GANA KIRITHARAN

"Providing Customers with Quality Information."

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Dear Lawyer

I Gana Kiritharan would like to hire your legal expertise to represent me in a complex Intellectual property issue. You can nick name this as "2nd Galileo trial". During a private scientific study I discovered present explanation for type 2 diabetic mellitus (T2DM) is fundamentally wrong. After overcoming various challenges I published my discovery in an article format by end of December 2017. Even though I expected recognition for my intellectual work in few week times, even after 9 months I failed to receive any professional recognition for my work.

At this juncture I like to hire your services to further promote my explanation of T2DM. In this issue my claims to medical profession either directly or through appropriate legal proceeding may as follows:

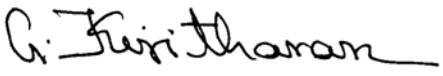
Claim 1.
Requesting the Medical Profession to accept the fact that present explanation to Type 2 Diabetic Mellitus is wrong and Gana Kiritharan's explanation is the correct one.

Claim 2.
Requesting Medical Profession to organize a medical research administrative frame work under the leadership of Gana Kiritharan to further develop Gana Kiritharan's Explanation to T2DM into a complete treatment system.

Claim 3.
Requesting the Medical Profession to provide Gana Kiritharan with necessary recognition and royalty payment for his work about T2DM.

If you are prepared to represent me in this issue kindly contact me for further discussion.

Yours truly,



Gana Kiritharan

Note: This letter and attachments are available in PDF format.

What is Present Explanation of Type 2 Diabetes Mellitus and how Gana Kiritharan's explanation to the disease differ from present explanation.

Presently diabetes defined as elevated blood sugar level due to deficiency in Insulin secretion or function or both. Presently medical knowledge further conclude elevated blood sugar level causes damages to body organs and causes kidney failure and other organ failure.

Gana Kiritharan's explanation T2DM argues that in T2DM various factors cause resistance to function of Insulin on cells. As a result not enough glucose enters the cells which lead to glucose deficiency in a form of chronic level inside the cells. This glucose deficiency causes body cells go in to a chronic stage of fasting. As a result body stated to break down protein and keep the blood sugar level up. This elevated blood sugar level helps the cells to get the glucose by simple diffusion so that cells can overcome glucose deficiency inside the cells.

In few words while present medical Profession says "Elevated blood sugar level poisonous for body cell Gana Kiritharan argues it is actually medicine for cellular health.

Gana Kiritharan manages his personal diabetic problem based on above explanation and was able to prevent renal failure or any other serious damages to body organs over last 5 years or more.

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This is an Interactive PDF file format. By Clicking on the Headings you can reach the first page of Appropriate Documents; then Clicking on the Bottom of Each Page can return to the First page

Annex

Related Gana Kiritharan's Intellectual Works:

Published December 2017

- **GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS [NON-INSULIN DEPENDANT DIABETIC MELLITUS] AND RELATED DISORDERS OF HUMAN HEALTH.**

Published September 2018

- **GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.**
  – How Metformin Works. –

- **GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.**
  - Preventing Renal Failure in Type 2 Diabetic Mellitus Patients –

- **GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.**
  - Continuous Blood Sugar Monitoring –

Published 2011

- **CHRONIC TOXIC METAL TOXICITY AND OTHER CHRONIC MEDICAL PROBLEMS.**

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS [NON-INSULIN DEPENDANT DIABETIC MELLITUS] AND RELATED DISORDERS OF HUMAN HEALTH.

BY:
GANA KIRITHARAN
Bachelor of Medical Science [Jaffna; Sri Lanaka]
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Gana Kiritharan makes following intellectual property rights claims about following article “Gana Kiritharan’s explanation of Type 2 Diabetic Mellitus and Related disorders of Human Health.”

(1) Gana Kiritharan makes full intellectual property rights claim for his work in all format of publication in all places of world.
(2) Gana Kiritharan claims this work formally identified in all places of publication; either as full article or a part of the article incorporated in other intellectual works; as “Gana Kiritharan’s explanation of Type 2 Diabetic Mellitus and Related disorders of Human Health”, or in a short format as “Kiritharan’s explanation of T2DM”.
(3) Five years exclusive rights: Gana Kiritharan makes a claim of 5 years exclusive rights to conduct any major researches or any other form of Scientific Experiments, to write any detail article and/or write any books about his explanation of Type 2 Diabetic Mellitus and Related Disorders of Human Health.
(4) If any body wanted to conduct smaller studies or conduct regional based scientific studies about Gana Kiritharan’s explanation of Type 2 Diabetic Mellitus and related disorders of Human health, please contact Gana Kiritharan for proper permission.

For other claims regarding this issue and claim of royalty please refer to the conclusion section of this articles and other documents published by Gana Kiritharan related to this issue.

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This Intellectual Work Dedicated to:

My Parents:

Parameswary Kanagalingam [1933 – 1994]
Chelliah Kanagalingam [1929 – 2016]

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS [T2DM] AND RELATED DISORDERS OF HEALTH.

ABSTRACT

Present explanation of T2DM saying increased consumption of high energy food, increased adaptation of sedentary life style and urbanization as the cause T2DM is fundamentally wrong. What actually happen is insulin resistance due to various reasons causes glucose deficiency inside the cells. This deficiency makes the body tissue to go into chronic fasting stage. But as blood glucose and insulin levels remains high, lipolysis do not happen in usual way. As a result muscle cells started to break down proteins for energy. Elevated blood glucose levels help the tissue get better supply of glucose by simple diffusion. If insulin given to bring down the blood glucose level in T2DM it will force muscles to brake down more protein to maintain high blood glucose level and lead to several toxic symptoms related to Hyperinsulinemia. Treatment of T2DM should aim at identifying and treating the root cause casing Insulin resistance and small frequent meals of small amount of carbohydrate and large amount of proteins.

1. INTRODUCTION

While medical profession has successfully overcome some challenge of human health like infectious disease and surgical techniques, new challenges are coming to the front. The three most important challenges in front of medical profession now a day may be:

i) Viral Disease like AIDS and other.
ii) Cancer
iii) Metabolic Diseases like Diabetes.

Even though Diabetes does not have high mortality rate like other two, still it can cause serious damages not only to human health but community economy as well. It is estimated 415 million people has Diabetes world wide. It is equal to 8.3 % of adult population. It is predicted the occurrence of Diabetes will continue to rise in human population. Why is this situation? Is there any mistake in understanding the disease?

I got diagnosed with Type 2 Diabetic Mellitus (T2DM) in 2005 (when I was 37 years old) and started treatment for it. From the beginning of itself I had several confusions about the course of my disease. My blood sugar value unexpectedly goes up or down in several instances. Most important discovery may come in year 2010 (When I was 42 years old). In that year I realized I am a victim of chronic form of Toxic Metal Toxicity possibly due to criminal intention and started treatment for it. Based on several observation I have made during the treatment of my chronic form of toxic metal toxicity and treatment of my T2DM problem and my parents experience of managing their chronic diseases and based on the intellectual research I have done about the diseases, I came to a conclusion that present explanation T2DM is fundamentally wrong and need to be redefined.

2. PRESENT EXPLANATION OF DIABETIC MELLITUS AND T2DM.

Presently diabetic mellitus defined as a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action or both. Then diabetic mellitus further divided into two major group Type 1 and Type 2. There are other smaller groups of diabetic disease as well. This article mainly talks about Type 2 Diabetic Mellitus (T2DM) which make 95% diagnosed diabetic patients. T2DM remains asymptomatic for several years and there for remains undetected for several years and there for remains undetected in nearly 50% of persons affected by the diseases. Present medical profession has conducted several studies focused on molecular mechanism underlying T2DM without much success.

Present Medical knowledge blames increased consumption of high energy foods, increased adaptation of sedentary life style and urbanization as the course of increased incidents of T2DM. I claim these conclusions are fundamentally wrong. My conclusion based on my observation of my health problems and my parents' health problems leads to a different explanation.

3. GANA KIRITHARAN'S EXPLANATION OF T2DM.

According to my explanation in a T2DM patient first happens is development of Insulin resistance. Though I do not completely disagree with the argument that high energy food, sedentary life style and urbanization causes obesity and this obesity lead to insulin resistance, I do not believe this is the main cause of insulin resistance. As I understand the main cause of Insulin resistance probably come from toxins, chronic form of viral, bacterial and/or fungal infection of internal organs. How these factors causing insulin resistance is another subject matter. My previous article "Chronic Toxic Metal Toxicity and other Chronic Medical Problems" may explain how heavy metals toxicity causes insulin resistance.

