Dietrich Klinghardt, MD, PhD, is a practicing physician in Woodinville, Washington with a focus on the treatment of chronic neurological conditions such as Lyme disease, autism, and CFIDS. In the years that he has treated patients with chronic infections, he has observed that, for many, recovery is often elusive. Patients may plateau or find that their recovery is stalled. In other cases, patients may not succeed in their attempts to rid the body of a particular toxic or infectious burden, such as in patients with long-standing or therapy-resistant, late-stage Lyme disease.

In looking for possible explanations as to why some patients struggle more than others to regain their health, co-author Klinghardt has found a high correlation between patients with chronic Lyme disease and those with kryptopyrroluria (KPU), or more precisely hemopyrrollactamuria (HPU). The condition is alternatively known as the “mauve factor” or “malvaria.”

KPU may be an inherited condition, but it can also be induced by psychological trauma or chronic infections. Epigenetic influences such as intrauterine, birth, childhood, or transgenerational trauma may trigger KPU; other triggers may include a car accident, divorce or emotional trauma, and physical or sexual abuse. Chronic infections, such as Lyme disease, may themselves serve as a trigger for the condition.

The HPU complex is a biochemical marker and neurotoxic substance frequently identified in the urine of patients with autism, learning disabilities, alcoholism, substance abuse, schizophrenia, ADHD, Down syndrome, depression, bipolar disorders, and even criminal behavior. Some estimate the incidence of KPU to be 40-70% in schizophrenia, 50% in autism, 30% in ADHD, and 40-80% in alcoholism and substance abuse.

Based on testing with Klinisch Ecologisches Allergie Centrum (KEAC; http://www.hputest.nl) in Holland, Klinghardt has found the incidence of KPU in Lyme disease to be 80% or higher; in patients with heavy metal toxicity (lead, mercury, aluminum, cadmium, and others) over 75%; and in children with autism over 80%. These are very significant percentages of the patient population with chronic illness that may benefit from a treatment program that addresses KPU. Healthy controls do not test positive for KPU.

History
In 1958, a psychiatric research program in Saskatchewan, Canada, led by Abram Hoffer, MD, PhD, the father of orthomolecular psychiatry, was looking for the possible biochemical origin of schizophrenia and a lab marker that would make it easier to identify affected individuals. One study involved evaluating the urine for certain chemical fractions and evaluating those of schizophrenic patients and those of normal controls. The effort yielded “the mauve factor,” a specific substance that reliably allowed the examiners to identify the schizophrenic patients, as it was not found in the normal controls.

Early on, the substance was known as “the mauve factor” due to the mauve color that was observed on the stained paper. It was then termed “kryptopyrrole”, later identified as hydroxyhempyrrolin-2-one (HPL). The researchers first called the disease associated with this condition “malvaria,” but it was renamed by Dr. Carl Pfeiffer, MD, PhD to “pyroluria” which was, for no obvious reason, consistently spelled “pyrroluria” in later publications. Today, the condition is generally referred to as “pyrroluria.” In the 1970s, Dr. Pfeiffer created an assay for the condition and was able to show clinical improvement in positive patients with high doses of zinc and vitamin B6 (between 400 mg and 3,000 mg B6).

Associated Conditions
A partial list of conditions where KPU may be a factor include ADHD, alcoholism, autism, bipolar disorders, criminal behavior, depression, Down syndrome, epilepsy, heavy metal toxicity, learning disabilities, Lyme disease, multiple sclerosis, Parkinson’s disease, schizophrenia, and, substance abuse. The items listed in bold are those in which Klinghardt has observed a connection to KPU in his patient population.

