latter half of the luteal phase of the menstrual cycle (day 22-26) in a menstruating woman, signals failure of adequate production of progesterone by the ovarian corpus luteum following ovulation. This can be due to a number of possible causes including deficient amounts of Vitamin B6 or Magnesium, both of which are common problems with today's diet; excessive intake of animal fat providing arachadonic acid producing prostaglandin E3 which shunts hormone production away from Progesterone; stress and chemical imbalances in the acidic direction which affects cholesterol metabolism and therefore hormone production. Treatment should be centered around correcting the underlying dietary and metabolic disorders coupled with replacement therapy using natural Progesterone (1,25 Di-hydroxy-Prog.). Progesterone deficits also occur as a normal aspect of the aging process, associated with declining hormone production throughout the endocrine system. A low level of Progesterone poses an increased risk for uterine and breast cancer, since Progesterone provides a protective factor for these conditions. A topical Mexican yam product, an actual hormone product such as ProGest cream or natural progesterone, or an herbal form such as Sarsaparilla can also be effective in elevating progesterone depletions.

-a LOW PROGESTERONE coupled with a LOW DHEA indicate inadequate production of Dhea from the adrenals and progesterone from the adrenals and/or ovaries. DHEA is also related to the gonadal system since, although this hormone is primarily an estrogen precursor, lower levels tend to indicate an adrenal problem, commonly adrenal fatigue. DHEA (the aging hormone) tends to decline with age and is believed to be a major cause of estrogen deficiency commonly seen in menopausal women. Progesterone depletions can result from stress as well as chemical, vitamin and mineral imbalances, and are a common cause of poor protein utilization and osteoporosis since progesterone functions as an anabolic bone building substance.

-a LOW DHEA can result from an underactivity of the adrenals which is where DHEA is actually produced. This can be due to a primary or secondary effect, and can also affect Estrogen levels since DHEA is a direct precursor for Estrogen. Adrenal function supplies only small amounts of progesterone compared to ovarian production in a menstruating woman. Further studies can clarify the cause of the adrenal fatigue: ACTH (adrenocorticotrophic hormone) MEASUREMENTS ARE INDICATED TO CLARIFY THIS CONDITION, if hi - primary fatigue of the adrenals, which calls for adrenal stimulation directly; if low - secondary fatigue coming from the anterior pituitary, which calls for pituitary stimulation along with adrenal. DHEA deficiency occurs with age, as part of the natural aging process. DHEA has been shown to prevent and even reverse osteoporosis, relieve adrenal fatigue, lower LDL and raise HDL levels, retard the aging process, improve memory, accelerate weight and fat loss, increase the sex drive and act as an anti-depressant. DHEA is contraindicated in cases of prostate or testicular cancer since it may promote hormone sensitive tumor growth.

-fluctuations in gonadal hormone levels can lead to changes in protein (amino acid) metabolism and enzyme synthesis.

-sex hormones cause the retention of salt and water from the kidneys. Gonadal activity thereby has a regulatory effect on total body water.

SECONDARY TYPE: LOW GEAR

VeryHigh BMercury, **VeryHigh HepatitisCAB**, **VeryLow % TRNFSat**, **VeryHigh Liver Symptoms**, **VeryHigh Musculoskeletal Symptoms**, **VeryHigh Connective Tissue Symptoms**, **VeryHigh Digestion Symptoms**, **VeryHigh Colon Symptoms**, Low BBoron, Low BCobalt, Low BCopper, Low BGermanium, Low BIodine, Low BLithium, Low BManganese, Low BMolybdenum, Low BUN, Low Ferritin,

Low BMagnesium, High MCV, High Platelets, High PTH, High Posterior Pituitary Symptoms, High Low Blood Sugar Symptoms, High High Blood Sugar Symptoms, High Pancreatic Digestion Symptoms, High Upper GI Symptoms, High GI Infection/Inflammation Symptoms, High GI Systemic Effects Symptoms, Low Sodium, High TIBC, Low TotIron, Low UrinepH

Analysis of the blood chemistries, laboratory measurements and results of your symptomatic survey questionaire (if submitted) indicate you have a metabolic pattern referred to as the "Low Gear System." The Low Gear System consists of the pancreas, liver/spleen, parathyroid and the gastrointestinal tract (mouth, salivary glands, esophagus, stomach, small and large intestine). These are often collectively referred to as the digestive or elimination system. The Low Gear System can also be referred to as the anabolic or tissue building component of the metabolic process, since together these organs are primarily concerned with the assimilation or utilization of food nutrition. As a whole they determine the nutritional benefit derived from the food we eat.

Activation of any or all of these glands/organs is generally associated with eating, since the consumption of food leads to stimulation of the parasympathetic nervous system and initiation of the digestive process. In addition, inactivity and/or certain relaxing techniques, especially while eating a meal, can be helpful in promoting Low Gear System responses. This type of "dining enviroment" may help facilitate the absorption and uptake of the nutritional content of food, resulting in a better overall nutritional status as it relates to the quality of food in general. If the Low Gear System is functioning properly, we should be able to fully absorb and assimilate the food we eat, such that the nutritional content of the food closely correlates to the benefit derived. Our overall nutritional status should essentially be similar to the quality of the food choices we consume. Thus the saying "you are what you eat." The higher the nutritional quality of the food, the greater the outcome we should achieve in a nutritional sense. This would be the case with an ideal or optimal performance level of functioning.

In contrast, the absorption and uptake of the nutritional content of food may be impaired if the Low Gear System is suppressed or reduced in its functioning. A decrease in the strength of Low Gear System responses could impair the digestive process in such a way that we would absorb and acquire less of the nutritional content of food in an overall sense. Therefore, even though we are eating a well planned nutritionally sound diet, we could still have poor nutrition. This is the difference between diet and nutrition, and under conditions of Low Gear System imbalances, the saying "you are what you assimilate of what you eat" actually becomes more appropriate. Suppression of Low Gear System glands and organs has been shown to be associated with physical and/or emotional stress, eating in a hurried or stressful enviroment, and with congestion or toxicity of the digestive organs.