Next thing happens after Insulin resistance is glucose deficiency inside the cells. This glucose deficiency pushes the cells to metabolic stage of chronic starvation. But starvation due to insulin resistance differs from starvation due to person not taking food for several days. In normal starvation blood level of glucose and insulin level will go down and this will lead lipolysis of adipose tissue which will lead to energy supply of the tissue. In insulin resistance as blood level glucose and insulin do not go down, so that lipolysis do not happen. But still there is not enough supply of glucose into muscle cell. This leads to catabolism of protein in muscle cells. This catabolism of muscle cells not only supplies glucose for muscle cells but also to nerve cells, red cells and also kidney tissue as well. While Insulin resistance not helping to get enough supply of glucose for energy need, elevated blood sugar level help to improve the supply of glucose for tissue by increase simple diffusion of glucose across cell membrane. What level of blood glucose will supply enough glucose for cell energy need, will be the blood level of glucose. When Insulin resistance is sever the blood glucose level will go up and when insulin resistance is mild the blood glucose level will come down. My above explanation of T2DM needs to be expanded in more detail. I will full fill this responsibility in coming weeks. For now please let me explain some associated issues of my explanation to T2DM.

3.1 Increased Blood Sugar Level and Pathological Damages to the Tissue.

Almost all the researches conducted so far about T2DM gave a direct relationship between elevated blood sugar level and pathological damages to the tissue. Why is this? If you agree with my explanation you may accept that the elevated blood sugar level directly proportional to insulin resistance and glucose deficiency inside the cells. So pathological damages; actually

caused by increased insulin resistance and increased glucose deficiency in side the cells, not because of increased blood glucose value. Increased blood glucose value is protective mechanism which we should not interfere with.

3.2 What is the treatment for T2DM.

If we should not interfere with elevated blood glucose level then what is the treatment for T2DM. First we should identify the root cause causing Insulin resistance and treat the problem. Secondly we should give small frequent meals of small amount of carbohydrate and large amount of protein. This will help to supply necessary glucose to the tissue and will try to balance the catabolism of protein. A meal of large amount of carbohydrate will exhaust the available Insulin pumping mechanism and increase the blood insulin level, which will lead to pumping of more glucose into fat cells. A large fat meal will also make the adipose tissue to grow, which won't be used in a T2DM patient. Most important thing may be T2DM patients should not fast. If they fast their adipose tissue won't be utilized but the protein will burned to create energy.

4. WHAT WILL CHANGE? WHAT WILL NOT CHANGE?

Let us look into how my explanation of T2DM will change or will not change present way of managing the disease. I say glucose deficiency inside the cells is the root course of T2DM. But is it possible to measure the glucose deficiency inside the cells. It won't be possible for two reasons. Preparing micro needles and utilizing them to measure the glucose deficiency inside the cells is not possible in normal clinical setup. Secondly in a T2DM patient glucose deficiency inside the cells may be corrected by the mechanism I have explained above. So to determine the glucose deficiency inside the cells the possible clinical way is to measure the blood sugar level. But when interpreting the results we are going to worry about glucose deficiency inside the cells not going to worry about elevated blood sugar level.

But when it comes to the treatment several things will change. Are we going to give Insulin to bring down the blood glucose value? Even though I can roughly say Insulin is contra indicated in T2DM, only a detail clinical study in the future will determine which kind of Insulin is useful and in what type of situation. But in general if you accept my explanation, when Insulin given in order to bring down the blood glucose level muscle will try to break down protein and try to keep the blood glucose level up. This will lead to muscle wasting and high blood pressure like toxic symptoms. Diabetic ketoacidosis where Insulin is mandatory is a problem of T1DM not of T2DM.

I stop here on listing what should be done and what should not be done. I will work with appropriate medical societies and will conduct several detail scientific studies and other forms of intellectual discussions which will decide and tabulate the proper patient care for T2DM patients.

5. TIME NEEDED FOR CHANGE AND WARNINGS.

William Edward Deming who is considered father of Quality Management concepts says "A big ship traveling in full speed need distance and time to turn around." Today T2DM is a ship carrying more than 400 million patients and traveling in wrong direction. Nobody can expect a change in treatment of T2DM in few weeks time. Every body may have to wait 3 months or more for medical profession to reach a conclusion about my explanation to T2DM.

I am inviting World Health Organization, Indian and Singapore Medical councils to lead this complex turn around procedure. Depending on the response I will work with them in this turn around procedure over next few years.

I also wanted to warn any body or institution or organization who will try to take my explanation in their hand without my permission or participation and try to develop in into a complete treatment protocol. You may end up in a disaster like Iraq and Syria in which situation even myself can not give any big help.

6. METHODOLOGY

Before conclusion let me talk about methodology I followed and other scientific details of my explanation. To reach above conclusion about T2DM, I followed methodology of giving logical organization of few observations I have made about T2DM. Same methodology may have been followed by Charles Robert Darwin on making conclusion about his theory of evolution.

Following are the observation I have made about T2DM which lead to my conclusion.

i) I experienced high level of fluctuation of fasting glucose value on daily basis.
ii) Previous day 25 grams containing coconut syrup help to reduce the fasting glucose value of the next day.
iii) Attempt to protect myself from poising attempts from toxic metals and proper Detox protocols helped to reduce the fasting glucose value.
iv) When I tried to take Insulin to control my blood glucose value it failed to bring any big control but experienced increased toxic symptoms which include high level of increase in blood pressure and increased muscle and joint pain.
v) Several times I observed; longer the fasting duration the blood glucose value started to go up.
vi) On 25th of October 2010, I experienced increased fasting glucose value than previous night post brandial blood glucose value.
vii) My father who was a T2DM patient, never took Insulin, did regular exercise lead to a normal life with out any diabetic complications and lived up to 87 years of age.

In addition following already established intellectual conclusion about glucose metabolism also helped to lead to my conclusions.

i) Daily blood level of insulin goes up and down based on blood glucose level but blood level of glucagon level stays same most of the time.
ii) In chronic fasting stage of metabolism muscles break down of protein and convert it into glucose

6.1 Discovery vs Invention

My conclusions about T2DM are discovery not invention. If you want me to bring an example from the history of science, Sir Isaac Newton's falling apple experience and creating Law of Gravity may be a good example. Newton was able to create his Law of Gravity without much experiment as it was established truth that any fruit or any other object when left free, will fall towards earth.

Same way my explanation to T2DM also based on established facts, so it can be accepted without spending time on scientific experiments or research.

7. CONCLUSION

On conclusion I wanted to make following statements about T2DM and my explanation to the disease.

- Present Explanation of T2DM fundamentally wrong.
- T2DM cased by factors causing barriers for glucose entry into cells.
- In T2DM body goes into a modified chronic stage of fasting, in which body breaks down protein and convert it into glucose.
- Present treatment like giving insulin to T2DM make the pathological damages become more severe.
- I invite medical professionals (WHO, India and Singapore) to work with me to work out a detailed treatment protocols based on my explanations.
- In addition to intellectual property rights claim, I also make a royalty claim of 75% of money going to be saved as a result of my explanation to T2DM for next 20 years.
- I request WHO to appoint a panel of experts to verify whether Tamil Community (or any others) poisoned with toxic metals or any other toxins with criminal intention and take necessary actions to treat and protect such victims, also hand over the findings to appropriate International Criminal Judicial system for further actions.

REFERENCE:

1. Holt, Richard I. G,… [et al]: Text Book of Diabetes; 5th Edition: Wiley Black Well: 2017.
2. Global Report on Diabetes: World Health Organization.
3. Article about Diabetes Mellitus: Wikipedia: 2017
4. Trotora, Gerald J: Anatomy and Physiology : Wiley India Pvt Ltd; Indian Edition: 2016.
5. Article about Charles Darwin : Wikipedia: 2017
6. Article about Isaac Newton : Wikipedia: 2017
7. Article about William Edward Daming : Wikipedia: 2017

GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.

- How Metformin Works. -

BY:

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.

- How Metformin Works. -

One of the first line of medication in Diabetic management is Metformin (Dimethylbiguanide). Biguanides history come from guanide rich herb "Galega *officinalis*" (goat rue or French lilac) that was used as a traditional treatment in Europe. Metformin is the most prescribed glucose lowering agent world wide.