Symptoms
The KPU condition results in a significant loss of zinc, vitamin B6, biotin, manganese, arachidonic acid, and other
nutrients from the body via the kidneys. There are many symptoms of KPU, which may result from deficiencies of these nutrients. Those in **bold** are tell-tale signs of the condition. Klinghardt finds that depression is often a leading symptom of the condition. Symptoms may include the following:

- Abdominal tenderness
- Abnormal fat distribution
- Acne, allergy
- Amenorrhea, irregular periods
- Anxiety / Nervousness
- Attention Deficit / ADHD
- Autism
- B6-responsive anemia
- Cold hands or feet
- Constipation
- Course eyebrows
- Crime and delinquency
- Delayed puberty, impotence
- Depression
- Emotional liability
- Eosinophilia
- Explosive or episodic anger

**Impact of Nutrient Loss**

Elevated levels of HPL found in urine are the result of an abnormality in heme synthesis. Hemoglobin is the substance that holds iron in the red blood cells. Heme is also the principal building block of many enzymes involved in detoxification (cytochromes), enzymes involved in healthy methylation (MSR and CBS), and NOS – a significant enzyme in the urea/BH4-cycle. HPL is a byproduct of dysfunctional heme synthesis and can be identified in the urine. HPL binds to zinc, vitamin B6, biotin, manganese, arachidonic acid (omega-6), and other important compounds that, as a result, are excreted via the urine. This leads to a significant depletion of these nutrients throughout the body and to the synthesis of non-functioning or poorly functioning enzymes. Turning to the importance of zinc, vitamin B6, biotin, manganese, and arachidonic acid in the body, it becomes clear how widespread the problems may be that are created by this condition.

Zinc deficiency may result in emotional disorders, food allergies, insulin resistance, delayed puberty, rough skin, delayed wound healing, growth retardation, hypogonadism, hypochlorhydria, mental lethargy, short stature, stretch marks or striae (which may be misinterpreted as *Bartonella* in some patients with Lyme disease), white spots on the fingernails, reduction in collagen, macular degeneration, dandruff, skin

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lesions such as acne, hyperactivity, loss of appetite, reduced fertility and libido, transverse lines on the fingernails, defective mineralization of the bones leading to osteoporosis, and many others.

Zinc is a powerful antioxidant, and lower levels lead to an increase in oxidative stress. Lower levels are correlated with lowered glutathione, an important part of the detoxification system. Zinc is required to support proper immune function. “White blood cells without zinc are like an army without bullets,” says Klinghardt.

Vitamin B6 deficiency is thought to be a rare occurrence. However, in those with KPU, this is not the case. B6 deficiency may lead to nervousness, insomnia, irritability, seizures, muscle weakness, poor absorption of nutrients, decrease of key enzymes and cofactors involved in amino acid metabolism, impairment in the synthesis of neurotransmitters, impairment in the synthesis of hemoglobin, seborrheic dermatological eruptions, confusion, and neuropathy. Like zinc, B6 is an antioxidant and correlates to levels of glutathione.

Biotin deficiency may be evidenced by rashes, dry skin, seborrheic dermatitis, brittle nails, fine or brittle hair, and hair loss. More importantly, however, it may be associated with depression, lethargy, hearing loss, fungal infections, muscle pain, and abnormal skin sensations such as tingling. Biotin is an important co-factor in the production of energy in the mitochondria. Biotin is essential for a healthy brain and nervous system. Biotin deficiency is associated with many aspects of the aging process.

Manganese deficiency may be associated with joint pain, inflammation, and arthritis. Deficiency may result in a change in hair pigment or a slowing of hair growth. It is essential for normal growth, glucose utilization, lipid metabolism, and production of thyroid hormone. It may be associated with diseases such as diabetes, dyslipidemia, Parkinson’s disease, osteoporosis, and epilepsy.

Klinghardt finds that it is rare for a patient to have chronic symptomatic Lyme disease as an adult without the patient having developed KPU. He postulates that the biotoxins from microbes block one or more of the eight enzymes of heme synthesis. This leads to a significant loss of key minerals in the white blood cells, which effectively disarms cellular immunity.

In those where KPU was triggered by infection with Lyme organisms, Klinghardt has observed that the KPU is often an unstable form of the condition where there are times of higher levels of pyrroles being excreted and times when this is not observed. If a person has episodes of depression, these episodes generally correlate to times when pyrroles are being released in higher levels in the urine.