An imbalance of the Low Gear System can be associated with a number of metabolic effects:

Liver

-an impairment in the uptake/absorption of iron and/or possible blood loss may result in a depletion of total body iron stores and a reduction in oxygen carrying capacity. This can lead to the development of an iron deficiency anemia if not corrected.

-a HIGH HEPATITIS C ANTIBODY titer indicates either an active infection with a viral form of hepatitis, or the condition of a carrier state for such an infection, which is considered to be possibly a public health concern since the disease can be transmitted through contact. The active form of Hepatitis C is an actual inflammatory condition involving the liver which, if left untreated, can progress to liver failure. The carrier state of Hepatitis C is a dormant infection which can progress to the active state, but is not presently doing so. Both conditions are transmittable, and measures must be taken to identify carriers so the infection will not be passed on to others through blood transfusions or bodily fluid contact. Hepatitis C should be suspected whenever there are elevated liver enzymes either for which there is no obvious explanation, or which do not resolve with appropriate treatment. Certain steps should be taken to prevent the passive form of Hepatitis C from progressing to an active state. These include detoxification, removal of heavy metals, use of the Silimarin containing herb Milk Thistle, and thinning the bile with herbal formulas to provide for its ease of passage through the caniliculea (tubules).

p. 263 Henry, John Bernard. *Clinical diagnosis and management by laboratory methodsi0 (19th ed.). Philadelphia: Saunders, 1996.*

-an impairment in the uptake/absorption of iron and/or possible blood loss may result in a depletion of total body iron stores and a reduction in oxygen carrying capacity. This can lead to the development of an iron deficiency anemia if not corrected.

-An increased platelet count (HIGH PLATELETS), collectively known as thrombocytosis, can occur due to a benign reactive process, associated secondarily with other conditions including drug reactions, or as part of a myeloproliferative process such as leukemia. Benign causes include response to exercise, pregnancy, prematurity, and vitamin E deficiency. These are usually associated with modest elevations above the physiologic upper limit of the lab range. Associated conditions producing more marked elevations in platelet count include acute infections or inflammatory disorders, osteoporosis, cardiac disease, diabetes, renal (kidney) failure, and seizure disorders. Myeloproliferative conditions generally stem from bone marrow production, and are often found to be associated with abnormal levels of arachidonic acid (a fatty

acid) which can come from dietary sources.

pp. 709-710 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

-a disturbance in the neutralization and/or elimination of environmental toxins through the liver and gastrointestinal tract can result in the accumulation of unwanted substances. This can produce irritation and/or inflammation of the liver, with resultant underperformance of it's functions.

Gastrointestinal

-poor uptake of magnesium and iron from dietary sources which can result from relative malabsorption. This can also be seen with inadequate dietary supply.

-a decrease in HCL acid secretion from the stomach can be associated with acidification of urine.

-low levels of trace minerals and similar elements can result from underperformance of the digestive system with a condition known as relative malabsorption. This can occur when physical, chemical or psychological stress is present. Stress conditions tend to reduce the proper digestion and absorption of foodstuffs leading to poor uptake and assimilation. The presence of elevated levels of certain trace elements can also produce relative malabsorption by suppressing the absorption of food in general.

-a disturbance in the elimination of environmental toxins from the gastrointestinal tract can occur due to an impairment in digestive function, resulting in the accumulation of unwanted substances.

Parathyroid

The primary function of Parathyroid Hormone (PTH) is to regulate the extracellular ionized calcium concentration. PTH normally causes a rise in serum calcium levels, primarily through bone resorption (demineralization), by increasing osteoclastic activity to act on the more stable or established component of bone causing the mineral to be extraced. Additional effects of PTH on bone are increased formation of collagenase, which degrades the matrix of bone, and increased breakdown of the ground substance of bone as well. PTH also acts on the kidneys to increase serum calcium by enhanced reabsorption at the distal tubule. This is accompanied by phosphorous excretion and the development of a metabolic acidosis due to a loss of sodium as well as bicarbonate in the urine. PTH also stimulates the production of a vitamin D metabolite which results in increased intestinal absorption of calcium.

pp. 176-177 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

-a HIGH PARATYHROID HORMONE (PTH) level indicates that the parathyroid glands are in an overactive functioning state. This can occur as a primary condition due to parathyroid tumors or with multiple endocrine neoplasia (MEN) syndromes in combination with other glandular components. In the majority of instances where a high PTH level is found, however, additional factors related to mineral metabolism are usually responsible (secondary hyperparathyroidism). Detection of the responsible mineral imbalance may require whole blood or RBC levels, and is often not detected with serum measurements alone. The primary function of PTH is to regulate the extracellular ionized calcium concentration. PTH normally causes a rise in serum calcium levels, primarily through bone resorption (demineralization), by increasing osteoclastic activity to act on the more stable or established component of bone causing the mineral to be extraced. A NORMAL CALCIUM level will tend to result from the process of bone demineralization due to PTH elevation. Additional effects of PTH on bone are increased formation of collagenase, which degrades the matrix of bone, and increased breakdown of the ground substance of bone as well. PTH also acts on the kidneys to increase serum calcium by enhanced reabsorption at the distal tubule. This is accompanied by phosphorous excretion and the development of a metabolic acidosis due to a loss of sodium as well as bicarbonate in the urine. PTH also stimulates the production of a vitamin D metabolite which results in increased intestinal absorption of calcium. The combination of these mechanisms may eventually produce normal measurements of calcium and/or magnesium from bone demineralization, ultimately resulting from as well as associated with an elevated PTH level.