Present medical knowledge says that Metformin exerts a range of actions that counter insulin resistance and lower blood glucose; the drug also offers some protection against vascular complications independently of its anti hyperglycemic effect. During my private study to understand relationship between chronic form of toxic metal toxicity and T2DM, I developed suspension that Metformin also works same way other Chelation agents work. When I stopped and restarted Metformin for CAT scan, it bring down the blood glucose level in a same way other chelators does. I waited for invitation and permission to do my scientific study in proper medical premises. As invitation and permission is being delayed I conducted a limited experiment in my own body.

I stopped all medication including Metformin for 24 Hrs. [I still took Ramipril, Atorvastating and Metoprolol during this time.] Then collected urine for next 24 Hrs. After this I started Metformin again (1000 mg bid) and collected urine for 24 hrs. Send both Urine samples to Doctors Data lab to measure the toxic metals levels in urine. I also conducted Challenged urine test 2 weeks before this tests. Results are on following page:

When analyzing the results you can make following conclusions.

- Chelators like CaEDTA, DMPS has superior effect on removing toxic metals from body.
- Metformin helps to remove toxic metals to some extent (Arsenic excretion increased by 20 times.)

This scientific study may inform Metformin works by removing toxic metals from body. I invite medical professionals to conduct similar scientific study to verify any other medication used in chronic medical problems work in similar way.

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Date	17 Sep 20018	18 Sep 2018	4 Sep 2018
Provocation Agent / Toxic Metal Scale $\mu g/g$ Creatinine	No Medication	Metformin 2000 mg	DMPS 180 mg CaEDTA 2 g
Aluminum (Al)	6.4	4.8	8.7
Antimony (Sb)	< dl	< dl	.4
Arsenic (As)	6.7	140	29
Barium (Ba)	3	2.7	6.4
Beryllium (Be)	< dl	< dl	< dl
Bismuth (Bi)	< dl	< dl	.6
Cadmium (Cd)	.2	.1	.5
Cesium (Cs)	7.2	5.9	5.2
Gadolinium (Gd)	< dl	< dl	< dl
Lead (Pb)	1.2	.7	12
Mercury (Hg)	3.1	.6	4.7
Nickel (Ni)	4.4	2	16
Palladium (Pd)	< dl	< dl	< dl
Platinum (Pt)	< dl	< dl	< dl
Tellurium (Te)	< dl	< dl	< dl
Thallium (Tl)	.3	.5	.4
Thorium (Th)	< dl	< dl	< dl
Tin (Sn)	.2	.3	5.2
Tungsten (W)	.1	.1	.2
Uranium (U)	< dl	< dl	< dl
Total	26.4	152.9	80.6

Attachments: Toxic Metal Reports.

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.
- Preventing Renal Failure in Type 2 Diabetic Mellitus Patients -

BY:

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- Preventing Renal Failure in Type 2 Diabetic Mellitus Patients - "

(1) Gana Kiritharan makes full intellectual property rights claim for his work in all format of publication in all places of world.
(2) Gana Kiritharan claims success of his explanation depends on patients' and medical professionals' ability to identify the root cause causing insulin resistance and treating it.
(3) If you want to republish Gana Kiritharan's intellectual works please contact Gana Kiritharan for proper permission.

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.
– Preventing Renal Failure in Type 2 Diabetic Mellitus Patients –

Present medical explanation says a micro vascular damage in T2DM is the root cause of renal failure. Even though I do not completely disagree with this explanation according to my explanation renal failure in T2DM is caused by toxic damages due to root factors causing insulin resistance and glucose deficiency inside renal tissue. In this article I will explain how to manage these two issues so that renal failure can be prevented in T2DM.

1) Preventing toxic damages due to root factors causing insulin resistance.

Important step in managing T2DM and preventing complications due to T2DM is identifying the root factor causing insulin resistance and treating it. Most common causes may be chronic form of toxic metal toxicity and chronic form of viral, bacterial and fungal infections of internal organs. If you suspect infection of internal organs you can do necessary tests to identify the responsible microorganisms and treat them with appropriate antibiotics.

Let us talk in details about how to diagnose and treat chronic form of toxic metal toxicity. Proper treatment of chronic form of toxic metal toxicity may involve doing test like hair mineral analysis and challenged urine test and following treatment with appropriate chelation agents and high dose of vitamins, minerals and other naturopathic medications. But this facility is not available for many patients. So, I am giving following simplified treatment for chronic form of toxic metal toxicity with 3 medications.

a. (Buffered) Vitamin C.

Vitamin C helps human health in several ways. One important function is anti oxidant. When Vitamin C buffered with calcium or other minerals, it helps to prevent irritation of stomach due to acidity of Vit C. Buffered Vit C when taken in large amount helps to prevent pathological damages due to oxidation effect of toxic metals and other toxins.

b. N-Acetyl Cysteine [NAC]

NAC is the precursor of Glutathione. Glutathione is an antioxidant of the body. It not only protects cells from oxidation damages of toxins but also help to remove toxic metals from body.

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c. Chlorella

It is an alga which has high nutrient value. It also has the property of absorbing heavy metals into it. When give in an appropriate dose it absorbs heavy metals released into gut by the liver and prevent them reabsorbed back into the system.

d. How to administer them.

The better order to begin their administration my as follows.

d.1. Start with Buffered Vit C starting 500 mg once a day, can increase up to 1000 mg three times a day.

d.2. After 4 weeks of starting Vit C start to add chlorella. Starting 500 mg once a day can go up to 500 mg 3 times a day.

d.3. After 4 weeks of adding chlorella start NAC. Again start 500 mg once a day can go up to 500 mg 3 times a day.

At any time if experience abdominal discomfort or allergy – especially when taking NAC or Chlorella – Stop them immediately and try to restart after 2 weeks. If the problem persists consult a qualified naturopathic doctor.

2) Managing Glucose Deficiency inside the tissue.

Important reason for renal failure in T2DM patients is glucose deficiency inside the renal tissue. The main reason for this is misunderstanding of the disease. Present explanation saying elevated blood sugar as the main cause of pathological damage is a mistake. Actual problem is glucose deficiency inside the tissue and elevated blood sugar is a body defensive mechanism through which body try to get enough glucose supply. To maintain good supply of glucose to kidney and other tissue please follow the following advises.

a. Reduce or Stop Insulin.
If you are a T2DM (not T1DM) patient and you are on Insulin you may be making a mistake. Especially if you are experiencing elevated blood pressure and pain in muscles and joints your body started to break down protein to keep the blood sugar level up. The only way to prevent this is to reduce or stop taking Insulin.

b. Taking food with small amount of Carbohydrate or sugar every 2-3 hours will help to maintain a sustained elevated blood sugar level so that kidney and other tissue will get enough glucose supply.

By following above advises only I, Gana Kiritharan, was able to prevent any serious damages to Kidney. I strongly believe above advise will help you to prevent any damages to kidney and any other tissue.

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GANA KIRITHARAN'S EXPLANATION OF TYPE 2 DIABETIC MELLITUS AND RELATED DISORDERS OF HUMAN HEALTH.
- Continuous Blood Sugar Monitoring -

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Gana Kiritharan's Explanation of Type 2 Diabetic Mellitus and Related Disorders of Human Health

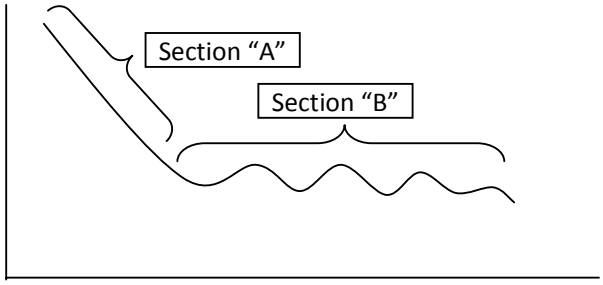
– Continuous Blood Sugar Monitoring –

One of the new facilities available in diabetic mellitus management is continuous blood sugar monitoring. The pharmaceutical company Abbott in their line of blood sugar monitoring devices named Free style released a new device named "Free style Libre". Even though named blood sugar monitoring device, it actually monitor the sugar level of Inter tissue fluid, which may be slightly lower than blood sugar level.

I bought this device in July 2018 and started to monitor the blood sugar level from 2nd of August 2018. I am sharing my observation and my explanations for the observations through this article.