One young adult female struggling with Lyme for several years had severe multiple chemical sensitivities (MCS) that were not improved by any previous treatment. After starting the KPU protocol, she noticed improvements in her MCS for the first time since she became ill. Other patients with intractable chronic infections have experienced significant improvements in immune function and a resulting lowering of total microbial burden.

Klinghardt has observed numerous patients that have struggled to rid the body of parasitic infestations. In these patients, regardless of the interventions used, the patient continues to expel these parasites on an ongoing basis. Therapy-resistant infections are a hallmark sign of KPU. Klinghardt has found that once the KPU protocol is put in place, there is often swift resolution of long-standing infections and infestations. This includes patients who have failed years of antibiotic therapy for chronic or late-stage Lyme disease.

Sandee Gupta, MD, from Australia has stated that parasites and pyrroluria almost always go together. He has observed that almost every chronically unwell individual seems to have both; one opens the door to the other. Chronically low levels of zinc allow parasites to invade the mucosal layer of the gut. Parasites may then move to the liver and gallbladder. They interfere with mood, energy levels, and sleep. Addressing the parasites while restoring zinc and B6 often makes a tremendous difference in his patients.
Kryptopyrroluria

Chronic Lyme disease patients often suffer from severe jawbone infections that may require cavitation surgery, which often tends to fail in this population. When the clients are pre-treated for KPU, the outcome of the surgical procedure is generally much better. In some mild cases, ozone treatment of the jaw may be sufficient to turn things around.

Klinghardt has followed the interest in HLA-DR genetic typing in regard to biotoxin illnesses such as Lyme disease and mold. Prior to KPU, patients with certain haplotypes were considered more difficult to treat as the body could not properly and effectively respond to and remove biotoxins from Lyme disease, molds, or in the worst cases, both. In his experience, once the KPU issue is addressed, these HLA types become far less of a concern in most patients and no longer hold them back on their road to regaining health.

Once bodily systems are back online and functioning properly, a few months after introducing the KPU protocol, patients become less vulnerable to Lyme disease, to mold, and even to heavy metals. Their bodies are now much better equipped to deal with these conditions when they have appropriate levels of zinc, vitamin B6, biotin, manganese, and arachidonic acid to support optimal functioning of numerous bodily processes.

KPU and Methylation

In Klinghardt’s work, if a patient has KPU, treating the KPU condition first is a foundational intervention before pursuing more specific methylation support. Specific enzyme blockages are discussed earlier in this article.

In people with cancer and active EBV infection, EBV triggers a hypermethylation inside the cancer cells that may accelerate cancer cell growth. If methylation support is introduced based on genetic SNPs or other lab testing but the patient has an untreated, active EBV infection (such as is common in chronic fatigue syndrome, Lyme disease, and other related conditions) or an EBV-related cancer such as throat, stomach, breast, prostate, or Hodgkin lymphoma, supporting methylation may lead the patient to an increased risk of cancer or accelerated rate of cancer growth.

This potential makes treating KPU first even more important as balancing the zinc and B6-dependent enzymes indirectly without the addition of methyl groups is generally a safer way to restore healthy methylation on all fronts as opposed to directly supporting methylation with methyl donors.

When people begin to explore methylation, KPU should always be evaluated and addressed first. Several enzymes in or adjacent to the methylation cycle use the heme molecule which utilize zinc and vitamin B6 as primary building blocks. By supporting KPU, the methylation cycle works more smoothly, both in its ability to methylate and demethylate, and at a lower risk to the patient.

KPU and Heavy Metal Toxicity

When KPU is present and zinc and vitamin B6 are depleted, the detoxification pathways are overwhelmed and ineffective as the heme molecule is an integral part of many detoxification enzymes. Both zinc and vitamin B6 deficiencies, which are important cofactors in the methylation cycle, reduce levels of glutathione in the body. Glutathione is important for the detoxification of heavy metals and other toxins.