pp. 2223, 2241-42 Harrison, T.R. *Principles of Internal Medicine*(14th ed.) McGraw-Hill, 1998.

pp. 176-177 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

-a HIGH PARATYHROID HORMONE (PTH) level associated with a LOW MAGNESIUM would implicate the magnesium depletion as the chief cause of the parathyroid stimulation, a condition known as secondary hyperparathyroidism. Replacement of the depleted mineral level will likely help to relax the parathyroids, and also prevent bone demineralization from occuring to replenish the deficit. In the majority of instances where a high PTH level is found, a deficient level of serum calcium is often responsible, except in the unlikely case where an autonomously secreting tumor is responsible for the hormone elevation (primary hyperparathyroidism). It is just as possible however, that a depleted magnesium level, which is the rule rather than the exception in today's world of depleted soils and deficient food supplies, can result in PTH elevation. When magnesium, which is primarily an intracellular mineral, is depleted, this produces an ionic imbalance inside the cell. This ionic imbalance consists of a deficit in intracellular positive charges, and this deficit allows calcium, which also has a positive charge, to move inside the cell along calcium transport channel pathways to replace the positive charge deficit. This process is called "calcinosis", and results in calcification of muscular tissue with a loss of contractility. Magnesium intake from dietary sources is usually below RDA levels (300 mg/day vs. the RDA of 400), so that even if one assumes that the soils are not depleted and 100% of the magnesium in food is fully absorbed, magnesium deficiency can and often still does occur and is responsible as a common cause of PTH excess.

pp. 176-177 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

-Parathyroid imbalances can occur as a result of a magnesium deficiency (LOW MAGNESIUM), since parathyroid hormone (PTH) secretion is initially stimulated with marginal deficits, but then will be suppressed when magnesium (Mg) levels are markedly depleted on a prolonged basis. Restoration of the Mg deficiency must usually occur before PTH secretion will resume with chronic mineral depletion. PTH levels are thus in a state of dynamic flux, elevated early on in Mg deficiency and declining through the normal range to decreased levels with persistant deficits. Therefore PTH MEASUREMENTS ARE INDICATED and can be helpful in evaluating both the severity and chronicity of Mg depletion, which is a common problem in modern society. When magnesium, which is primarily an intracellular mineral, is depleted, this produces an ionic imbalance inside the cell. This ionic imbalance consists of a deficit in intracellular positive charges, and this deficit allows calcium, which also has a positive charge, to move inside the cell along calcium transport channel pathways to replace the positive charge deficit. This process is called "calcinosis", and results in calcification of muscular tissue with a loss of contractility. The loss of muscle contractility can be especially detrimental in the heart, where it can lead to cardiac arrythmias as well as infarction (heart attack). In smooth muscle such as in the intestines, constipation often results from calcinosis, and in skeletal muscle such as in the legs, muscle aches and cramps are often experienced. Magnesium intake from dietary sources is usually below RDA levels (300 mg/day vs. RDA of 400), so that even if one assumes that the soils are not depleted and 100% of the magnesium in food can be fully absorbed (usually not the case), magnesium deficiency can and often still does occur.

pp. 176-177 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

-changes in bone metabolism, which may result from an alteration in thyroid gland function can lead to an imbalance in calcium and phosphorous levels.

TERTIARY TYPE: IMMUNE

VeryHigh BMercury, **VeryHigh Immune Symptoms**, **VeryHigh Connective Tissue Symptoms**, **VeryHigh HepatitisCAB**, Low BArsenic, Low Basophils, Low AbsBasophils, Low BCobalt, Low BCopper, Low BGermanium, Low BLithium, Low Eosin, Low AbsEosinophils, High MCV, Low AbsMonocytes, High Platelets, High Genital Disorders Symptoms, High GI Infection/Inflammation Symptoms, High Neutrophils %, Low TotIron

This type indicates an imbalance in the immune system. The immune defense system is designed to protect the body from infection against bacteria, virus, parasites and fungal agents. It also serves to maintain internal homeostasis by removing damaged cells and unwanted substances. An immunologic reaction to the presense or accumulation of waste products, acids, and toxic elements can stimulate the response of several different types of white blood cells, some of which become elevated and others depleted. The immune system can also become involved with allergic reactions against outside irritants. When persistant and severe these allergic responses can lead to inflammation and even tissue destruction, potentially harming one's own body. Under certain conditions, the immune system can become "the enemy" and turn against us, causing untold damage to our cells and organs.

The immune system overall, is truly a wonder of nature and the God that created us. Immune system components consists of the red and white blood cells, lymphatic system, the lymphatic organs (appendix, tonsils, lymph nodes), thymus, bone marrow and portions of the liver and spleen. The immune stem or precursor cell originates in the bone marrow of adults, and can divide into two different cell lines. The lymphoid precursors give rise to B and T lymphocytes and natural killer (NK) cells, while the myeloid line can develop into red blood cells, platelets, monocytes (macrophages), neutrophils, basophils and eosinophils.

T cell maturation occurs in the thymus, expressing either the CD4 (helper) or CD8 (cytotoxic) cell surface molecule characteristics. B-lymphocytes mature in the bone marrow and play an important role in the so called "primary immune response" through the production of

antibody. Myeloid cell differentiation also occurs in the bone marrow, producing mast cells and dendritic cells in addition to the commonly recognized white blood cell differential outlined herein. Monocytes, in particular, mature into macrophages (phagocytes) and then migrate to specific locations in the body to provide specialized functions, such as Kupffer cells of the liver, osteoclasts of the bone and microglia of the brain. Extracellular organisms such as bacteria or parasites, as well as unnatural molecules such as metallic toxins, can either bind to macrophages which then engulf the organism and/or toxic substance, or they can sensitize neutrophils which would activate the "complement" fixation pathway leading to direct lysis of the organism.

