Practical application of Functional & Integrated Practic in Psychology & Psychiatry (With particular consideration to conditions related to infections by Mold (Mycotoxins), Long COVID and Mitochondrial Dysfunction).

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Common Presentations

•Depression and Bipolar Disorder

•<u>Anxiety</u>

•Attention, processing and cognitive deficit disorders

Addiction

• Eating Disorders

Mood disturbance

•Obsesive Compulsive Disorder

•Alzheimer's and other dementias

•<u>Many others...</u>

•Less common: Psychosis, Schizophrenia, Mania etc

Causes of Psychiatric Conditions:

(for Dummies!)

Psychological - Reactive ("My cat has died...")

Can develop into

Physical -

Endogenous (Brain dysfunction)

- ✤ Any cause of neuroinflammation
- Nutritional deficiencies
- ✤ Brain Trauma

Can be both....

Neuroinflammation (inc. Oxidative stress) as a cause of psychiatric issues

Condition	No. of papers found in Google Scholar
Depression	142,000
Anxiety	77,000
Mood disorder	52,000
Addiction	22,400
Eating disorders	22,600
► OCD	9,040
► ADHD	11,700
Alzheimer's	307,000
Psychosis	28,6000

Areas & Systems Affected

As examples:

Limbic-cortical dysregulation - **Depression**

Prefrontal cortex, hippocampus, anterior cingulate, amygdala - Mania

Frontal-subcortical, right caudate - OCD

Atypical *serotonin* system, right frontal and right caudate dysfunction, mesolimbic *dopamine* pathways - **Eating disorders**

Sympathetic and Parasympathetic ANS, *adrenalin*, *noradrenalin*, *GABA etc* - all conditions

Psychology of Home, Work and School Environs

Poverty

High stress in your specific role Imbalance of effort vs. rewards Low social support in the workplace Not having control over your job Feeling connected and safe Lack of access to instruction Unclear or unfocused objectives Experiencing bullying Low relational and procedural justice Not feeling valued or respected Not having a sense of belonging Absence of a support system Bosses and peers not understanding

Environmental Factors Affecting Mental Health

- **Climate** warmth, sunlight, cloud & rain, length of daylight, increased frequency of disasters contributing to the development of depression, adjustment disorder, and post-traumatic stress disorder.
- **Crime levels**. affecting females more strongly, increasing their risk of depression and anxiety.
- Stigma racism, sexism, sexual orientation, supporting Spurs etc.
- **Pollution** increased rates of depression and anxiety in more polluted areas.

Environmental Toxins

- Sleep deprivation
- Smoking
- Substance abuse
- Pollution
- Exposure to toxins through off-gassing (glues, sealants, pesticides, fire retardant, VOC from computers, cleaning products, mold etc.
- Hazardous conditions at work

References (EXCLUDING EVIDENCE OF SUPPORTING THE WRONG FOOTBALL TEAM)

Home Environment

- Padhy SK, Sarkar S, Panigrahi M, Paul S. <u>Mental</u> <u>health effects of climate change</u>. Indian J Occup Environ Med. 2015;19(1):3-7. doi:10.4103/0019-5278.156997
- Dustmann C, Fasani F. <u>The effect of local area crime</u> on mental health. *Econom J*. 2014;126(593):978-1017. doi:10.1111/ecoj.12205
- Washington HA. <u>A terrible thing to waste:</u> <u>Environmental racism and its assault on the</u> <u>American mind</u>.
- Braithwaite I, et al <u>Air pollution (particulate matter)</u> exposure and associations with depression, anxiety, bipolar, psychosis and suicide risk: A systematic review and meta-analysis. Environ Health Perspect. 2019;127(12):126002. doi:10.1289/EHP4595
- Bornschein et al <u>Psychological Medicine</u>, <u>Volume</u> <u>32</u>, <u>Issue 8</u>, November 2002, pp. 1387 - 1394
- DOI: https://doi.org/10.1017/S0033291702006554

Work and School | Environs

- Knifton L, Inglis G. Poverty and mental health: policy, practice and research implications. BJPsych Bulletin. 2020;44(5):193-196. doi:10.1192/bjb.2020.78
- Harvey SB, Modini M, Joyce S, et al. <u>Can work</u> <u>make you mentally ill? A systematic meta-</u> <u>review of work-related risk factors for</u> <u>common mental health problems</u>. *Occup Environ Med*. 2017;74(4):301-310. doi:10.1136/oemed-2016-104015
- Schulte-Körne G. <u>Mental health problems in a school setting in children and adolescents</u>. *Dtsch Arztebl Int*. 2016;113(11):183-190. doi:10.3238/arztebl.2016.0183

Overall causes of neuroinflammation

- Infection
- Toxicity
- Autoimmune disease.
- Inflammation from peripheral organs.
- Directly from mental stress
- Metabolic disorders
- Poor lifestyle.

Sun Y, Koyama Y, Shimada S. Inflammation From Peripheral Organs to the Brain: How Does Systemic Inflammation Cause Neuroinflammation? Front Aging Neurosci. 2022 Jun 16;14:903455. doi: 10.3389/fnagi.2022.903455. PMID: 35783147; PMCID: PMC9244793.