Replacing missing zinc and vitamin B6 increases glutathione. This, in turn, increases the rate of detoxification of heavy metals and other body burdening toxins. Once KPU treatment is introduced with zinc and B6, reducing the metal burden no longer requires heroic measures.

However, it is also the case that incorporating the KPU protocol will liberate additional heavy metals within the body. This aspect of the KPU protocol is discussed later in this article and is important for the practitioner to understand before beginning to treat patients for the condition as additional detoxification support is generally needed. This protocol is intended to be done only with the guidance of a knowledgeable practitioner.

KPU and Porphyrin Disorders

There is a group of disorders related to pyrroluria called porphyrinas. KPU is one of a group of conditions known as porphyrin diseases. In 100% of porphyrin diseases, the HPL compound is found in the urine.

Porphyrin testing is readily available and is a reliable tool. Klinghardt prefers to send a urine sample to Laboratorie Philippe Auguste (http://labbio.net) in France for testing. Other options are also available in the US, such as through Genova Diagnostics, Doctor’s Data, and Great Plains Laboratory.

In the US, pyrroluria and porphyrina are viewed as separate conditions. However, in collaboration with the Dutch lab KEAC, it has been established that everyone with elevated porphyrins has pyrroluria. When pyrroluria is addressed, the porphyrins go down.

In porphyrin testing, uroporphyrin is an indicator for aluminum, coproporphyrin for lead, and precoproporphyrin for mercury. Klinghardt has not seen a case with elevated porphyrins that did not have KPU, and when the KPU was corrected, aluminum, lead, and mercury are excreted from the body, and the porphyrins go down.

This is, in part, due to the fact that when the body has been deficient in zinc for a long period of time, it may retain heavy metals much more readily. When zinc is missing from the body, it is replaced in our bones with lead. If zinc is supplemented, lead is expelled. Secondly, the enzymes needed to detoxify these metals are heme-dependent enzymes, and these metals accumulate when heme synthesis is abnormal.

Klinghardt notes that discussions on the topic of porphyria are much more widely accepted than those on pyrroluria. In his experience, he finds that almost all of his patients have elevated porphyrins, and that pyrroluria is the deeper core issue.

KPU and Histamine

When a KPU patient is having a good day, low histamine levels are observed; on a bad day, higher histamine levels are observed. It is the relative elevation of histamine in response to foods, inhalants, allergens, emotional stressors, and electromog that is problematic and causes the allergic phenomena, not the absolute histamine level. When histamine levels rise from a low level to a moderate level, the reactions are often severe.

When exploring histamine levels in a KPU patient at a time when they are experiencing hives or asthma, the histamine levels are elevated, but not to levels that would create a problem for
others. The relative rise in histamine, however, in KPU patients is experienced in a far more significant way.

Klinghardt has worked with biochemists in Germany that are beginning to link KPU with mastocytosis or mast cell activation syndrome (MCAS). They have observed that KPU treatment repairs the heme molecule, which notably stabilizes the mast cells and lowers the response to these relative rises in histamine.

**KPU and Multiple Sclerosis**

Klinghardt has treated many patients with multiple sclerosis. The MS patients that he has tested have been highly positive for KPU. Over time, he has concluded that KPU is a frequent cofactor in MS. He has found that patients with MS respond favorably to KPU treatment.

In patients with KPU, absolute histamine levels are almost always low. The treatment for MS patients with KPU may include histamine in addition to the KPU protocol outlined in this article. Treatment with histamine may be either with oral or transdermal products. Prokarin is a transdermal patch which delivers histamine and has been used by some practitioners in the treatment of MS.

**Evaluation and Testing**

Klinghardt recommends that people start with the HPU Questionnaire (http://www.hputest.nl/evraag.htm). Once the questionnaire is completed, a score is calculated to provide a probability that a person may have KPU. If the score is 10-14, Klinghardt will often recommend proceeding with treatment without the need for confirmatory testing as the treatment itself is generally well-tolerated. If the score is 0-9, he may suggest testing for the condition using additional lab work.