The thymus gland plays an important role in regulating the immune system throughout life. Once thought to involute and die in the adult, thymic hormones have now been shown to be continually involved in not only immune defense, but also hypothalamic functions. These have profound affects on the entire hormonal system, including the thyroid, adrenals and gonads. A discussion of the immune system would not be complete without special mention of the lymphatic circulation. The lymphatic system drains waste materials from tiny areas deep within our tissues, where blood capillary flow does not reach, to the liver and eventually out of our bodies. As such the lymphatic system plays a major role in total body cleansing, in addition to its protective function as outlined above. Without a heart to pump the circulatory flow as is the case with blood, adequate drainage of lymphatic waste requires muscle contraction, better known as exercise, to move it along the circulatory path. Massage, chiropractic adjustments, rebounding or even shaking movements can be helpful in promoting lymph drainage, so important in the maintainance of optimal health.

pp. 1753, 1760-76 Harrison, T.R. *Principles of Internal Medicine*(14th ed.) McGraw-Hill, 1998.

pp. 847-50 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

An imbalance of the immune system can be associated with a number of metabolic effects:

-a heightened immune response due to an acute infectious-like process which is present or resolving.

LOW EOSINOPHILS can occur in any situation of acute stress where there is an increase in the secretion of adrenal hormones (cortisol and/or adrenalin), and in conditions associated with hyperadrenalism such as Cushings. Acute inflammatory states can also produce this effect due to eosinophil migration into involved sites. Suppression of eosinophil production may result from exposure to medications containing Atropine or Atropine derivatives, such as the Belladonna alkaloids and several anti-cholinergic preparations commonly used for abdominal spasm (pain).

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

LOW MONOCYTES can be seen in conditions where monocyte production is impaired, such as with states of immune depletion (AIDS, viral infection, leukemia and exposure to chemicals or steroid hormones). Since a large percentage of the monocyte population is produced within the GI (Gastrointestinal) system, disorders that compromise GI function can have a detrimental effect on the monocyte count.

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

LOW BASOPHILS can occur in any situation of acute stress or infection where there is an acute increase in the secretion of adrenal hormones (cortisol and/or adrenalin), and in chronic conditions associated with hyperadrenalism such as Cushing's disease and metabolic acidosis. About half of hyperthyroidism cases develop a basopenia. Basophil levels show a diurnal variation, with the lowest occuring in the morning and the highest at night.

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

pp. 295, 297-298 Wallach, Jaques B. *Interpretation of diagnostic tests* (6th ed.). Boston: Little, Brown, 1996.

-a disturbance in the neutralization and/or elimination of environmental toxins from the liver and gastrointestinal tract can result in the accumulation of unwanted substances, producing an autoimmune type reaction.

-an impairment in iron utilization by the RBC recycling mechanism of the spleen. This may also be due to a deficiency of dietary iron uptake and result in an iron deficiency anemia.

-a disturbance in electrolyte balance due to an alteration in adrenal function which can affect the immune response potential. The secretion of certain adrenal hormones has been shown to suppress immune function.

-an imbalance of the anterior pituitary gland which can lead to an alteration of the immune response.

-a HIGH HEPATITIS C ANTIBODY (anti-HCV Ab) indicates active infection with a Hepatitis C virus, (HCV)-the Flavivirus family of RNA viruses. Hepatitis C is most commonly acquired from blood transfusions, but can also be transmitted by intravenous drug abuse. Many cases are known as cryptogenic i.e. no predisposing cause can be identified. Antibodies to the virus develop during the incubation period of two to

twenty six weeks, and can be detected using the ELISA method. Over 75% of infected people become chronic carriers, where elevated ALT (SGPT) levels and antibodies to the virus persist. Screening for hepatitis C carriers in transfusion donor blood has reduced the transmission of HCV from 21% to less than 1%. The fatality rate of HCV infection is between 1% to 2%.

p. 263 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

QUATERNARY TYPE: ADRENAL

VeryLow Progesterone, **VeryHigh Adrenal Symptoms**, Low BCopper, Low BIodine, Low BLithium, Low BManganese, Low BMolybdenum, Low BUN, High Cortisol/DheaRatio, Low DHEAS, High Homocysteine, Low BMagnesium, High MCV, High Low Blood Sugar Symptoms, High High Blood Sugar Symptoms, Low Sodium, High T3Uptake, Low UrinepH

This type indicates an imbalance of the adrenal glands. These are two small glands that rest on top of the kidneys.

There are two main functional areas within each adrenal gland. The adrenal medulla is a glandular derivative of the sympathetic portion of the autonomic nervous system. It produces two hormones: epinephrine (adrenaline) and norepinephrine which stimulate the sympathetic nervous system (flight or fight mechanism). These hormones also increase blood glucose and fatty acid (triglyceride) levels, and express the dopamine excitatory neurotransmitter pathway.

The adrenal cortex secretes an entirely different group of hormones called corticosteroids i.e. aldosterone and cortisone, as well as DHEA and small quantities of estrogen, progesterone and testosterone. These hormones are all synthesized from cholesterol. The corticosteroids function in elevating blood glucose, increasing fat and protein breakdown, the balancing of electrolytes (sodium/potassium) and anti-inflammatory responses. Several studies have shown that psychological stress and sympathetic activation cause sodium retention from the kidneys, a function of aldosterone secretion by the adrenal cortex. Since aldosterone can only be derived from progesterone, via pathway #1, a deficiency of either of these hormones can have profound effects on acid/alkaline equilibrium.