Infective causes of neuroinflammation 1. Viral

Chronic viral (HIV, EBV, CMV, Coxasckie, various Herpes viruses, SARS CoV-2 and no doubt many others)

"Chronic Viral Neuroinflammation: Speculation on Underlying Mechanisms"

- Foci of cytotoxic inflammation.
- Human immunodeficiency virus (HIV)-associated neurocognitive disorders.
- Macrophages in the CNS spur development of encephalitis (HIVE).
- Continual activation of astrocytes drive neurocognitive disorders/subclinical disease, and <u>neuroinflammation</u>.
- Activation of glial cells.
- CNS innate immune system is distinct from the rest of the body, there could be a number of activation profiles not observed elsewhere.

Delery EC, MacLean AG. Chronic Viral Neuroinflammation: Speculation on Underlying Mechanisms. Viral Immunol. 2019 Jan/Feb;32(1):55-62. doi: 10.1089/vim.2018.0093. Epub 2018 Sep 27. PMID: 30260764; PMCID: PMC6350055.

The causes of neuroinflammation 2. Mold & mycotoxins

Google scholar - 4,730 papers

Here's one regarding Mycotoxins & Psychiatry with Mold as a cause :

"Brain inflammation in the hippocampus, the area of the brain that governs memory, learning, and the sleep-wake cycle.

Decreased neurogenesis, or the formation of new brain cells. Impaired memory. Increased sensitivity to pain. Increased anxiety"

Cheryl F. Harding, et al Mold inhalation causes innate immune activation, neural, cognitive and emotional dysfunction. Brain, Behavior, and Immunity, Volume 87, 2020, Pages 218-228, ISSN 0889-1591, https://doi.org/10.1016/j.bbi.2019.11.006

The causes of neuroinflammation – 3. Bacterial or Parasitic

Rarely presents except in A&E as much more aggressive requiring acute, emergency treatment

Inflammation from Peripheral Organs. - The Gut

Google Scholar - 15,700 papers Neuroinflammation + microbiome + psychiatry

Google Scholar - 61,100 papers Microbiome + psychiatry

1. "Stress-induced enteric dysbiosis and intestinal permeability [leaky gut] confer risk for negative mental health outcomes through immunoregulatory, endocrinal, and neural pathways."

Liu, R. T. (2017). The microbiome as a novel paradigm in studying stress and mental health. *American Psychologist*, 72(7), 655-667. <u>https://doi.org/10.1037/amp0000058</u>

Common conventional view:

2. "There is **interest** among both the research and lay communities in understanding the effects of the microbiome on the brain."

G. MacQueen has received honoraria from Allergan, Pfizer, Lundbeck and Janssen. P. Moayyedi has accepted speaker fees from Allergan Inc. and Abbvie Pharmaceuticals

MacQueen G, Surette M, Moayyedi P. The gut microbiota and psychiatric illness. J Psychiatry Neurosci. 2017 Mar;42(2):75-77. doi: 10.1503/jpn.170028. PMID: 28245172; PMCID: PMC5373703. been on the advisory boards of Allergan Inc, Shire and Salix Pharmaceuticals.

Mitochondria in Psychiatry

Patients with mitochondrial disorders can present with primary psychiatric symptomatology, including mood disorder, cognitive impairment, psychosis, and anxiety."

The Psychiatric Manifestations of Mitochondrial Disorders: A Case and Review of the Literature. Rebecca E. Anglin, MD, FRCP(C); et al J Clin Psychiatry 2012;73(4):506-512

Google scholar 205,000 papers Mitochondria + Psychiatry

- Mitochondrial disease differs from disorder and dysfunction
- 4000-5000 Mito per cell.
- Catecholamines and cortisol directly affect Mitochondria as do infections, toxins and nutritional deficiencies

Therapeutic Approaches - Psychological

Meditation

Breathing - Yoga, Tai Chi, Qi Gong

Sleep training

Exercise

On line options such as 'The ThinQ Fitt training - a method of developing awareness of your thinking habits." <u>https://www.get-fitt.com/thinq-fitt-training/</u>

Refer for Counselling

Exercise Rx in Psychiatry (2.7 million papers on Google Scholar).

Roger Seheult, MD of MedCram - <u>https://www.youtube.com/watch?v=QevFo8wsXZ4</u> In depression