1. **Elevated Blood Sugar Level in the Morning Hours.**
   Elevated Blood Sugar level in the morning hours is an observation made by several medical professionals previously. I also observed this previously and mentioned in my article "Gana Kiritharan's explanation of T2DM and related disorder of Human health." Present Explanation for This Phenomena as follows
   a. High Carb bedtime snacks and not enough diabetic medication.
   b. Dawn Phenomenon which says body prepare for next day energy need.
   c. Somogyi effect or rebound hyperglycaemia. According to this explanation your blood sugar drops too low so that body will release hormones in attempt to rescue you from dangerously low blood sugar level.

According to my observation a long night fasting blood sugar level will be something as follows.



If you accept my explanation of T2DM it may be easy to explain above phenomena. According to my explanation during "A" section of graph blood sugar level is high so the cells receive enough sugar supply through insulin pump and additionally by simple diffusion. When, the situation reaches the "B" section of the graph; sugar supply to tissue due to simple diffusion decrease. It leads to stress hormone released by body tissue. These stress hormone forces liver to release more sugar and when fasting continues body even started to break down protein for energy need.

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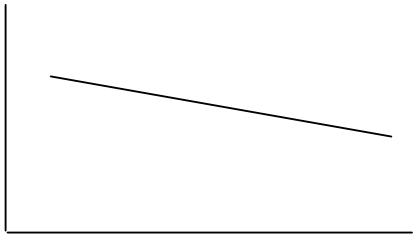
But as I argue, lipolysis will not happen in normal ways as blood sugar and insulin level will not come down. This phenomenon will continue up to next meals. By bringing next meals closer you can avoid this phenomenon from happening.

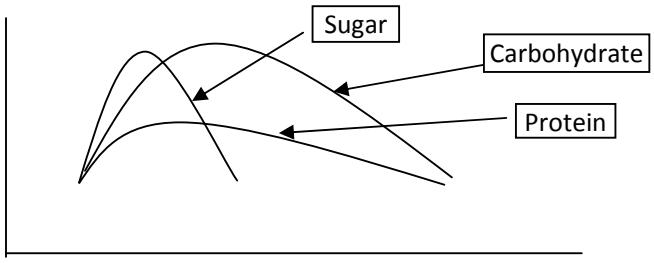
2. Effect of Exercise in T2DM Patients' Blood Sugar Level.

Even though I was not able to perform heavy exercise during last few weeks, I was able to do some mild exercise like moving boxes in a storage etc. During exercise whether it follows a fasting stage or after a meal, blood sugar level tries to come down gradually.



3. Various Types of Meals.

During last few weeks I tried various types of meals and observed how they influence blood sugar graph. My observation as follows:



While a food or drink with sugar in it pushes the blood sugar level high but quickly bring it back to normal. A meal with large amount of carbohydrate pushes the blood sugar level high mean time take a while to bring it back to normal. A meal with large amount of protein pushes the blood sugar level up by minimum high and gradually bring it back to normal.

4. Effects of chelation medication.

Another observation how various chelation medications push the blood sugar levels up. I observed while some chelation medications push the blood sugar level up others help to bring it down. CaEDTA pushes the blood sugar level by large limit but Plaquex and DMSA helps it to bring it down.

Attachments:

My continues glucose monitoring graph from 2nd of August 2018.

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Chronic Toxic Metal Toxicity and other Chronic Medical Problems.

Prepared by

Gana Kiritharan

ABSTRACT

I, Gana Kiritharan, am experiencing chronic medical problems from year 2002 (age of 34) which can be identified as Metabolic Syndrome. On May 2010, I discovered that I am a victim of chronic form of Toxic Metal Toxicity (Mercury, Lead, Cadmium and other) possibly due to criminal intention. After I started treatment for my Toxic Metal Toxicity I experienced high level of fluctuation of my Fasting Blood Glucose value. During last 12 months my fasting blood glucose value went above 16 mmol/L twice and it went below 8 mmol/L twice. Already conducted research explains toxic metals like Cadmium can cause impairment of Glucose Tolerance in Rats. These researches explain Cadmium can seriously reduce the number of Insulin Receptors in fat cells but can cause moderately elevated insulinemia. When I tried to take Insulin injection for my diabetic problem it not only failed to bring any big control but also caused complications which can be attributed to Hyperinsulinemia. When I recalled my mother's medical problems I realized she may suffered chronic form of toxic metal toxicity for a long period of time and she may get these toxic metals from fire woods used in the kitchen. When I searched for more evidence for toxic metal toxicity as the cause for woman health problem, I found toxic metal toxicity may me an important contributory factor for menopausal syndrome and several other psychological problems suffered by woman. Estrogen or some of it byproducts in synthesis may give protection for woman from toxic metals until menopause. Through this article I want to call medical profession to abundant its present attitude of denial and refusal and come forward to establish a proper preventive, diagnostic, and treatment protocols for this complex medical problem.

Introduction:

I born on September 1967 and selected for Medical Studies in 1987 and was in Jaffna (Sri Lanka) medical school till 1995. In our medical school; as our part of studies; we carried out several lab experiments on our body. Most of the lab experiments conducted on my body (WBC/DC to ESR) brought normal results. As a part of small medical research my Glucose Tolerance Test was checked in 1989 - 1990 (When I was 22 or 23). Out of 3 or 4 medical students checked, mine was the one showed most intolerance. Any how it was far beyond for diagnosis of diabetic mellitus. The available record of lab experiment was conducted in August 1999 in Ottawa, Canada. On that report random serum glucose was 5.6 mmol/L and all other indices were within normal range.

The first medical report which showed some chronic medical problem was conducted on June 2002 (Age 34).

Triglyceride	7.8	mmol/L
Cholesterol/HDL	6.03/.99	mmol/L
Fasting Glucose	6.2	mmol/L

My family doctor gave some dietary advise and we repeated test after 3 months.

Triglyceride	>11.4	mmol/L
Cholesterol/HDL	7.69/1.29	mmol/L
Fasting Glucose	6.5	mmol/L

Doctor started Fenofibrate as medication for my Triglyceride and Cholesterol problem and tests were repeated after two months.

Triglyceride	2.74	mmol/L
Cholesterol/HDL	5.35/1.55	mmol/L

Fasting Glucose was not checked at that time.

In year 2005 (Age of 37) my fasting blood glucose level went up and doctors started treatment for diabetes. Important Information about my chronic medical problems may come to light on 18th or 19th of May 2010. On that date, while I was cooking some curry at home, I saw small amount of glittering fluid was running in cooking utensil. On a suspicion that the glittering fluid may be toxic metals like mercury, I carried out appropriate lab experiments and found several toxic metals got accumulated in high amount in my body.

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Before moving further let me tell you a brief past medical history and family medical history.

Past Medical History:

I came to this world through 3rd cesarean section for my mother. First child for my mother was a still birth and I do not know the year of this first cesarean section. Then my elder brother born on October 1964 through second cesarean section and after 3 years I cam to this world through third cesarean section. As my parents already had a baby boy they may be expected a baby girl. Unfortunately I came out as a boy and this may lead some unwanted baby boy treatment in early childhood. Anyhow I received much better care than any other average child in my community. One example may be when my brother was preparing for his grade 5 (10 years old) IQ test; I was 7 years old and able to solve most of the problems. When my turn came my parents arranged best private tuition available in our village and I scored 157 for 200. That was the highest at least in our school.

Following table may give important medical incidents in my life.

Age	Incident
5 – 10	I may be a very week person. Once one of my pear group friends described my body as a good model to teach bones in the body. A regular upper respiratory track infection may take more than two weeks to heal and once family doctor threatened I may suffering from TB. When I started to grow up I started to eat and any nutritional problems may disappeared.
Around 12	I may developed quadriceps tendonitis and it healed with enough rest and appropriate medication.
18 - 19	Around my high school exams I may developed sinusitis and took Ampicillin antibiotics through out my exam period.
21	When I went to my medical school, for the first time I may took food out side my home for a long period of time and I was taking vegetarian food as well. After 3 months or so I developed Urinary Track Infection. I took Nalidixic acid. After this my parents moved with me and I started to eat home cooked food again.
24 or 25	When I went to rescue some war injured casualty I got trapped in cross fire and got a small injury in right upper arm. Through a small surgery the fragment was removed next day. During treatment for this injury; for the first time; I developed a Malaria infection. It healed with one full dose of treatment.
21 – 28	During my Medical School I may developed Upper Respiratory track infection more frequently, may be every two months. Also Viral Warts and on and off also experienced weight gain.
29 – 31	When I waiting to leave Sri Lanka to India I may developed a Malaria infection. Again while in India I developed Malaria infection 3 – 4 times. Then I took some broad spectrum anti-malarial drugs and did not developed malaria infection after that.