An imbalance of the adrenal gland can be associated with a number of metabolic effects:

-chronic overstimulation of the adrenal glands due to stress, toxin accumulation, diuretic usage and other metabolic disorders can result in electrolyte (sodium/potassium) alterations and lead to adrenal fatigue.

-a LOW DHEA level indicates a reduction in the testosterone/estrogen production pathway for which DHEA is a precursor. This is usually the result of inadequate hormone synthesis since Dhea levels generally decline on a continual basis after age 30 in most people as a result of, and consistent with, the aging process. Replacement therapy is usually helpful in restoring energy and vitality. In adults, DHEA replacement may be effective in preventing osteoporosis (bone loss), and in women would tend to elevate estrogen levels without the side effects of estrogen therapy alone. DHEA supplements are contraindicated in cases of breast or prostate cancer; adequate screening is necessary before beginning replacement (mammograms in women, PSA and digital exam in men).

pp. 2036-2037 Harrison, T.R. *Principles of Internal Medicine*(14th ed.) McGraw-Hill, 1998.

A LOW DHEA can also be due to primary/secondary adrenal fatigue, the gland where Dhea is produced. The former condition would be associated with a normal cortisol level; the latter with a low ACTH. In this particular condition, it is associated with a HIGH CORTISOL/DHEA RATIO, consistent with a moderate degree of fatigue in the Adrenal Stress Response progression pattern, where the Cortisol has already declined to a NORMAL CORTISOL level. In the expected pattern of adrenal hyperstimulation/fatigue, the Cortisol and ACTH initially become elevated in response to a declining DHEA. As the stress continues these hormones begin to drop; first becoming normal and then low.

pp. 347-351 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

pp. 2039-2040 Harrison, T.R. *Principles of Internal Medicine*(14th ed.) McGraw-Hill, 1998.

-a metabolic acidosis and/or overstimulation of the adrenal glands can develop from the buildup of acid toxins within the body as well as excessive intake of acid producing foods. Acidification of the urine, saliva or venous blood (LOW PH) may also result from this alteration in acid/alkaline equilibrium.

-a LOW MAGNESIUM level can be a consequence of overstimulation of the adrenal glands. This can also be due to poor absorption or deficiencies of magnesium in the diet.

-there is a direct relationship between anterior pituitary and adrenal gland function. An imbalance in the anterior pituitary can lead to stimulation of the adrenals.

-a HIGH T3 UPTAKE indicates the receptors for T3 (active thyroid hormone) are, for the most part, unsaturated. This is consistent with underproduction of thyroid hormone from the gland itself, a secondary consequence of pituitary fatigue, under-medication with a thyroid

hormone supplement, or blocked expression of the hormone at the tissue level resulting from mineral or other hormone imbalances, stress or toxicity. This latter condition is referred to in the medical literature as "peripheral resistance to thyroid hormone," or decreased peripheral T3 production due to reduced T4 to T3 conversion. Further lab studies can help differentiate between these conditions: a Low T3 hormone level and a Low T4 would suggest underproduction from the gland itself; a Low T3 hormone with a High Reverse T3 suggests the blockage in the conversion of T4 to T3 at the tissue level (tertiary hypothyroidism). A high T3 Uptake can result from under-medication with active thyroid hormone even with a normal T4RIA (Wilson's Syndrome), and one could expect a normal or a high TSH with this condition, suggestive of an adequate (or less than) circulating thyroid hormone level. The T3 Uptake reflects the " percent" of available free T3 (active hormone) receptors for absorption on thyroid binding globulin compared to the population at large used as a control. A high reading generally indicates a blockage in the activation of T4 to T3 producing a high level of available T3 receptors, and a deficiency in the active T3 hormone level. The T4 may be normal since the thyroid gland can, and usually will, increase production to make up for the deficit.

pp. 335-40 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

pp. 528-29 Wallach, Jaques B. *Interpretation of diagnostic tests* (6th ed.). Boston: Little, Brown, 1996.

pp. 476-78 Ravel M.D., Richard *Clinical Laboratory Medicine* (6th ed.). St. Louis: Mosby, 1995.

-a LOW PROGESTERONE level indicates there is a blockage in the production pathway from cholesterol/pregnenolone which can be the result of several possibilities including vitamin B6 or magnesium deficiency, a low cholesterol level, stress, acidic chemical imbalance and pituitary fatigue associated with a low LH (Leutinizing Hormone). Since progesterone is also produced by the ovaries in women after ovulation, inadequate production in women should be related to the gonadal system, esp. with PMS.

-HIGH HOMOCYSTEINE levels indicate an increased risk for cardiovascular disease (CVD). Homocysteine is now considered to be a more important marker for cardiac risk than even cholesterol, and high levels are associated with a 2-4 fold risk of acute myocardial infarction (heart attack). Elevated homocysteine levels have also been shown to correlate with the development of carotid-artery stenosis. In distinction to the commonly held belief that cardiac disease primarily results from an abnormality in fat metabloism, homocysteine is actually a protein amino acid, and not even a fat. Since high homocysteine levels result from the incomplete metabolism of the essential amino acid methionine, an amino acid analysis may also be helpful in determining the most appropriate therapy for this condition. Elevated homocysteine levels are also considered to be a sensitive indicator of pyridoxine (Vit. B6), cobalamin (Vit. B12) and Folate deficiency. This is because vitamin B6 is required to convert homocysteine into cysteine, and B12 is a necessary co-enzyme in the conversion of homocysteine back to methionine if needed to reduce homocysteine levels. When either of these vitamins are deficient, homocysteine levels will increase in both serum and urine. Vitamin B12 deficiency, a relatively common problem due to impaired absorption from dietary sources especially in the elderly, causes a megaloblastic anemia to develop which is characterized by the enlargement of early developing red cells (megaloblasts) and an elevation of the MCV (mean corpuscular volume) measurement. Folic acid (FA) or Folate deficiency can result from inadequate intake or improper absorption of this important water soluble B vitamin. Humans with nutritional folate deficiency also develop homocysteine elevations. The blood and bone marrow features of megaloblastic anemia are similar to those seen with cobalamin or vitamin B12 deficiency, and many authors feel the anemia of B12 deficiency is really due to a tissue folate deficiency. Recent reports indicate an elevated neutrophilic hypersegmentation index (NHI) may also be helpful as an early indicator of B12 deficiency and a developing megaloblastic anemia.