Pyroles are impacted by light, temperature, oxygen, and time; and they readily break down. Once they begin to break down, the likelihood of detection is significantly lowered. Ideally, testing would be performed within eight hours after the collection, though this is not practical and rarely possible.

Within the United States, two of the available labs for testing include the following:

- **DHA Laboratory** (https://www.pyroliuratiestesting.com) uses a frozen one-time collection at a cost of $80. They recommend the collection be the second urination of the day. They suggest avoiding all supplements, vitamins, and minerals for 12-24 hours prior to the specimen collection. The lab is testing for hydroxyhemopyrrolin-2-one (HPL).

- **Health Diagnostics and Research Institute** (http://www.hdri-usa.com) charges $140 for a 24-hour collection and $90 for a random collection. HDRI suggests stopping zinc and B6 as well as antidepressant medications for 48 hours prior to the collection. They suggest not smoking or consuming caffeine for 24 hours prior. While there is no additional cost for testing the hydroxyhemopyrrolin-2-one (HPL) compound, this must be specifically ordered on the requisition form as it is not part of their KPU assay by default. If you do not specify HPL as an add-on, you will get kryptopyrrole (2,4 dimethyl-3-ethyl pyrrole) only.

In Europe, Klinghardt uses the Dutch Lab KEAC (http://hputest.nl) for HPU testing. The lab is guided by microbiologist Dr. John Kamsteeg, a world leader in HPU. The results of HPU testing with this lab align closely with the percentages of patients with chronic Lyme and other conditions that Klinghardt identifies with the HPU condition.

In Australia, KPU testing is available through SAFE Analytical Laboratories (http://safelabs.com.au) and Applied Analytical Laboratories Pty Ltd (http://www.apanlabs.com).

Each lab has their own very specific instructions for performing the test. This includes information such as shielding the specimen from light as well as how to handle and ship the specimen. It is important that the recommendations be closely followed to optimize the sensitivity of the test result.

To further maximize the sensitivity of testing, it may be best for the patient to be under stress at the time the test is being performed as HPL excretion is known to increase during times of stress.

In some circumstances, however, patients may still test negative even when the condition is suspected. In those cases, it may be best to repeat the test. In many cases, the result will be positive on the second or third test. In some patients, an empiric trial of the KPU protocol may be indicated despite repeated negative KPU tests, and this often leads the patient to higher ground.

WBC (not RBC) intracellular zinc may be a useful tool for exploring the potential for zinc deficiency where it matters most - in the white blood cells.

Other laboratory indicators that may be suggestive of KPU include the following:

- WBC < 5000/mcL (due to low levels of zinc)
- High LDL / Low HDL
- Low normal alkaline phosphatase (<60U/L)
- Low omega-6 fatty acids in red cell membrane test
- Low taurine in amino acid profile
- High MCV
- Low glutathione
- Low ATP
- WBC and RBC zinc and manganese levels may be normal while biopsies from bone and CNS are completely deficient.
- Bone biopsies are a reliable predictor of KPU. Severe deficiencies of zinc, manganese, lithium, calcium, magnesium, and molybdenum are often found.

Alkaline phosphatase (ALP) is a zinc and magnesium dependent enzyme. When someone is consuming adequate magnesium and is still presenting with low ALP, zinc deficiency is a likely consideration, and this may represent another indication for KPU. When ALP is below 55, zinc deficiency can be suspected; when below 40, it is likely.

A consequence of KPU is low glutathione and low ATP. In the realm of chronic illnesses, low reduced glutathione and low ATP are common and should alone trigger the suspicion that KPU may be a factor.

**Treatment**

KPU is a severe but reversible deficiency of zinc, vitamin B6 (or P5P), biotin, manganese, arachidonic acid, and other co-factors. It is important to recognize, however, that treatment with zinc and vitamin B6 does not result in fewer pyroles being excreted in the urine. KPU orthomolecular treatment does not fix the underlying condition; it substitutes what is being lost as a result of the condition such that the person is no longer deficient in key nutrients needed by the body to move towards health.

The general KPU substitution treatment that Klinghardt uses in his practice is as follows (dosages for 160 lb.