Table 1: Past Medical History of Gana Kiritharan.

Family Medical History:

Let me give you a small family medical history. The only significant medical problem my brother developed may be Rheumatic Fever. He may developed this medical problem around 15 years old and followed medically advised monthly Benzathine Penicillin injection and escaped without any permanent damage.

My dad is a Diabetic Patient developed this problem in his late 30s or early 40s. He managed this problem on oral hypoglycemic drugs, never took insulin Injection. He did regular exercise (went work on bicycle and managed our 2 acre land mostly without hiring additional labor). He may not be worried about his diet much. He used to eat regular food with others but avoided sugar. I can confirm his abdominal circumference more than 40 inches. His triglyceride level may never been checked and I do not remember any doctor diagnosed him as metabolic syndrome patient. My mother may had many medical problems and I will discuss these in detail later.

Details Medical History:

Following table gives lab experiments conducted during last 10 years and any change in the environment or medication.

DATE	ENVIRONMENTAL DETAILS	TRIGLY CERIDE	CHOLES TEROL	HDL	FASTING GLUCOSE	MEDICATION FOLLOWED BEFORE LAB EXPERIMENT.
19-Jun-2002	In Ottawa. Exercise in form of walking	7.08	6.09	.99	6.2	No Diet or Medication
11-Sep-2002	In Ottawa. Exercise in form of walking	11.4	7.69	1.29	6.5	Some Diet
14-Oct-2002	In Ottawa. Exercise in form of walking	2.74	5.35	1.55		Fenofibrate
7-Oct-2003	In India. Exercise in form of Walking	2.24	6.9			Fenofibrate
22-Oct-2004	In Toronto. Exercise in form of heavy manual work.	6.63	5.86	1.04	8.16	Atorvastating
4-Jan-2005	In Toronto. No Exercise for 2 week. Holiday Period.				18.1	Fenofibrate
2-Mar-2005	In Toronto. Exercise in form of heavy manual work.	2.25	5.07	1.19	7.4	Metformin, Fenofibrate
8-Mar-2006	In India. Exercise in form of Walking	2.56	6.81	2	6.67	Metformin, Glyburide
30-Mar-2006	In India. Exercise in form of Walking	1.93	5.2	1.4	5.11	Metformin, Glyburide, Atorvastating
23-Sep-2006	In Toronto. Exercise in form of heavy manual work.	3.73	4.38	.96	11.5	Metformin, Glyburide, Atorvastating
28-Dec-2006	In Toronto. Exercise in form of heavy manual work.	4.79	4.56	1.13	8.2	Metformin, Rosiglitazone, Atorvastating
17-Apr-2007	In Toronto. Exercise walking and climbing stairs.	2.27	3.84	1.22	7.9	Metformin, Rosiglitazone, Fenofibrate
7-Mar-2008	In Toronto. Exercise walking and climbing stairs.	3.14	5.84	.98	9.2	Metformin, Rosiglitazone, Fenofibrate
28-Mar-2009	In Toronto. Exercise walking and climbing stairs.	3.16	5.3	1.19	11.9	Metformin, Rosiglitazone, Fenofibrate
12-Dec-2009	In Toronto. Exercise walking and climbing stairs.	4.18	6.39	1.08	11.8	Metformin, Rosiglitazone, Fenofibrate
26-May-2010	In Toronto. Exercise walking and climbing stairs.	3.25	4.92	1.17	14.7	Metformin, Rosiglitazone, Gliclazide, Fenofibrate, Atorvastating
Jul – Sep 2010	Diagnosed with Chronic form of Toxic Metal Toxicity and Started Treatment for it.					
6-Dec-2010	In Toronto. Not much Exercise.	4.63	5.79	1.30	14.6	Metformin, Gliclazide, Atorvastating
29-Jan-2011	An Ultra Sound Reveled, a small cyst in right kidney and Fatty Liver Infiltration.					
8-Feb-2011	In Toronto. Not much Exercise.	5.48	5.42	1.27	11.8	Metformin, Insulin, Atorvastating
28-Apr-2011	An repeated Ultra Sound revels gradually enlarging cyst in right kidney, Fatty Liver and Mild Hepatomegaly.					
9-May-2011	In Toronto. Strict Diet and Moderate level of Exercise	2.81	4.77	1.2	12	Metformin, Herbal, Fenofibrate.
21-July-2011	In Toronto. Strict Diet and Moderate level of Exercise				6.9	Metformin, Fenofibrate.

Table 2: Lab Experiments Conducted on Gana Kiritharan.

My first two medical reports may enough to suspect that I may be suffering from Metabolic Syndrome. Further details tests may have helped to correct diagnose. When I started to take Insulin, it not only brought any big improvement but my BP started to go up. This only led me to search for more explanation about my medical problem and help me to find out the words Insulin Resistance, Hyperinsulinemia and Metabolic Syndrome. Above details presented in following graph for easy understanding of fluctuation.

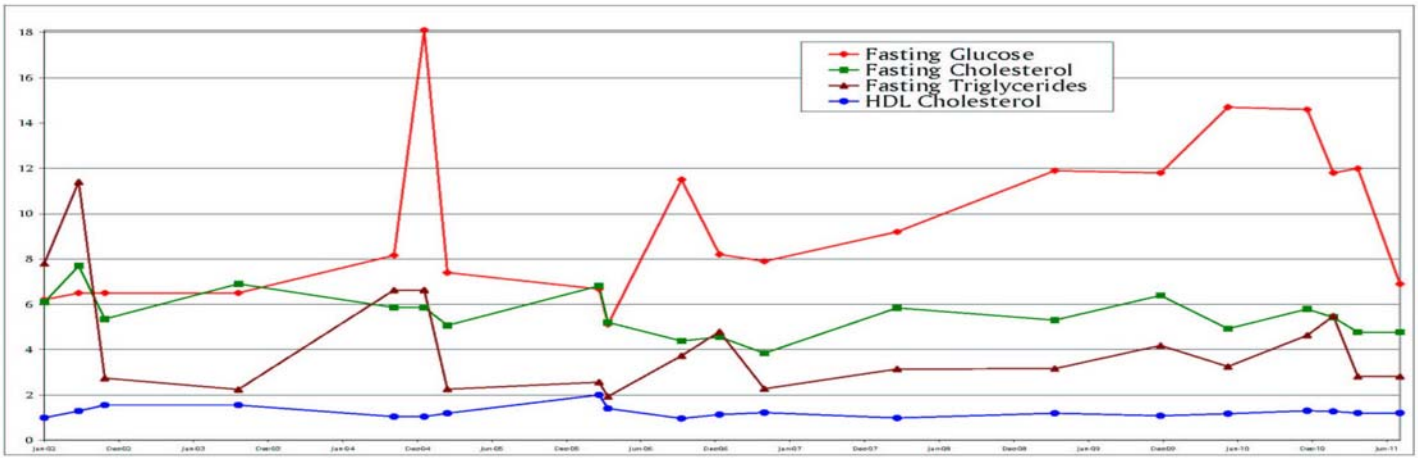
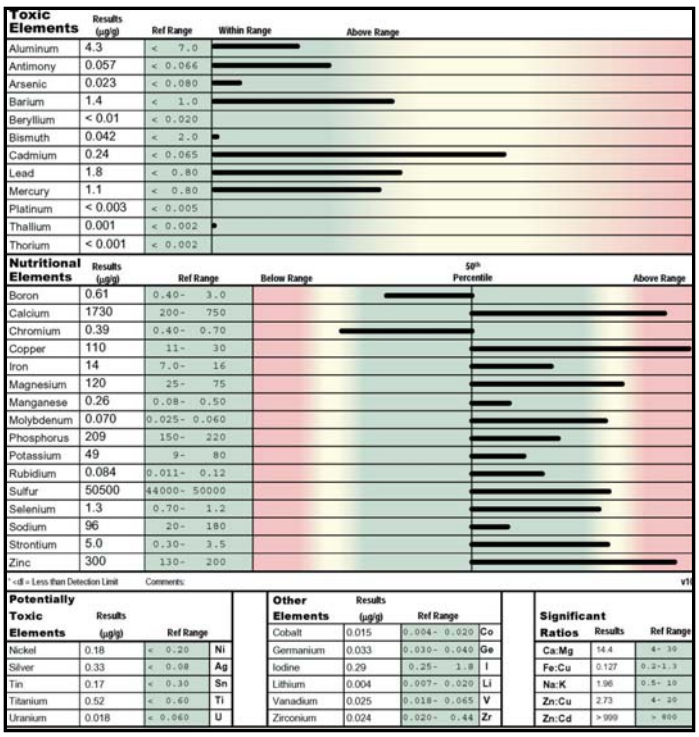


Fig 1: Fluctuation of Gana Kiritharan's Fasting Blood Glucose, Cholesterol/HDL and Triglyceride Value.