		Unadjusted model		Fully adjusted model*	_
	-	IRR (95% CI)	p value	IRR (95% CI)	p value
	Exposure at 12 years (n=2486)	~			
	Count per minute (per 100)	0.910(0.882-0.939)	<0.0001	0.941 0.910-0.972)	<0.0001
	Sedentary behaviour (per 60 min)	1.108 (1.054-1.165)	<0.0001	1.111 (1051-1.176)	<0.0001
d	Light activity (per 60 min)	0.883 (0.834-0.933)	<0.0001	0.9040.850-0.961)	0.0012
	Moderate-to-vigorous activity (per 15 mins)	0.848 (0.863-0.965)	<0.0001	0.910 (0.857-0.966)	0.0018
	Exposure at 14 years (n=1938)				
	Count per minute (per 100)	0.933 (0.902–0.965)	<0.0001	0.965 (0.932-0.999)	0.0443
	Sedentary behaviour (per 60 min)	1.114 (1.057–1.175)	<0.0001	1.080 (1.012–1.152)	0.0200
	Light activity (per 60 min)	0.908 (0.851-0.970)	0.0044	0.922 (0.857-0.992)	0.0299
	Moderate-to-vigorous activity (per 15 mins)	0.913 (0.863–0.965)	0.0409	0.960 (0.905-1.018)	0.1691
	Exposure at 16 years (n=1220)				
	Count per minute (per 100)	0.939 (0.896–0.983)	0.0072	0.984 0.937-1.033)	0.5092
	Sedentary behaviour (per 60 min)	1.101 (1.026–1.180)	0.0068	1.107 (1.015-1.208)	0.0210
4	Light activity (per 60 min)	0.882 (0.810-0.961)	0.0040	0.889 (0.809–0.974)	0.0133
	Moderate-to-vigorous activity (per 15 mins)	0.938 (0.883–0.997)	0.0413	1.001 (0.936–1.071)	0.9002
	Depression at 18 years of age was asses *Adjusted for sex, maternal social class and total accelerometer wear time at e	ssed with the Clinical Interv 5, parental psychiatric histo ach timepoint.	iew Schedule ry, parental e	e-Revised. IRR=incidence ra ducation, ethnicity, baselin	te ratio. e depression,
	Table 3: Longitudinal associations activity and sedentary behaviour a	between depression sco at 12 years, 14 years, an	ores at 18 ye d 16 years o	ears and different levels of age	of physical

Gizmos and Gadgets

The Sensate - a sound wave instrument that interplays with an App.

Used daily initially for a few minutes building up to longer spells as required.

Retrains the body's stress response in the same way meditation does.

Works by stimulating the Vagus nerves which control many functions including heart rate, blood pressure, breathing rate, the gut movement, and the psychological stress-response and indirectly hormonal balance.

Details can be read here www.getsensate.com

Therapeutic Approaches -Detoxification & Detoxicants

- Eliminate constipation and ensure regular bowel movements
- Stimulate all detox pathways
- Methylation (top of the list)
- Liposomal Glutathione
- Trans-Sulphuration
- Glucuronidation

Therapeutic Approaches - Pointers

Aim treatment at the following:

- Lifestyle changes
- Psychology
- Infection
- Detoxification
- Nutrition
- Neuronal and Nerve receptor repair
- Neurotransmitter activity

Therapeutic Approaches - Nutritional

- Essential Fatty acids varying authorities range balance between Omega3:Omega 6 as 1:1, 2:1 & 1:4 !
- Phosphatidylcholine
- Phosphatidylserine
- Amino acids Tyrosine, L-Theanine, 5-HTP or Tryptophan and several others
- Minerals especially magnesium
- Vitamins especially B6 & B12 but all those in B-Complex

Guidance from investigations helps narrow down requirements

Therapeutic Approaches -Immune stimulation

Aim at activating the innate immunity

(particularly if viral and mould infection is suspected)

- Keep the skin healthy
- Keep the gums healthy
- Avoid any foods 'that hurt' (i.e. cause heart burn, reflux, abdominal bloating) including too hot drinks, alcohol without food, NSAID drugs
- Antioxidants vitamin E, D &., vitamin C, β-carotene, selenium, copper, iron, and zinc
- Polyphenols (Curcumin especially, resveratrol, and sulforaphane)

Therapeutic Approaches -Immune stimulation.

- Monolaurine
- Beta-glucans
- Adaptogens
- > Rhodiola
- > Ashwaganda (Withania somnifera)
- Eleutherococcus senticosus Siberian ginseng root
- Schizandra chinensis berries

► ASPERGILLUS PROTOCOL

- Liposomal Glutathione
- Amino acid broad supplement (? Pea powder)
- Activated Charcoal start with minimum rising to maximum dose
- Bentonite Clay build up to max dose
- Zeolite
- Guggul gum build up to max on label
- N Acetyl cyteine 500 mgs bd
- High dose antioxidants

Covid - SARS CoV-2 & Psychiatric Affects

- SARS-CoV-2-human protein interactions may lead to the development of delirium, psychosis, seizures, encephalitis, stroke, sensory impairments, peripheral nerve diseases, and autoimmune disorders
- Yapici-Eser H, et al Neuropsychiatric Symptoms of COVID-19 Explained by SARS-CoV-2 Proteins' Mimicry of Human Protein Interactions. Front Hum Neurosci. 2021 Mar 23;15:656313. doi: 10.3389/fnhum.2021.656313. PMID: 33833673; PMCID: PMC8021734.
- Indirectly the effects of lockdown, social distancing, isolation, mask wearing.

Long Covid & Spike Proteins

Theories of causation of Long Covid include long term manufacture or maintenance *in situ* of Spike protein

"Mechanistically, we demonstrated that purified SARS-CoV-2 **spike glycoprotein** activated the NLRP3 inflammasome in LPS-primed microglia"

Albornoz EA, et al SARS-CoV-2 drives NLRP3 inflammasome activation in human microglia through spike protein. Mol Psychiatry. 2022 Nov 1. doi: 10.1038/s41380-022-01831-0. Epub ahead of print. PMID: 36316366.

Therefor this might indicate risk from mRNA vaccination production of Spike protein.