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**Kryptopyrroluria**
Kryptopyrroluria

adult and should be adjusted based on weight; may be customized for specific patient needs):

With Breakfast
- Zinc 25-30 mg (as picolinate, gluconate, sulfate, or zinc l-carnosine). Nausea after zinc supplementation may be a sign of hypochlorhydria or low stomach acid; this often resolves after a few months on treatment.
- Vitamin B6 50-100 mg (split between pyridoxine HCl and P5P, with P5P being the predominant form)
- Biotin 3-5 mg for brain, skin, hair, and nails
- Magnesium 100 mg (glycinate, bisglycinate, or malate) – or titrate to bowel tolerance.
- Arachidonic acid from omega-6 oils (Ghee such as Mt. Capra Goat Milk Ghee, Evening Primrose Oil, Hemp Seed Oil, Black Currant Oil, Borage Oil, Pumpkin Seed Oil; 4-6 capsules of Evening Primrose Oil per day is commonly used.)

With Dinner
- Zinc 25-30 mg
- Vitamin B6 50-100 mg
- Biotin 3-5 mg
- Magnesium 100 mg
- Omega-6 Oils
This is the core treatment Klinghardt utilizes for KPU.

Additional Support
- Vitamin A 1,500-3,000 IU per day to improve the absorption of zinc in the gut
- Niacin 40-50 mg per day for psychiatric symptoms. (Abram Hoffer used up to 3000 mg per day.)
- Taurine 100 mg twice per day (up to 2,000 mg at bedtime) for brain-related symptoms such as seizures, brain fog, and memory loss. Supports elimination of neurotoxins, improves bile quality, increases glutathione, and normalizes brain rhythms.
- Lithium 5-10 mg per day (Orotate or Aspartate); Lithium is lost in the urine in some patients with KPU.
- Manganese 2-5 mg per day (Patients with joint problems may require additional manganese above the dosages recommended here; see additional considerations later in this article on manganese for patients with Lyme disease.)
- Chromium 250-500 mcg per day

- Molybdenum 100-500 mcg per day
- Boron 1-3 mg per day
- Trace Minerals - As more is learned about KPU, additional elements are found to be lower in those with the condition. Thus, supplementing trace minerals may be a supportive strategy. BioPure MicroMinerals, Quinton Isotonic, or similar mineral products may be helpful.

As compared to the first version of this article which was published in 2009, Klinghardt has found that many of his patients do quite well with lower dosages of some of these key nutrients than were originally utilized.

In Europe, Depyrrol is one product which provides support for KPU. Additionally, and in the United States, BioPure CORE and CORE-S are available to support those dealing with the condition. Another product in this realm is Mensah Medical’s Pyrole Pak. These products serve as a solid foundation for KPU treatment; though additional cofactors may be needed for a given patient. Some patients may not tolerate both vitamin B6 and P5P as contained in some products and may find it necessary to take each component of the KPU program separately.

In terms of BioPure’s CORE and CORE-S, CORE-S is a recent reformulation of the CORE product which has been available for many years. While either may be an appropriate option, CORE-S generally results in less nausea, better absorption, and is often better tolerated by those patients with Lyme disease as it does not contain manganese. While many with pyroluria may benefit from manganese, it may act as a growth factor for untreated Lyme disease, and thus, some may prefer to avoid its use in this patient population. The reformulated CORE-S contains horsetail as people with KPU excrete higher levels of silica in the urine, which leads to higher levels of aluminum toxicity. With either CORE or CORE-S, two capsules twice daily are a common target dose for a 160 lb. adult. When first starting to introduce products in support of KPU, it is best to start with lower dosages and to take them towards the end of a meal and to gradually work up to the target dosage. Levels of B6, taurine, or biotin may be additionally and individually titrated upwards depending on the patient’s symptoms and needs.

With the introduction of zinc, it is best to monitor copper levels after a few months on the protocol as copper replacement may also be needed. Zinc, vitamin B6, and manganese are copper antagonists. Thus, monitoring levels of copper and supplementing where needed is an important part of the treatment protocol.