Above graph may explain how some values are changing over last few years. Though medication, food and exercise habit may be responsible for some improvement, the fact whether I was being exposed to the toxic metals environment may be the important factor determining the value.

When I realized I may be a victim of chronic form of Toxic Metal Toxicity I end up in a big confusion. First thing the reason for the toxicity may be a criminal intention. Based on previous experience I was not able to call Police immediately. Secondly that may be the first time I am hearing about Mercury Poisoning. Though we studied about several poisoning materials at my medical studies I do not remember some body taught me mercury poisoning in detail. When I approached medical professional who were treating me I experienced several denials and refusals. My family doctor told me in his 20 years of career he never seen a patient with mercury poisoning. When Ontario health care system failed order proper lab reports, I went on my own and obtained following reports which may explain my toxic status. Appropriate explanations follows the reports.

Diagnosing Toxic Metal Toxicity; Still a Challenge for Patient and Medical Profession:



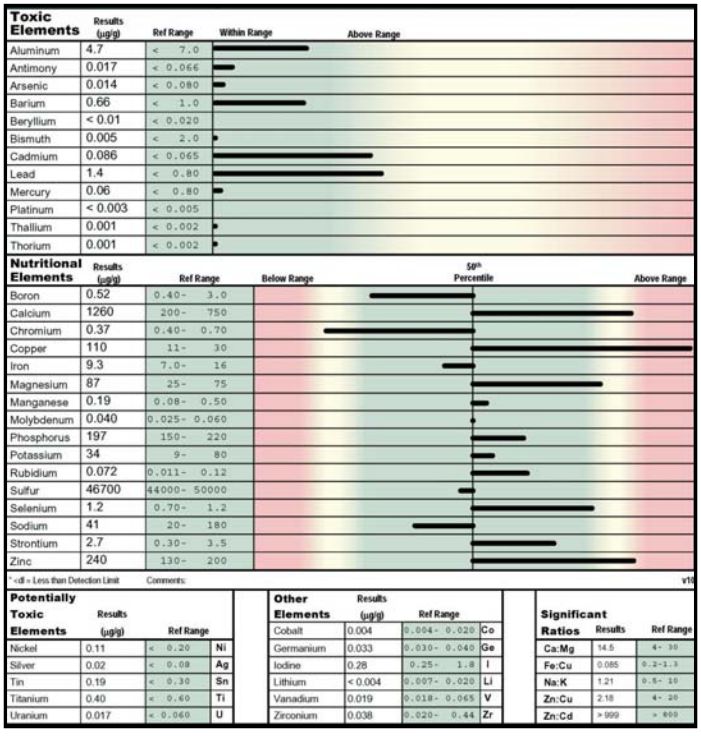
Toxic Elements	Results (µg/g)	Ref Range	Within Range / Above Range
Aluminum	4.3	< 7.0	
Antimony	0.057	< 0.066	
Arsenic	0.023	< 0.080	
Barium	1.4	< 1.0	
Beryllium	< 0.01	< 0.020	
Bismuth	0.042	< 2.0	
Cadmium	0.24	< 0.065	
Lead	1.8	< 0.80	
Mercury	1.1	< 0.80	
Platinum	< 0.003	< 0.005	
Thallium	0.001	< 0.002	
Thorium	< 0.001	< 0.002	

Nutritional Elements	Results (µg/g)	Ref Range	Below Range / 50th Percentile / Above Range
Boron	0.61	0.40- 3.0	
Calcium	1730	200- 750	
Chromium	0.39	0.40- 0.70	
Copper	110	11- 30	
Iron	14	7.0- 16	
Magnesium	120	25- 75	
Manganese	0.26	0.08- 0.50	
Molybdenum	0.070	0.025- 0.060	
Phosphorus	209	150- 220	
Potassium	49	9- 80	
Rubidium	0.084	0.011- 0.12	
Sulfur	50500	44000- 50000	
Selenium	1.3	0.70- 1.2	
Sodium	96	20- 180	
Strontium	5.0	0.30- 3.5	
Zinc	300	130- 200	

* <dl = Less than Detection Limit Comments: v10

Potentially Toxic Elements	Results (µg/g)	Ref Range		Other Elements	Results (µg/g)	Ref Range		Significant Ratios	Results	Ref Range
Nickel	0.18	< 0.20	Ni	Cobalt	0.015	0.004- 0.020	Co	Ca:Mg	14.4	4- 30
Silver	0.33	< 0.08	Ag	Germanium	0.033	0.030- 0.040	Ge	Fe:Cu	0.127	0.2-1.3
Tin	0.17	< 0.30	Sn	Iodine	0.29	0.25- 1.8	I	Na:K	1.96	0.5- 10
Titanium	0.52	< 0.60	Ti	Lithium	0.004	0.007- 0.020	Li	Zn:Cu	2.73	4- 20
Uranium	0.018	< 0.060	U	Vanadium	0.025	0.018- 0.065	V	Zn:Cd	> 999	> 800
				Zirconium	0.024	0.020- 0.44	Zr			

Fig 2.1: Test Conducted in July 2010



Toxic Elements	Results (µg/g)	Ref Range	Within Range / Above Range
Aluminum	4.7	< 7.0	
Antimony	0.017	< 0.066	
Arsenic	0.014	< 0.080	
Barium	0.66	< 1.0	
Beryllium	< 0.01	< 0.020	
Bismuth	0.005	< 2.0	
Cadmium	0.086	< 0.065	
Lead	1.4	< 0.80	
Mercury	0.06	< 0.80	
Platinum	< 0.003	< 0.005	
Thallium	0.001	< 0.002	
Thorium	0.001	< 0.002	

Nutritional Elements	Results (µg/g)	Ref Range	Below Range / 50th Percentile / Above Range
Boron	0.52	0.40- 3.0	
Calcium	1260	200- 750	
Chromium	0.37	0.40- 0.70	
Copper	110	11- 30	
Iron	9.3	7.0- 16	
Magnesium	87	25- 75	
Manganese	0.19	0.08- 0.50	
Molybdenum	0.040	0.025- 0.060	
Phosphorus	197	150- 220	
Potassium	34	9- 80	
Rubidium	0.072	0.011- 0.12	
Sulfur	46700	44000- 50000	
Selenium	1.2	0.70- 1.2	
Sodium	41	20- 180	
Strontium	2.7	0.30- 3.5	
Zinc	240	130- 200	

* <dl = Less than Detection Limit Comments: v10

Potentially Toxic Elements	Results (µg/g)	Ref Range		Other Elements	Results (µg/g)	Ref Range		Significant Ratios	Results	Ref Range
Nickel	0.11	< 0.20	Ni	Cobalt	0.004	0.004- 0.020	Co	Ca:Mg	14.5	4- 30
Silver	0.02	< 0.08	Ag	Germanium	0.033	0.030- 0.040	Ge	Fe:Cu	0.085	0.2-1.3
Tin	0.19	< 0.30	Sn	Iodine	0.28	0.25- 1.8	I	Na:K	1.21	0.5- 10
Titanium	0.40	< 0.60	Ti	Lithium	< 0.004	0.007- 0.020	Li	Zn:Cu	2.18	4- 20
Uranium	0.017	< 0.060	U	Vanadium	0.019	0.018- 0.065	V	Zn:Cd	> 999	> 800
				Zirconium	0.038	0.020- 0.44	Zr			

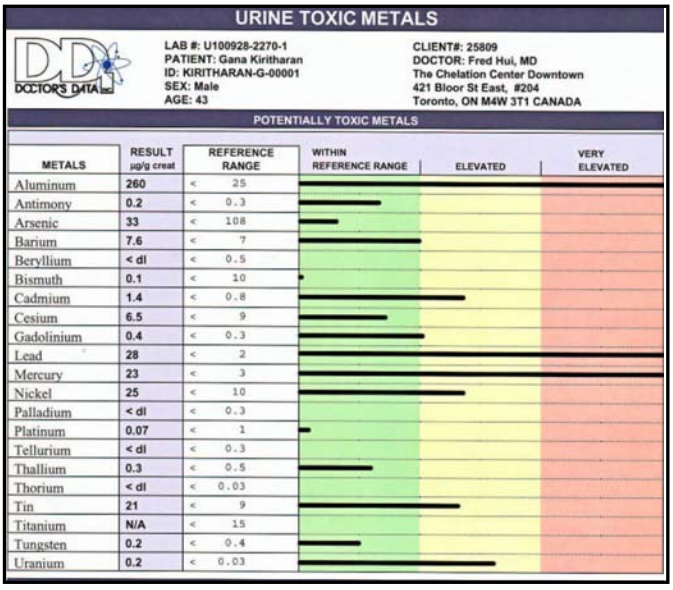
Fig 2.2: Test Conducted in July 2011 (After 1 Year)