- The accumulation of SARS-CoV-2 spike protein in the skull-meninges-brain axis presents potential molecular mechanisms and therapeutic targets for neurological complications in long-COVID-19 patients.
- SARS-CoV-2 Spike Protein Accumulation in the Skull-Meninges-Brain Axis: Potential Implications for Long-Term Neurological Complications in post-COVID-19 Zhouyi Rong et al doi: https://doi.org/10.1101/2023.04.04.535604

(Other theories include: Chronic SARS CoV -2 infection, Mast Cell Activation, Permanent DNA alteration, Permanent metabolic damage and others.

Long Covid. The Front Line Covid-19 Critical Care Alliance

PREVENTION PROTOCOLS https://covid19criticalcare.com/treatment-protocols/i-recover-longcovid-treatment/

I-RECOVER [™] An Approach to Treating Long COVID

I-RECOVER [™]

Vaccine Injury

Long Covid & Chronic Viral Infections -First Line Treatments

- Intermittent daily fasting or periodic daily fasts
- Moderating physical activity
- Sunlight and Photobiomodulation (previously known as Low Level Laser Therapy -LLLT)
- Probiotics/prebiotics
- Nattokinase
- Aspirin
- Magnesium
- Methylene blue
- Resveratrol

Long Covid & Chronic Viral Infections -Second/Third Line Treatments

- Vitamin D (with Vitamin K2)
- N-acetyl cysteine
- ► Cardio Miracle[™] L-arginine/L-citrulline supplements
- Omega-3 fatty acids
- Nigella sativa
- Vitamin C & Intravenous Vitamin C
- Spermidine
- Mitochondrial therapy
- Non-invasive brain stimulation
- Behavioural modification, relaxation therapy, mindfulness therapy, and psychological support
- Hyperbaric oxygen therapy
- Low Magnitude Mechanical Stimulation

Long Covid & Chronic Viral Infections -Prescription only Treatments

First Line

- Ivermectin
- Melatonin
- Low-dose naltrexone

Second/third line

- Nicotine (2023 Jan 18;9(1):2. doi: 10.1186/s42234-023-00104-7)
- Sildenafil (Viagra) with or without L-arginine- L-Citrulline
- Hydroxychloroquine
- Low-dose corticosteroid

Special Mention Low Dose Naltrexone

Low Dose Naltrexone (LDN) is a low dose of a drug used conventionally for opiate addiction at doses of 50 mgs +.

LDN dosage 0.125mg - 4.5mgs (1/200th to 1/10th the conventional dose)

Mechanism based on a mild blockage of the body's own opiate receptors which causes the body to increase its production of natural opiates (Endorphins and encephalins).

Opiate receptors found throughout the body effecting nearly all systems in the body. <u>https://ldnresearchtrust.org/what-is-low-dose-naltrexone-ld</u>

Studies in Psychiatric and mood disorders are presented by the LDN Research Trust here: <u>https://ldnresearchtrust.org/sites/default/files/inline-files/Dr-Mark-Shukhman.pdf</u>

This peer reviewed paper discusses LDN's effects in mood disorder amongst many other complaints <u>https://www.naturalmedicinejournal.com/journal/2018-04/uses-low-dose-naltrexone-clinical-practice</u>

Tests & Investigations

Neuroinflammation – blood test through Lab 4 More Neurotransmitter – useful advice & for persuasion **Precision Point Advanced Intestinal Barrier** Organic Acid Test (OAT) – highlights mycotoxins, mitochondrial dysfunction and other Mycotoxin **OAT + Mycotox Combo** Viral Infections through Immunoscience Labs. **Cyrex Tests: Alzheimer's LINX Array 20 - Blood Brain Barrier Permeability** Array 14 - Mucosal Immune Reactivity Screen (saliva)

Neuro Basic Profile

Neuro Basic Profile; urine						<	LABR X
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Analyte	Result	Unit per Creatinine	L		WRI	н	Reference Interval
Serotonin	95.2	hð/ð			Z		50-98
Dopamine	164	µg/g					110-200
Norepinephrine	4.2	µg/g					18-42
Epinephrine	2.4	µg/g					1.3-7.3
Norepinephrine / Epinephrine ratio	1.8						< 12
Glutamate	21	µmol/g					9.0-40.0
Gamma-aminobutyrate (GABA)	1.7	µmol/g		Δ			1.6-3.5
Glycine	2543	µmol/g					350 - 1500
Histamine	24	µg/g					12-30
Phenethylamine (PEA)	36	nmol/g					26-70
Creatinine	40.7	mg/dL					35-240

Neurotransmitter Comments:

- Urinary neurotransmitter levels provide an overall assessment of the body's ability to make and break down neurotransmitters and are
 representative of whole body levels. They are required for neurotransmission throughout the body. Direct assessment of neurotransmitter levels
 and metabolism in the central nervous system is not clinically feasible and approximately twenty percent of the total urinary levels are derived
 from the brain. The enzymes, cofactors and precursors in neurotransmitter metabolism in general are the same in the periphery and in the
 central nervous system. Therefore, alterations in urinary neurotransmitter levels assessed in urine provide important clinical information, and may
 be associated with many symptoms including cognitive and mood concerns, diminished drive, fatigue and sleep difficulties, cravings, addictions
 and neurons.
- Upper range serotonin may be associated with symptoms of increased anxiety, agitation, and diarrhea (IBS-like symptoms). Serotonin levels
 may be increased by low protein or high-carbohydrate meals, insulin and tryptophan or 5-HTP supplementation. Many mood altering
 medications, including SSRIs and SNRIs. may influence serotonin levels. L-theanine may affect serotonin function.
- Low norepinephrine and low range epinephrine may be associated with depression and mood changes as well as fatigue, difficulty
 concentrating, decreased ability to stay focused on tasks and diminished sense of personal/professional drive. Norepinephrine is converted from
 dopamine requiring vitamin C, copper and niacin (B3). L+tyrosine, L+theanine and Mucuna pruriens influence this pathway.
- Low range GABA may be associated with anxiety, poor impulse control, depression, pain and decreased sleep quality. Low GABA may be seen
 in individuals deficient in vitamin B6. L-theanine, GABA and glutamine may positively affect functional GABA activity, and phenibut exerts GABAlike effects (experimental models).
- Glycine is a non-essential amino acid that acts as an inhibitory neurotransmitter in the central nervous system. Elevated glycine levels may be
 associated with compromised cognitive processing. Elevated levels may be seen with glycine supplementation. Glycine may be given in
 conjunction with pharmaceutical agents when supporting schizophrenia or psychosis. Lipoic acid may enhance glycine break down. Break down
 of glycine requires vitamin B6 and tetrahydrofolate as cofactors. Note: High levels of glycine may interact with clozapine and decrease its clinical
 efficacy.
 Considerations to address the demonstrated imbalances bevond the identified co-factors and amino acid precursors may include dosace
- Considerations to address the demonstrated imbalances beyond the identified co-factors and amino acid precursors may include dosage
 adjustments if indicated, as well as nervine and adaptogenic herbs, methylation support, vitamin D, and gastrointestinal health optimization.

Notes: Results are creatinine corrected to account for urine dilution variations. Creatinine is not meant to be used as an indicator of renal function. RI = Reference Interval, L (blue) = Low (below RI), WRI (green)= Within RI (optimal), WRI (yellow)= Within RI (not optimal), H (red)= High (above RI) Methodology: LCMS QQQ.creatinine by Jaffe Reaction

Analyzed by DOCTOR'S DATA, INC. • 3755 Illinois Avenue, St. Charles, IL 60174-2420 USA • LAB DIR: Erlo Roth, MD • CLIA ID: 14D0646470

Neuroinflammation Profile

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Laboratory diagnostics carried out and validated by MVZ Labor Bavariahaus, in the case of individual parameters by the authorised partner laboratory, where applicable

General Information:

Interleukin 1ß

InterreukIn 16 IL-16 - together with IL-6 and TNF-alpha – is the major proinflammatory cytokine, produced mainly by monocytes/macrophages but also by many other cells like vascular endothelia, epithelial cells, fat and nerve cells. Most of the time IL-18 is released in the initial phase of inflammatory reactions together with TNF -alpha and IL-6, with TNF-alpha being the main cytokine of the reaction.

IL-1ß is produced and released together with its analogue IL1 -alpha. The two IL1 variants have a very similar activity profile but IL 1B is generally the more active and more potent variant. The major functions of IL -1B are activation of the cellular immune response (T cells), stimulation of TNF -alpha and IL 6 release, increased secretion of stress hormones (CRH, Cortisol) and induction of central reactions like fever, pain, metabolism (insulin secretion), cognitive performance, etc. Its inflammatory si gnals generate prostaglandins via induction of cyclooxygenase 2, stimulate phospholipid breakdown and increase the production of inflammatory NO. All these effects are mediated by NF -kB.

OAT - Organic Oat Test

The Great Plains Laboratory, Inc. William Shaw, Ph.D., Director Fax (913) 341-6207 11813 West 77th Street, Lenexa, KS 66214 (913) 341-8949 Requisition #: Physician. Patient Name Date of Collection: 40 07:30 AM Patient Age Time of Collection Patient Sex: F Print Date: 04/18/2019 **Organic Acids Test - Nutritional and Metabolic Profile** A Metabolic Markers in Urine Reference Range Patient Reference Population - Females Age 13 and Over (mmol/mol creatinine) Value Intestinal Microbial Overgrowth Yeast and Fungal Markers 1 Citramalic ≤ 3.6 H 3.7 ≤ 14 31> 2 5-Hydroxymethyl-2-furoic H 31 3 3-Oxoglutaric ≤ 0.33 H 3.6 3.6 4 Furan-2,5-dicarboxylic (Aspergillus) ≤ 16 15 15 H 5 Furancarbonylglycine (Aspergillus) ≤ 1.9 1.1 <1.1 6 Tartaric (Aspergillus) ≤ 4.5 3.7 7 Arabinose ≤ 29 н 167 (167) 8 Carboxycitric ≤ **29** 0.46 44 9 Tricarballylic ≤ 0.44 H 0.45 Bacterial Markers 10 Hippuric ≤ 613 н 615 -615 11 2-Hydroxyphenylacetic 0.06 - 0.66 0.27 020 12 4-Hydroxybenzoic ≤ 1.3 0.29 13 4-Hydroxyhippuric 0.79 - 17 8.8 14 DHPPA (Beneficial Bacteria) ≤ 0.38 H 0.61 4.60 Clostridia Bacterial Markers 15 4-Hydroxyphenylacetic (C. difficile, C. stricklandii, C. Im 5.1 ≤ 19 <5.1> 16 HPHPA (C. sporogen 26 ≤ 208 . C. caloritolerans. C. botulinum & 17 4-Cresol ≤ 75 6.0 -(6.0> 0.28 18 3-Indoleacetic ≤ 11 (C. stricklandli, C. lituseburense, C. subterminale & others) 12