Copper deficiency can lead to hemorrhoids, varicose veins, fatigue, edema, hair loss, anorexia, skin problems, osteoporosis, cardiovascular disease, aneurisms, and many other undesired conditions. Current nutritional teachings are misinformed on the topic of copper toxicity. The immune system uses copper and iron to fight infections associated with Lyme disease. As a result, oxidized copper is displaced in the connective tissue and may appear as though the patient is copper toxic by some testing methods when in fact copper supplementation may be appropriate. High dose Vitamin C has the effect of reducing oxidized copper to a form that can be reused by the body.

Detoxification and Course of Treatment
As treatment for KPU is implemented, this often can result in toxin mobilization as the body begins to release heavy metals. Symptoms may include muscle aches, bowel problems, or those normally associated with cleansing or detoxification reactions. Additionally, the immune system begins to become more active which can result in a Herxheimer-like reaction as the immune system begins to better respond to the backlog of microbes that it was previously unable to adequately address.

One approach for minimizing these reactions is to start slowly with introduction of the KPU nutrients and work up over time. In most cases, there is no reason that the treatment course must be an aggressive one. Nonetheless, this treatment should always be guided by a knowledgeable practitioner. In addition to the KPU treatment discussed earlier, consideration should be given to detoxification support and to protection of the red blood cells as the treatment is initiated.

According to Klinghardt, many of our metabolic enzymes use zinc as part of their molecular makeup. However, in patients with KPU, there is not enough zinc available to satisfy the need. In these cases, lead, mercury, and other 2-valent metals bind to these sites instead in a poor attempt to fulfill the role of zinc.
Once zinc is reintroduced into the body, 2-valent metals such as mercury, cadmium, aluminum, and lead are liberated. The patient may now have dislodged heavy metals circulating throughout the body. These may be competing for the already overtaxed detoxification pathways or may be redistributed to places where they may be more problematic. Lead moves back into the blood, which can cause problems including damage to red blood cells. To protect the red blood cells, freeze-dried garlic and Vitamin E are often used.

Incorporation of known toxin binders further supports the detoxification process. Some of the binders that Klinghardt uses in his practice include chlorella, Ecklonia cava, zeolite, and chitosan. Silica from horsetail supports binding of aluminum, and thus, the use of a high-silica zeolite such as BioPure ZeoBind is often utilized. It is critical to support the kidneys with specific drainage and organ support remedies in order to optimize the removal of heavy metals and to avoid stressing the kidneys.

An interesting observation has been that patients with KPU often get worse when an attempt is made to incorporate detoxification agents or antimicrobial agents prior to having first addressed the KPU condition. Once KPU has been addressed, other treatment options are often much more effective and better tolerated.

Additional Considerations

Many patients with chronic Lyme disease have issues with sulfur intolerance. This leads to a patient being unable to effectively utilize a number of detoxification agents such as alpha-lipoic acid, DMSA, DMPS, and glutathione; as well as supplements such as garlic. This may be related to genetic predisposition, but some of the enzymes involved in sulfur metabolism (CBS and others) are heme and B6 dependent; both of which are involved in sulfur metabolism (CBS and others) are heme and B6 dependent; both of which are involved in heme metabolism and require B6. As such, deficiencies in zinc, vitamin B6, biotin, manganese, and arachidonic acid are key pieces of the puzzle in addressing the complexities of chronic Lyme disease and many other conditions.

Resolution of KPU

For most with the condition, supplementation will be required for life. However, Klinghardt has seen complete resolution of the condition after having addressed epigenetic influences, trauma, or unresolved conflicts using tools such as mental field therapy, family constellation work, or EMDR. By resolving trauma in the ancestry, the epigenetics are influenced in a positive way and the condition resolves. Klinghardt has also observed complete resolution of Lyme-induced KPU when the infection is managed successfully with biological interventions.