Fig 2: Toxic Metals in Hair Mineral Analysis Report. (For Report in High Resolution Please Visit **http://www.gkiri.com)**

When a person need to be checked for chronic form of mercury or any other toxic metal toxicity, measuring either blood or random urine metal concentration may have little usefulness. 24 hours urine metal concentration may give some information but rarely performed. Checking fecal concentration of these toxic metals may explain how much being excreted through bile, again this test rarely performed. Most useful test may be analyzing mineral composition of the hair. In a normal adult person hair grow at the rate of 1 mm per day. So when you take a 1 inch hair, you are analyzing the nutritional supply for the hair follicle over a period of month. In addition hair may contain 5% or more sulfur in it. Toxic metals in the body usually circulate attached to this sulfur got concentrated in hair. This will happen even the circulation amount is a small one.

In my hair mineral analysis Toxic Metal like Cadmium, Lead and Mercury found to be concentrated in hair in large amount. After 1 year Chelation some of this toxic metal level has came down. Also my hair mineral analysis confirms some abnormality in nutritional status. While some nutritional elements in excess, some others in deficiency. This may be due to secondary effect of Toxic Metal Toxicity and the Chelation therapy followed.

When hair mineral analysis confirms some abnormality next useful test may be Challenged Urine Toxic Metal report. This test; when performed improperly; can bring serious damages for the body. If a decision is made to perform Challenged Urine Toxic Metal test, it should be done under proper medical supervision. Pictures on the following page represent various Challenged Urine test conducted on my body.



URINE TOXIC METALS

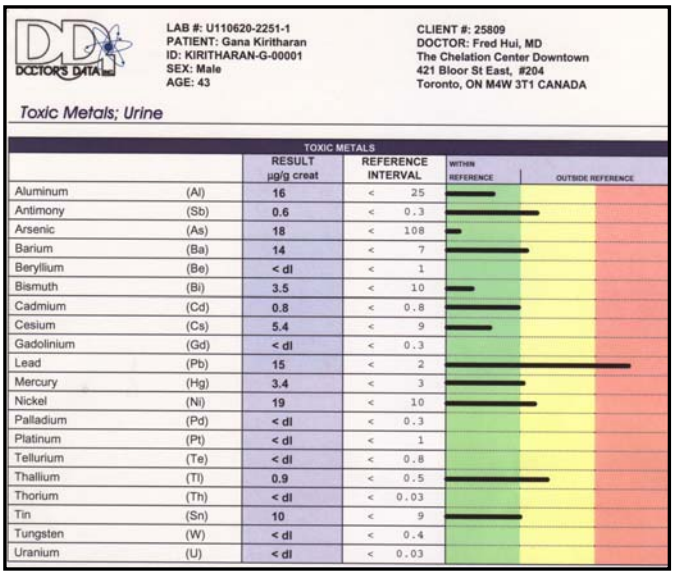
LAB #: U100928-2270-1
PATIENT: Gana Kiritharan
ID: KIRITHARAN-G-00001
SEX: Male
AGE: 43

CLIENT#: 25809
DOCTOR: Fred Hui, MD
The Chelation Center Downtown
421 Bloor St East, #204
Toronto, ON M4W 3T1 CANADA

POTENTIALLY TOXIC METALS

METALS	RESULT µg/g creat	REFERENCE RANGE	WITHIN REFERENCE RANGE	ELEVATED	VERY ELEVATED
Aluminum	260	< 25			
Antimony	0.2	< 0.3			
Arsenic	33	< 108			
Barium	7.6	< 7			
Beryllium	< dl	< 0.5			
Bismuth	0.1	< 10			
Cadmium	1.4	< 0.8			
Cesium	6.5	< 9			
Gadolinium	0.4	< 0.3			
Lead	28	< 2			
Mercury	23	< 3			
Nickel	25	< 10			
Palladium	< dl	< 0.3			
Platinum	0.07	< 1			
Tellurium	< dl	< 0.3			
Thallium	0.3	< 0.5			
Thorium	< dl	< 0.03			
Tin	21	< 9			
Titanium	N/A	< 15			
Tungsten	0.2	< 0.4			
Uranium	0.2	< 0.03			

Fig 3.1: Challenged (DMPS & CaEDTA) Urine Test on 27th September 2010



LAB #: U110620-2251-1
PATIENT: Gana Kiritharan
ID: KIRITHARAN-G-00001
SEX: Male
AGE: 43

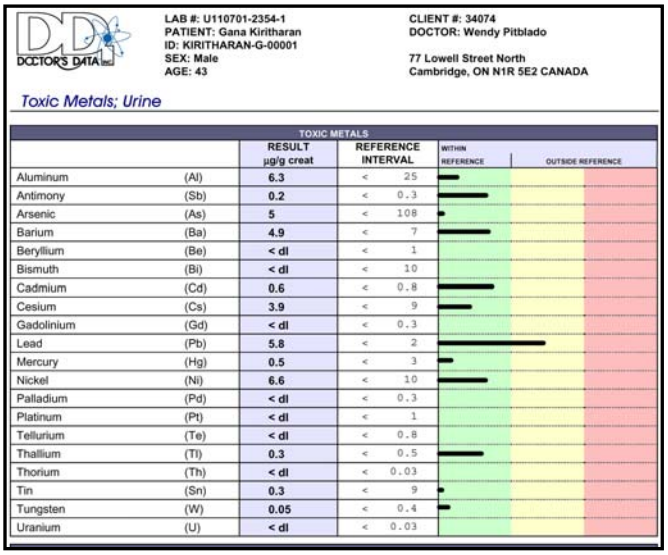
CLIENT #: 25809
DOCTOR: Fred Hui, MD
The Chelation Center Downtown
421 Bloor St East, #204
Toronto, ON M4W 3T1 CANADA

Toxic Metals; Urine

TOXIC METALS

		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE
Aluminum	(Al)	16	< 25		
Antimony	(Sb)	0.6	< 0.3		
Arsenic	(As)	18	< 108		
Barium	(Ba)	14	< 7		
Beryllium	(Be)	< dl	< 1		
Bismuth	(Bi)	3.5	< 10		
Cadmium	(Cd)	0.8	< 0.8		
Cesium	(Cs)	5.4	< 9		
Gadolinium	(Gd)	< dl	< 0.3		
Lead	(Pb)	15	< 2		
Mercury	(Hg)	3.4	< 3		
Nickel	(Ni)	19	< 10		
Palladium	(Pd)	< dl	< 0.3		
Platinum	(Pt)	< dl	< 1		
Tellurium	(Te)	< dl	< 0.8		
Thallium	(Tl)	0.9	< 0.5		
Thorium	(Th)	< dl	< 0.03		
Tin	(Sn)	10	< 9		
Tungsten	(W)	< dl	< 0.4		
Uranium	(U)	< dl	< 0.03		

Fig 3.2: Challenged (DMPS & CaEDTA) Urine Test on 16th June 2011 (After 10 Months)



LAB #: U110701-2354-1
PATIENT: Gana Kiritharan
ID: KIRITHARAN-G-00001
SEX: Male
AGE: 43

CLIENT #: 34074
DOCTOR: Wendy Pitblado

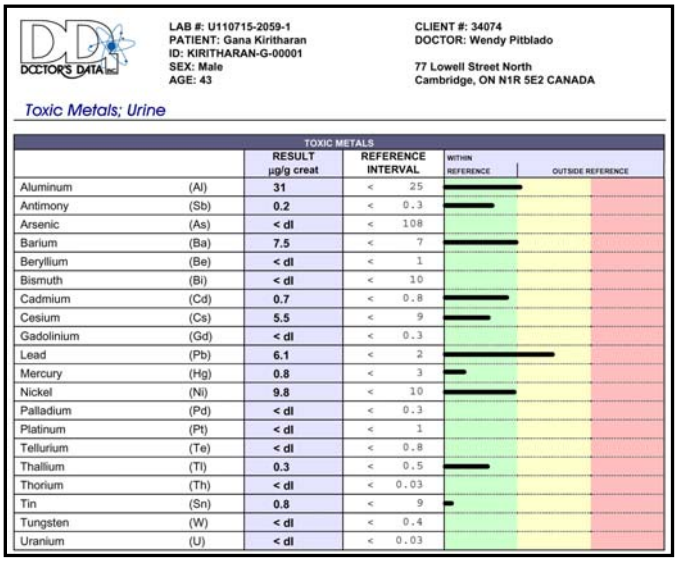
77 Lowell Street North
Cambridge, ON N1R 5E2 CANADA