Hepatic Detox Profile

0001672



LAB #: U000000-0000-0 PATIENT: Sample Patient ID: PATIENT-S-10000 SEX: Male AGE: 51 CLIENT #: 12345 DOCTOR: Doctor's Data, Inc. 3755 Illinois Ave. St. Charles, IL 60174

Hepatic Detox Profile; Urine

TOXIC EXPOSURE MARKERS									
	RE	RESULT		REFERENCE		PERCENTILE			
	per	creatinine	INTER	VAL	2.5 th	16 th	50 th	84 th	97.5 th
D-Glucaric Acid (Phase I)	430	nM/mg	25-	300			_		
Mercapturic Acids (Phase II)	67	μM/mM	36-	90			_		
URINE CREATININE									
			DEEED	ENIOE					

	RESULT mg/dL	REFERENCE	-2SD	-1SD	MEAN	+1SD +2SD	
Creatinine	113	45- 225			-		
	INFORM	IATION					

The human body attempts to eliminate xenobiotics (foreign organic chemicals) through a concerted effort of enzymatic "functionalization" (phase I) and conjugation (phase II). Functionalization involves chemical modification of the xenobiotic by the cytochrome P-450 or the "mixed function oxidase" enzyme systems. Once functionalized, the altered xenobiotic can then be conjugated and excreted. Urinary D-glucaric acid, a hepatic byproduct of enzymatic response to chemical toxins (phase I), is a reliable indicator of exposure to xenobiotics. Mercapturic acids are direct, excretory end products of the functionalized xenobiotics that have been conjugated with glutathione prior to excretion. Together, the urinary levels of these metabolites provide valuable information about exposure to xenobiotics, liver disease, and quantitative assessment of the status of hepatic phase II detoxification.

D-GLUCARIC ACID ELEVATED: The level of D-glucaric acid, a marker of exposure to hepatotoxic substances, is abnormally high for age and gender in this sample. The results are consistent with clinically significant exposure to xenobiotics and enhanced phase I detoxification. Check mercapturic acid levels to evaluate the status of phase II detoxification that is required for the final elimination of the toxin(s). Severe xenobiotic exposure with markedly elevated D-glucaric acid levels (>3X normal) may be associated with impaired chemical functionalization or limited phase II activity. Elevated urinary excretion of D-glucaric acid is an indication of induction of cytochrome P-450 enzymes (phase I) in the liver that may be the result of exposure to any of over 200 different xenobiotics (e.g. pesticides, herbicides, fungicides, petrochemicals, drugs, alcohol, toluene, xylene, formaldehyde, sytrenes, ibuprofen etc.). Occupational and environmental exposure to toxic compounds causes induction of the glucuronic acid enzyme pathway and production of D-glucaric acid, thus D-glucaric acid excretion is considered an indirect by-product of detoxification reactions. Elevated levels of urinary D-glucaric acid have been correlated with viral hepatitis and jaundice, and have also been found in patients receiving antirheumatic drugs, independent of disease activity. With elevated levels of D-glucaric acid, there is an increased need for antixidant protection because toxins that are processed through phase I generate free radicals that require quenching or neutralization. It is important to consider that phase I detoxification tends to become less active with aging.

MERCAPTURIC ACIDS MARGINALLY ELEVATED: The levels of mercapturic acids (MA) in this patient's urine sample are marginally elevated for age and gender, and may be consistent with mild exposure to xenobiotics and enhanced detoxification via glutathione conjugation (phase II). Check for elevated levels of D-glucaric acid as an indicator of xenobiotic exposure. MA are final excretory products of detoxification and include a variety of functionalized xenobiotics that have been conjugated with cysteine, or glutathione. Ideally, urinary levels of MA should be increased with exposure to xenobiotics and enhanced phase I detoxification; MA levels will gradually return to basal levels commensurate with successful hepatic detoxification and removal of the patient from the source of exposure. If warranted, detoxification should be supported with supplemental vitamins C, E, and lipoic acid, selenium, Mg, K, rGSH, and sulfur containing amino acids. It should be noted that falsely elevated levels of MA can occur in patients with cystinuria, or with the use of thiol chelators (D-penciallamine, DMSA and DMPS), and some 'thio-acpto' type medications (e.g. thioridazine, captodiamine).