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pp. 2199-2201 Harrison, T.R. *Principles of Internal Medicine*(14th ed.) McGraw-Hill, 1998.

BIOCHEMICAL TYPE: ACID

Acid Tests: **VeryHigh BMercury**, Low BMagnesium, Low Sodium, Low UrinepH

Alkaline Tests: none

Certain areas of the body actually have an acidic measurement (stomach, kidneys, bladder) but the overall pH of the body and blood normally must be maintained within a narrow range of alkalinity (pH 7.30 - 7.45). The term acidosis is relative and only meant to convey a shift in total body chemistry towards the acidic direction, since an absolute blood measurement of acidity (pH below 7.0) would be incompatible with life. In addition, this description is not meant to designate a diagnostic condition or medical disorder. Nor is the acidic biochemical type meant to represent the absolute pH measurement of the blood system, which generally requires a laboratory test performed in hospitals known as blood gases.

The principal sources of acid buildup are 1.) The metabolism and/or incomplete combustion (oxidation) of foodstuffs, metabolic "waste" produced as a by-product of cellular activity. Normal cellular functions constantly produce acids or "waste" products which must be "balanced", neutralized and removed by the body's buffering and detoxification systems, i.e., kidneys, lungs, liver, and blood. 2.) The consumption of acid in the air, food, and water supply. Nitrogen emmisions from automobiles and industrial plants, food dyes, sprays, waxes, perservatives, additives, artificial sweetners, fertilizers, water pollutants, and even chloride and flouride in tap water are just some of the

highly acidic chemicals we have to "cope" with on an everyday basis (many of which are not readily bio-degradeable).

Underlying regulatory forces must continually work to balance an acidic body chemistry. Until chemical balance is achieved, internal mechanisms will generally produce considerable "around the clock" tension (internal stresses) within us during the process of acid removal. These internal buffering mechanisms include the production of bicarbonate from the organs and cells of the body; the blowing off CO2 or carbon dioxide, an acid end product, from the lungs which can produce symptoms of shallow breathing, hyperventilation and persistant "nagging" cough; the release of alkaline bile from the liver and alkaline digestive secretions from the pancreas (which are lacking in enzymatic digestive power); and the retention of sodium from the kidneys in response to the secretion of the hormone Aldosterone, which results from adrenal gland stimulation producing the feeling of internal "stress".

An acidic imbalance can be associated with a number of metabolic effects:

-many of the minerals, and other factors involved in maintaining a normal body chemistry, fluctuate throughout the day in response to variations in serum and electrolyte concentrations, as well as dietary influences. Since a balanced chemistry is essential in order for proper enzyme activations to occur, shifts in the location of these minerals, etc. can be expected depending on the conditions. Calcium, for example, has been shown to enter the intracellular compartment in an effort to maintain ionic balance when magnesium is deficient, resulting in calcinosis (calcification) of the cell with consequences such as hypertension. Alterations in chloride levels can also affect cell membrane permeability, resulting in reductions in passive transport across the membrane. These and other similar changes can have profound effects on the physiology and biochemistry of the body.

-acidification of the urine (LOW URINE pH) reflects the removal of acidic elements from the blood with an overall acidic body chemistry. With a true metabolic acidosis, there is a compensatory acceleration of the respiratory rate producing a respiratory alkalosis. The lungs try to blow off CO2, producing a lowering of carbonic acid levels, which then produces an alkaline saliva pH. On the other hand, a low urine pH can be the result of a respiratory acidosis with a similar but opposite mechanism. The urine is again acid due to the loss of hydrogen ions and retention of bicarbonate and sodium, reflecting the compensatory alkalinization of the blood; but the saliva, in distinction to a metabolic acidosis, is also acid due to the increased levels of CO2 and carbonic acid. With metabolic acidosis, the high level of urine acidity is associated with a decrease in stomach acid production which can impair the process of digestion and result in relative malabsorption. The same reduction in gastric acid occurs with respiratory alkalosis, in spite of an alkaline urine with this condition, suggesting an indirect correlation between blood pH and stomach acid production i.e. as the overall acidic level rises, hypochlorhydria and consequent relative malabsorbtion become more of a problem.

-LOW SODIUM (Na) levels can impair the bicarbonate buffering system which normally functions to neutralize metabolic acids. Fluctuations in electrolytes (sodium) can occur in response to biochemical stresses attempting to correct a chemical imbalance. Low levels of sodium associated with acidosis occur in conditions of adrenal insufficiency due to decreased retention, and progesterone deficiency due to failure of aldosterone production. Aldosterone, a mineralocorticoid steroid hormone produced from progesterone within the adrenal cortex, is one of the main regulators of sodium status. Secretion of aldosterone prevents sodium excretion by the kidneys, and represents one of the primary mechanisms by which the acid buffering occurs. As sodium is retained , either potassium or hydrogen is excreted in exchange, depending on the predominance of either ion. The higher the serum acid level rises, the more likely hydrogen becomes the preferred choice for exchange, unless potassium levels become markedly elevated which is unlikely without hemolysis. Thus in the presence of acidosis, as adrenal stimulation occurs, sodium is retained and hydrogen is excreted so that the urine becomes more acidic. On the other hand, as the adrenals become progressively fatigued, this process begins to fail leading to losses of sodium in the urine and rising levels of acidity. Of course, a deficiency of sodium can also result from inadequete salt intake or salt containing foods in the diet.