Final Thoughts

Once patients are on the KPU protocol and mobilized metals have been addressed, the body begins to respond to backlogged infections and significant improvements in the patient’s condition are often observed. Hormonal status often improves. Some patients who have been on thyroid medication for years may even become hyperthyroid as the body begins to function more optimally. Other patients may lose weight. Symptoms directly related to low levels of zinc, vitamin B6, biotin, manganese, and arachidonic acid often resolve.

Just as homes are built by first laying a solid foundation, addressing KPU and the deficiencies in zinc, vitamin B6, biotin, manganese, and arachidonic acid are key pieces of the puzzle in addressing the complexities of chronic Lyme disease and many other conditions.

Evaluation for KPU is one of the first things that Klinghardt pursues in working with patients with chronic illnesses. Implementing the KPU protocol often yields progress that had not previously been possible, and patient recovery is accelerated in a very deep and profound way.

Disclaimer

This article is not intended to provide personal treatment recommendations or to facilitate self-treatment. Treatment should be done only under the care and supervision of a licensed medical authority. Attempts to self-treat the condition may result in unintended negative consequences.

Products

• Depyrol can be found at http://www.depyrol.de.
• Mensah Medical Pyrrole Pak can be found at http://www.mensahmedicalstore.com.
• Information on Prokarin is available at http://www.edmstl.com.

Useful Resources

• Mood Instability May Be Pyrrole Disorder, a possible cause of Bipolar, DSMIV. Albert Mensah, MD, October 15, 2015 http://www.mensahmedical.com/pyrrole-pyroline-disorder
• Pyrroluria: The Unknown Disorder. Dr. David Jockers, DC. http://drjockers.com/pyrroluria-common-unknown-disorder
• Pyrroluria, Jeremy E. Kaslow, MD, FACP, FACAAS. http://www.drkaslow.html/pyrroluria.html
• Change of Blood Ammonia Level and Efficiency of Nitrogen Utilization in Prangian Lambs Due to Klinoptiolit Addition in Ration. http://www.uaiasi.ro/zootehnie/Pdf/Pdf_Vol_56/Heni_Siti_Mansah.pdf
•mensahmedical.com/pyrrole-pyrrole-disorder

Scott Forsgren, FDN-P, is the founder of BetterHealthGuy.com, a health coach, blogger, podcaster, health writer, advocate, support group facilitator, and LymeLight Foundation board member. He recovered his own health after a 20-year journey through Lyme disease and mold illness. Today, Scott is grateful for his current state of health and all that he has learned on this life-changing journey. Dr. Klinghardt served as a powerful mentor, teacher, and guide as Scott worked to understand the disease which had previously taken so much of his life and moved toward a place of health and wellness. Scott continues to utilize a maintenance pyrrolelution protocol which he started almost a decade ago. To follow Scott’s work, visit http://www.betterhealthgy.com. His podcast “BetterHealthGuy Blogcasts” is available on his website and on YouTube, iTunes, Google Play, and Stitcher.

Dietrich Klinghardt, MD, PhD, studied medicine and psychology in Freiburg, Germany, completing his PhD on the involvement of the autonomic nervous system in autoimmune disorders. Early in his career, he became interested in the sequelae of chronic toxicity (especially lead, mercury, environmental pollutants, and electromagnetic fields) and its impact on chronic illness. Dr. Klinghardt has contributed significantly to the understanding of metal toxicity and its connection with chronic infections, illness, and pain. He has developed Autonomic Response Testing, a comprehensive evaluation system that has helped many practitioners to become accomplished holistic practitioners. He founded Sophia Health Institute (http://www.sophiahi.com) in 2012, and is actively involved in patient care at his clinic outside of Seattle. More information on his educational seminars can be found through the Klinghardt Academy (http://www.klinghardtacademy.com; US) and the Klinghardt Institute (http://www.klinghardtinstitute.com; UK).

Kryptopyrrolururia

Kryptopyrrolururia is one of the first things that Klinghardt pursues in working with patients with chronic illnesses. Implementing the KPU protocol often yields progress that had not previously been possible, and patient recovery is accelerated in a very deep and profound way.