	SPECIMEN DATA
Comments:	
Date Collected: 11/17/2011	Methodology:
Date Received: 11/21/2011	D-Glucaric: HPLC
Date Completed: 12/5/2011	Mercapturic: Enyzmatic

@DOCTOR'S DATA, INC. • ADDRESS: 3755 Illinois Avenue, St. Charles, IL 60174-2420 • CLIA ID NO: 14D0646470 • MEDICARE PROVIDER NO: 148453

Blood Brain Barrier



2602 S. 24th Street . Phoenix, AZ 85034 Tel 602 759 1245 . Fax 602 759 8331 . www.CyrexLabs.com

PRACTITIONER

DEMODR, DEMODR TEST 2602 S. 24th Street Phoenix, Arizona 85034 ACCESSION #: 17-108497 C REQUISITION #: T05170963 C SAMPLE TYPE: Serum C DOCTOR / PATIENT ID: PAGES: 1 of 1

DATE COLLECTED: 5/11/2017 DATE RECEIVED: 5/11/2017 DATE OF REPORT: 7/11/2017

PATIENT

Name: REPORT, SAMPLE DOB: 11/01/1990 Gender: M

TEST	RESULT			
Array 20 - Blood Brain Barrier Permeability Screen	IN RANGE (Normal)	EQUIVOCAL*	OUT OF RANGE	REFERENCE (ELISA Index)
Blood Brain Barrier Protein IgG+IgA			2.53	0.3-2.2
Blood Brain Barrier Protein IgM		1.75		0.3-2.2

* Reference ranges are calculated based on the mean ±2 standard deviations (SD). Results > 1 SD, and <2 SDs above the mean are considered to be equivocal. An equivocal result represents the range between negative and suspicious low positive results. Results >2 SDs are considered out of range, and positive.

Mark G. Kartub, M.D., Medical Director

Cyrex Laboratories is certified under the Clinical Laboratory Improvement Amendments of 1988 ("CLIA") as qualified to perform high-complexity clinical testing. Test result data on its own does not constitute a diagnosis. Only a physician or qualified healthcare professional should interpret the significance of a clinical lab test or make a diagnosis. This test was developed and its performance characteristics determined by Cyrex Laboratories, LLC. The names and titles of tests and arrays are for reference purposes only.



Sample Report

Advanced Intestinal Barrier Assessment



2602 S. 24th Street . Phoenix, AZ 85034 Tel 602 759 1245 . Fax 602 759 8331 . www.CyrexLabs.com ACCESSION #: 19-SAMPL DATE COLLECTED: 5/15/2019 REQUISITION #: TSAMPL DATE RECEIVED: 5/17/2019 SAMPLE TYPE: Serum DATE OF REPORT: 5/24/2019 DOCTOR / PATIENT ID: PAGES: 1 of 2



SAMPLE, DOCTOR 2602 S. 24th Street Phoenix, Arizona 85034

(PATIENT)

Name: SAMPLE, PATIENT DOB: 01/01/1971 Gender: F

TEST		RI	ESULT	
Alzheimer's LINX™ - Alzheimer's-Associated Immune Reactivity **	IN RANGE (Normal)	EQUIVOCAL*	OUT OF RANGE	REFERENCE (ELISA Index)
Brain Proteins				
Tau Protein	0.41			0.0-1.2
Amyloid-Beta Peptide		1.21		0.1-1.4
Rabaptin-5 + Presenilin			1.48	0.0-1.4
Alpha-Synuclein		1.10		0.3-1.3
Growth Factors				
Beta Nerve Growth Factor	1.11			0.3-1.5
Brain Derived Neurotrophic Factor	0.68			0.1-1.2
Neurotrophins	0.32			0.3-1.6
Somatotropin	0.85			0.3-1.4
Enteric Nerve, Enzymes and Neurological Peptides				
Enteric Nerve + Vasoactive Intestinal Peptide	0.36			0.0-1.0
Transglutaminases		1.38		0.4-1.5
Pathogens				
Oral Pathogens	1.11			0.2-1.6
Enterococcus faecalis	0.96			0.5-1.6
Escherichia coli CDT + Salmonella CDT	0.80			0.2-1.3
Campylobacter jejuni CDT	0.52			0.1-1.5
Herpes Type-1	0.45			0.0-1.5
Chemicals				
Aluminums	0.36			0.0-1.3
Dinitrophenyl	0.80			0.3-1.4
Ethyl + Methyl Mercury	0.49			0.2-1.1
Phthalates			2.99	0.2-1.2
Foods Cross-Reactive to Amyloid Beta				
Egg Yolk, Raw + Cooked			1.41	0.0-1.4

Download your Patient Educational Guide at https://www.cyrexiabs.com/docs/linx-peg.pdf

** For details on the method of cooking, please see specification sheets. All analytes are tested for IgG .

Long Covid Test

Immunoscience Labs: IgG - SARS CoV-2

Bruce Patterson - IncellKINE, IncellDx, Inc - £1,000 + test) using the following analytes: TNF-α, IL-4, IL-13, IL-2, GM-CSF, sCD40L, CCL5 (RANTES), CCL3 (MIP-1α), IL-6, IL-10, IFN-γ, VEGF, IL-8, and CCL4 (MIP-1β)