-LOW MAGNESIUM (Mg), an alkaline mineral and necessary co-factor in glycolysis pathways as well as hormone utilization, generally occurs with the american diet, due to problems with soil depletion and absorption. The average intake of 300 mg/day with a 2000 calorie diet, falls below even USRDA requirements (400mg/d). In acidotic conditions, Mg is called upon to act as a buffer, forcing it to move out of it's normal intracellular location to extracellular spaces, where increased urinary loss lowers plasma levels of this mineral. This creates an intracellular deficiency of Mg, further compounded by deficient serum levels, causing calcium to move inside the cell (calcinosis) and elevating the blood pressure (BP). Hypomagnesemia can produce life- threatening heart rhythm irregularities, muscle stiffness, osteoporosis and constipation. Mg replacement corrects BP, bone loss, cell toxicity and cardiac arrythmias (irregularity). Mg is the fourth most abundant cation in the body, and is second only to potassium intracellularly. The intracellular concentration of magnesium is 10 times greater than extracellular. The total body content of magnesium is about 24 grams, 50% is present in bone and 50% in soft tissue.

-a LOW URINE pH can develop from the buildup of acid toxins within the body as well as excessive intake of acid producing foods. Acidification of the urine may also result from alterations in acid/alkaline equilibrium as a compensatory adjustment, such as occurs with respiratory acidosis (hypoventilation). Elevated urine acidity levels are associated with a decrease in the production of stomach acid which can impair the process of digestion and assimilation of essential nutrients (relative malabsorption).

INDIVIDUAL LABORATORY RESULTS:

This sub-section lists each of the individual basic panel lab tests that fall outside of the optimal range, and provides a more detailed description of the test. Possible causes or implications of each test's variance from optimal range are presented.

LOW BUN levels suggest poor utilization of amino acids for protein synthesis, low protein/high carbohydrate diets, malabsorption, fasting liver disease and/or overactivity of the anterior pituitary gland.

LOW SODIUM: Sodium is an electrolyte regulated by kidney and adrenal activity. It is an important component of the blood buffering mechanism. Failure to maintain adequate sodium levels may be due to adrenal fatigue, malabsorption, kidney and metabolic disorders, diabetes, diarrhea, excessive sweating diuretic usage and inadequate dietary sources. A sodium deficiency can predispose one to metabolic acidosis due to a deficit in alkaline buffering capacity.

HIGH T3 UPTAKE: An elevated T3 Uptake indicates inadequete cleavage of active thyroid hormone (T3) from thyroxine (T4) at the tissue level. This finding is consistent with clinical hypothyroidism due to underactivity of the gland, as a primary condition of the gland itself; as a secondary consequence of pituitary fatigue; or due to blocked expression of thyroid hormone at the tissue level resulting from mineral or other hormone imbalances, stress or toxicity (tertiary hypothyroidism). In the medical literature this latter condition is referred to as "peripheral resistance to thyroid hormone," or decreased peripheral T3 production due to reduced T4 to T3 conversion. Elevated iodine levels from a high intake of iodized salt, for example, can suppress the cleavage of T4 to T3, since elevated iodine levels tend to block the reaction from progressing. The T3 Uptake reflects the " percent" of available free T3 (active hormone) receptors for absorption on thyroid binding globulin compared to the population at large used as a control. It is performed by mixing radioactive T3 with patient serum. Both T4 and T3 are allowed to compete for binding sites on TBG as well as an added hormone binding resin. If the TBG and/or T4 level is low, the percent of radioactive T3 resin uptake will be elevated compared to the controls. A decreased TBG produces a decrease in T4, and a consequent increase in the T3 resin uptake. To avoid confusion, the T3 Uptake is now referred to as thyroid hormone-binding ratio (THBR). A high reading generally indicates undersaturation of the receptors, a high level of available T3 receptors, and a deficiency in the active T3 hormone level. The T4 may be normal or low. AN ACTUAL T3 HORMONE TEST IS RECOMMENDED TO CLARIFY IF THIS FINDING IS CONSISTENT WITH HYPOTHYROIDISM.

pp. 335-40 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

pp. 528-29 Wallach, Jaques B. *Interpretation of diagnostic tests* (6th ed.). Boston: Little, Brown, 1996.

pp. 476-78 Ravel M.D., Richard *Clinical Laboratory Medicine* (6th ed.). St. Louis: Mosby, 1995.

LOW TSH levels indicate there is an undersecretion of Thyroid Stimulating Hormone from the anterior pituitary gland. This can result from either an adequate or an excessive amount of thyroxine (T4) coming from the thyroid causing negative feedback suppression of the master gland. Overmedication with a thyroid hormone supplement can also produce this effect. A low TSH coupled with a low T4 would suggest secondary hypothyroidism indicating the thyroid problem is stemming from an underactive anterior pituitary. A low TSH with a high T4 suggests primary hyperthyroidism meaning the problem is coming from the thyroid gland itself. Treatment must be directed to the source of the problem.

a LOW PROGESTERONE level can result from blockages in the steroid production pathway leading to progesterone, due to metallic toxins (lead, mercury, etc.), vitamin (B6) or mineral (Mg.) deficiency, stress and acidic chemical imbalance. This common hormone imbalance, which results in many of the symptoms of the PMS Syndrome, is best detected by testing on day 20-25 in a normal 28 day cycle. Low levels naturally occur in the early half of the cycle and in the menopause. Only natural progesterone (1-25 di-hydroxyp.) has been shown to provide a protective factor against breast cancer, whereas synthetic progestens such as provera do not offer the same protection. This is especially true when a high estrogen level tends to persist, which is commonly associated with this deficiency.