https://www.frontiersin.org/articles/10.3389/fimmu.2021.700782/full

Viral Immunity



	TEST	RESULTS NORMAL ABNORMAL	REFERENCE RANGE	UNITS
	VIR	AL PANEL COMPREHENSIVE		
IgG HSV 1+	2 (HERPES 1+2)	1.50	<16.0	EU/mL
	RESULTS REPO CONSIDERED EQ PREVIOUS IMM TO HSV 1 AND	RTED AS <16 ARE CONSIDERE QUIVOCAL; EQUAL TO OR GRE UNOLOGIC EXPOSURE AND IMM /OR HSV 2.	D NEGATIVE; 16-19.9 AR ATER THAN 20 INDICATE UNOLOGICAL EXPERIENCE	E
IgM HSV 1+	2 (HERPES 1+2)	0.80	<0.9	INDEX
	RESULTS REPOR 0.9-1.09 ARE 1.1 ARE CONSI	RTED AS < 0.9 ARE CONSIDE CONSIDERED EQUIVOCAL; EQ IDERED POSITIVE.	RED NEGATIVE; JAL TO OR GREATER THAN	
IgG HHV-6	(HERPES TYPE-6)	0.80	<37.00	EU
	RESULTS REPOR LIMIT OF DETE RESULTS >37 M 6.	RTED AS <8 EU ARE CONSIDE SCTION AND FROM 8-37 ARE 0 MAY INDICATE AN IMMUNE RE	RED WITHIN THE LOWER CONSIDERED NEGATIVE. SPONSE AGAINST HERPES	
IgM HHV-6	(HERPES TYPE-6)	0.80	<24.00	EU
	RESULTS REPOP LOWER LIMIT C NEGATIVE. RES AGAINST HERPE	RTED AS <8 EU ARE CONSIDE OF DETECTION AND FROM 8-2: BULTS >24 MAY INDICATE AN 25 6.	RED WITHIN THE 4 ARE CONSIDERED IMMUNE RESPONSE	
	HUMAN HERPESV NEUROTROPHIC DISEASE KNOWD ARE INFECTED PATHWAY IS TH SYSTEM. THE V GLIAL CELLS, IMMUNE REACTI OF BOTH IGM A	VIRUS TYPE 6 (HHV-6) TYPE VIRUSES THAT CAUSE THE CO I AS ROSECIA. BY ACE 3, 90 BY HHV-6 VIA THE NASAL CO IE MAJOR ROUTE OF ENTRY IN IRUS PERSISTS IN A VARIE: FOR THE REST OF THE AFFL CON AGAINST HHV-6 RESULTS IND IGG ANTIBODIES.	A AND TYPE B ARE MMON CHILDHOOD 0-100% OF HUMANS WITY. THE OLFACTORY WTO THE NERVOUS YT OF CELLS, INCLUDING COTED PERSONS LIFE. IN THE PRODUCTION	
CONTINUED	ON NEXT PAGE			

Viral Immunity

VICTOR CONTRACTOR CONT	ERTSON BLVD., STE. 312 LES, CA 90035 557-1077 FAX: (310) 657-1053 munsci@gmail.com	REFERRING PHY ******** RESEARCH ********	SICIAN ******** *******	
PATIENT NAME				AGE SEX
SAMPLE, REPORT				37Y F
ACCESSION NO. D.O.B.	COLLECTION DATE	LOG-IN DATE	TEST DATE	REPORT DATE
AAAA37 08/11/1984	11/5/2021	12/21/2021	12/21/2021	12/21/2021
TEST	RESULT NORMAL AB	'S NORMAL	REFERENCE RANGE	UNITS
IGM CYTOMEGALOVIRUS	0.80		<0.9	ISR
IGG AND IG EQUIVOCAL.	M RESULTS REPORTE	ED AS 0.9-1.09	ARE CONSIDERED	
IgG RUBEOLA/MEASLES	0.85		<0.9	ISR
RESULTS RE 0.9 AND 1. SUPER-IMMU	PORTED AS <0.9 AF 9 ARE CONSIDERED NE.	RE CONSIDERED I IMMUNE AND >1	NON-IMMUNE, BETW .9 ARE CONSIDERE	JEEN D
IgM RUBEOLA/MEASLES	0.00		<0.9	ISR
RESULTS RE RESULTS RE DICATE CUR	PORTED AS 0.9-1.0 PORTED AS EQUAL T RENT OR RECENT IN	9 ARE CONSIDE O OR GREATER S IFECTION WITH 1	RED EQUIVOCAL, A FHAN 1.10 MAY IN MEASLES VIRUS.	ND -
IGG EPSTEIN-BARR VCA	0.00		<0.9	ISR
IgM EPSTEIN-BARR VCA	0.00		<0.9	ISR
IgG EARLY ANTIGEN	0.00		<0.9	ISR
IgG EB NUCLEAR ANTIGEN	0.00		<0.9	ISR
IgM EB NUCLEAR ANTIGEN	0.80		<0.9	INDEX
INTERPRETAT	FIONS OF SEROLOGI Patients EBV	C PATTERNS IN Status	EBV INFECTION	
AB Sus	ceptible Primar EBV	y Convalescer (3 mo.)	nt Past Reactiv	ated
VCA-IgM	- +	+ or -		
VCA-IgG	- +	+	+ +	
EA-D	-	+	- +	
EBNA-IGG		+ or -	+ +	
EBNA-IgM	-	+ or -	- +	
CONTINUED ON NEXT PAGE				

Thank you for listening

Dr Rajendra Sharma MB BCh BAO LRCP&S(Ire) MFHom www.drsharmadiagnostics.com Link to my book: https://tinyurl.com/45njyut2