A LOW MAGNESIUM level may lead to a defect in hormone utilization, nervous system irritability and peripheral vasodilation. This can be seen in hyperthyroidism, hyperadrenalism, parathyroid disorders, intestinal malabsorption, and certain renal and bone diseases. Magnesium deficiencies are common with American dietary habits since the average diet contains (300mg/day) less than the USRDA (400mg/day).

A LOW IRON level may be due to a deficiency of dietary iron, blood loss or chronic anemia, nephrosis, pernicious anemia and/or intestinal malabsorption. Supplementation is indicated especially with the elderly or in cases of relative malabsorption. Excessive loss of iron in the urine and/or hair can have a negative impact on mineral status.

FERRITIN (IRON CARRYING PROTEIN) is the transport protein for iron in the blood. If low, this can cause iron deficiency leading to anemia, if high, poor saturation due to mineral depletion.

An increased platelet count (HIGH PLATELETS), collectively known as thrombocytosis, can occur due to a benign reactive process, associated secondarily with other conditions including drug reactions, or as part of a myeloproliferative process such as leukemia. Benign causes include response to exercise, pregnancy, prematurity, and vitamin E deficiency. These are usually associated with modest elevations above the physiologic upper limit of the lab range. Associated conditions producing more marked elevations in platelet count include acute infections or inflammatory disorders, osteoporosis, cardiac disease, diabetes, renal (kidney) failure, and seizure disorders. Myeloproliferative

conditions generally stem from bone marrow production, and are often found to be associated with abnormal levels of arachidonic acid (a fatty acid) which can come from dietary sources.

pp. 709-710 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

HIGH SEGS: An elevation in segmental WBC may be a result of an acute infection, certain drugs and chemicals, emotional stress, menstruation, tissue damage, leukemia, and/or other blood disorders.

LOW MONOCYTES can be seen in conditions where monocyte production is impaired, such as with states of immune depletion (AIDS, viral infection, leukemia and exposure to chemicals or steroid hormones). Since a large percentage of the monocyte population is produced within the GI (Gastrointestinal) system, disorders that compromise GI function can have a detrimental effect on the monocyte count.

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

LOW EOSINOPHILS can occur in any situation of acute stress where there is an increase in the secretion of adrenal hormones (cortisol and/or adrenalin), and in conditions associated with hyperadrenalism such as Cushings. Acute inflammatory states can also produce this effect due to eosinophil migration into involved sites. Suppression of eosinophil production may result from exposure to medications containing Atropine or Atropine derivatives, such as the Belladonna alkaloids and several anti-cholinergic preparations commonly used for abdominal spasm (pain).

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

LOW BASOPHILS can occur in any situation of acute stress or infection where there is an acute increase in the secretion of adrenal hormones (cortisol and/or adrenalin), and in chronic conditions associated with hyperadrenalism such as Cushing's disease and metabolic acidosis. About half of hyperthyroidism cases develop a basopenia. Basophil levels show a diurnal variation, with the lowest occuring in the morning and the highest at night.

pp. 670 Henry, John Bernard. *Clinical diagnosis and management by laboratory methods* (19th ed.). Philadelphia: Saunders, 1996.

pp. 295, 297-298 Wallach, Jaques B. *Interpretation of diagnostic tests* (6th ed.). Boston: Little, Brown, 1996.

LOW URINARY PH: Acidification of the urine may reflect a metabolic acidosis or a respiratory acidosis (hypoventilation), excessive intake of acid producing foods or food drugs like sugar, diarrhea, dehydration, malabsorption, a disturbance of the adrenal glands and the usage of certain drugs. A high level of acid in the urine (low urinary pH) has been shown to be associated with a decrease in stomach acid production (HCL deficiency).

HIGH HOMOCYSTEINE levels indicate an increased risk for cardiovascular disease (CVD). Homocysteine is now considered to be a more important marker for cardiac risk than even cholesterol, and high levels are associated with a 2-4 fold risk of acute myocardial infarction (heart attack). Elevated homocysteine levels have also been shown to correlate with the development of carotid-artery stenosis. In distinction to the commonly held belief that cardiac disease primarily results from an abnormality in fat metabloism, homocysteine is actually a protein amino acid, and not even a fat. Since high homocysteine levels result from the incomplete metabolism of the essential amino acid methionine, an amino acid analysis may also be helpful in determining the most appropriate therapy for this condition. Elevated homocysteine levels are also considered to be a sensitive indicator of pyridoxine (Vit. B6), cobalamin (Vit. B12) and Folate deficiency. This is because vitamin B6 is required to convert homocysteine into cysteine, and B12 is a necessary co-enzyme in the conversion of homocysteine back to methionine if needed to reduce homocysteine levels. When either of these vitamins are deficient, homocysteine levels will increase in both serum and urine. Vitamin B12 deficiency, a relatively common problem due to impaired absorption from dietary sources especially in the elderly, causes a megaloblastic anemia to develop which is characterized by the enlargement of early developing red cells (megaloblasts) and an elevation of the MCV (mean corpuscular volume) measurement. Folic acid (FA) or Folate deficiency can result from inadequate intake or improper absorption of this important water soluble B vitamin. Humans with nutritional folate deficiency also develop homocysteine elevations. The blood and bone marrow features of megaloblastic anemia are similar to those seen with cobalamin or vitamin B12 deficiency, and many authors feel the anemia of B12 deficiency is really due to a tissue folate deficiency. Recent reports indicate an elevated neutrophilic hypersegmentation index (NHI) may also be helpful as an early indicator of B12 deficiency and a developing megaloblastic anemia.

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END OF REPORT