Toxic Metals; Urine

TOXIC METALS

		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE
Aluminum	(Al)	6.3	< 25		
Antimony	(Sb)	0.2	< 0.3		
Arsenic	(As)	5	< 108		
Barium	(Ba)	4.9	< 7		
Beryllium	(Be)	< dl	< 1		
Bismuth	(Bi)	< dl	< 10		
Cadmium	(Cd)	0.6	< 0.8		
Cesium	(Cs)	3.9	< 9		
Gadolinium	(Gd)	< dl	< 0.3		
Lead	(Pb)	5.8	< 2		
Mercury	(Hg)	0.5	< 3		
Nickel	(Ni)	6.6	< 10		
Palladium	(Pd)	< dl	< 0.3		
Platinum	(Pt)	< dl	< 1		
Tellurium	(Te)	< dl	< 0.8		
Thallium	(Tl)	0.3	< 0.5		
Thorium	(Th)	< dl	< 0.03		
Tin	(Sn)	0.3	< 9		
Tungsten	(W)	0.05	< 0.4		
Uranium	(U)	< dl	< 0.03		

Fig 3.3: Challenged (DMSA) Urine Test on 30th June 2011 (After Two Weeks)



LAB #: U110715-2059-1
PATIENT: Gana Kiritharan
ID: KIRITHARAN-G-00001
SEX: Male
AGE: 43

CLIENT #: 34074
DOCTOR: Wendy Pitblado

77 Lowell Street North
Cambridge, ON N1R 5E2 CANADA

Toxic Metals; Urine

TOXIC METALS

		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE	OUTSIDE REFERENCE
Aluminum	(Al)	31	< 25		
Antimony	(Sb)	0.2	< 0.3		
Arsenic	(As)	< dl	< 108		
Barium	(Ba)	7.5	< 7		
Beryllium	(Be)	< dl	< 1		
Bismuth	(Bi)	< dl	< 10		
Cadmium	(Cd)	0.7	< 0.8		
Cesium	(Cs)	5.5	< 9		
Gadolinium	(Gd)	< dl	< 0.3		
Lead	(Pb)	6.1	< 2		
Mercury	(Hg)	0.8	< 3		
Nickel	(Ni)	9.8	< 10		
Palladium	(Pd)	< dl	< 0.3		
Platinum	(Pt)	< dl	< 1		
Tellurium	(Te)	< dl	< 0.8		
Thallium	(Tl)	0.3	< 0.5		
Thorium	(Th)	< dl	< 0.03		
Tin	(Sn)	0.8	< 9		
Tungsten	(W)	< dl	< 0.4		
Uranium	(U)	< dl	< 0.03		

Fig 3.4: Challenged (ALA & DMSA) Urine Test on 14th July 2011 (After Two Weeks)

Fig 3: Challenged Urine Toxic Metal Reports. (For Report in High Resolution Please Visit **http://www.gkiri.com)**

Fig 3.1 give the challenged urine test conducted at the beginning of the treatment. There is large amount of toxic metals being excreted through urine. This may be due to three reasons. 1) As I just started treatment, the Toxic Metals in my body started to come out. 2) I may got poisoned severely just before this period. 3) As I did not take NAC in large amount, Kidney may me the only source of excretion of these toxic metals (Only small amount through Liver). Fig 3.2 and 3.3 gives Challenged urine test conducted after 10 months of Chelation (One by IV medication (DMPS & CaEDTA) and other by Oral medication(DMSA)). It may explain how the toxic metals being excreted from the body has reduced because of the Chelation program including large amount of NAC (Which may help to remove Toxic Metals through Liver). These picture also explain difference between IV and Oral Chelation therapy. Though, IV can bring out the Toxic Metals fast out of the body, Oral Chelation also equally effective. Fig 3.4 gives challenged urine test conducted after taking Alpha Liphoic Acid (ALA) for 24 hours before starting the test. This test may explain how Aluminum like toxic metals secretion got increased by ALA.

My Battle with Toxic Metal Poisoning:

I do not know how long I am being poisoned with these toxic metals and what are the possible causes; but for last one year; as Ontario Medical and Judicial system are failing to come forward to protect me; I am fighting a personal battle with a series of poisoning attempts with criminal intention.

The possible incidents of poisoning attempts, changes in treatment of toxic metal toxicity and diabetes and changes in fasting blood glucose value of 2010 - 2011 are given on following table and graph. I will divide the last one year as following time frames for easy understanding. You may see high fluctuation of my Fasting Blood Glucose value. Though various variables in the treatment of diabetes and toxic metal toxicity can caused these, most logical explanation may be I was exposed to toxic metal environment.

TIME FRAME	POISONING SITUATION DETAILS	CHELATING THERAPY FOLLOWED	DIABETIC MEDICATION FOLLOWED	CHANGE IN FASTING GLUCOSE
May 19 2010 – Aug 17 2010	I started to avoid suspected poisoned food. There may be couple of attempts. On 5th of August 2010, I received the First confirming medical report (Hair Mineral Analysis). On August 17th 2010 I tried to make a Police Complaint to Toronto Police Services (TPS) but they failed to accept my complaint.	Started supportive medication on 22nd June 2010 and started DMSA from 6th July 2010. I added NAC to the program one week after. DMSA dose just 2 – 8 mg/Kg body weight per day. (Recommended 30 mg/Kg)	For few weeks **Metformin 3000mg Rosiglitazone 8 mg Glyburide 90 mg** Then **Metformin 2000mg Glyburide 60 mg**	From 14.7 mmol/L came down to 7.4 mmol/L on
Aug 18 2010 – Sep 26 2010	As I was abandoned by TPS there may be several sever attempt to Poison me through various means.	Continuous supportive medication. Continued DMSA and NAC till 13th Sep 2010.	Maintained **Metformin 2000mg Glyburide 60 mg**	Went up and Reached 17.4 mmol/L
Sep 27 2010 - Nov 25 2010	As I went into formal medical care number of attempts to poison me may reduced but there may be one or more attempts.	Challenged urine test was conducted on 27 Sep (DMPS, CaEDTA). I restarted DMSA after 3 days .	Maintained **Metformin 2000mg Glyburide 60 mg**	Reached a Value below 10mmol/L but soon went up.
Nov 26 2010 – Jan 24 2011	Number of Attempt to Poison me may have reduced, but few attempts may be there.	Increased DMSA dose to 15 mg/Kg. Started to add NAC in higher doses.	**Metformin 2000mg Insulin 16 Units/Day**	Stabilized between 10 – 12 mmol/L
Jan 25 2011 – Mar 14 2011	I started to experience unusual postal delays. DMSA supply also interrupted for two weeks. Attempts to Poison me may have increased.	Skipped one week DMSA. High dose of NAC. I started to add ALA to the chelating program.	**Metformin 2000mg Insulin 20 - 26 Units/Day**	Went up and stay between 10 – 14 mmol/L
Mar 15 2011 – April 30 2011	There may be One or More sever attempt.. I took more precaution (sealed ventilation duct of my room)	I took DMSA and ALA.. Increased the dose of Buffered Vit C and NAC.	**Metformin 2000mg Diet, Exercise, Herbal**	Stay between 10. – 16 mmol/L
May 1 2011 - Sep 30 2011	The poisoning attempts may have reduced or even stopped.	I maintained DMSA. High dose of NAC and Vit C. Conducted 3 different Challenged Urine tests.	**Metformin 2000 mg Diet, Exercise.**	Stayed around 7 mmol/L
Oct 1 2011 – Dec 12 2011	There may be attempt to poison with Arsenic.	DMSA, NAC, Vit C, ALA, and Coriander	**Metformin 2000 mg Diet, Exercise.**	Went Above 9 mmol/L

Table 3: Gana Kiritharan's battle with Toxic Metal Poisoning during 2010 – 2011 period.

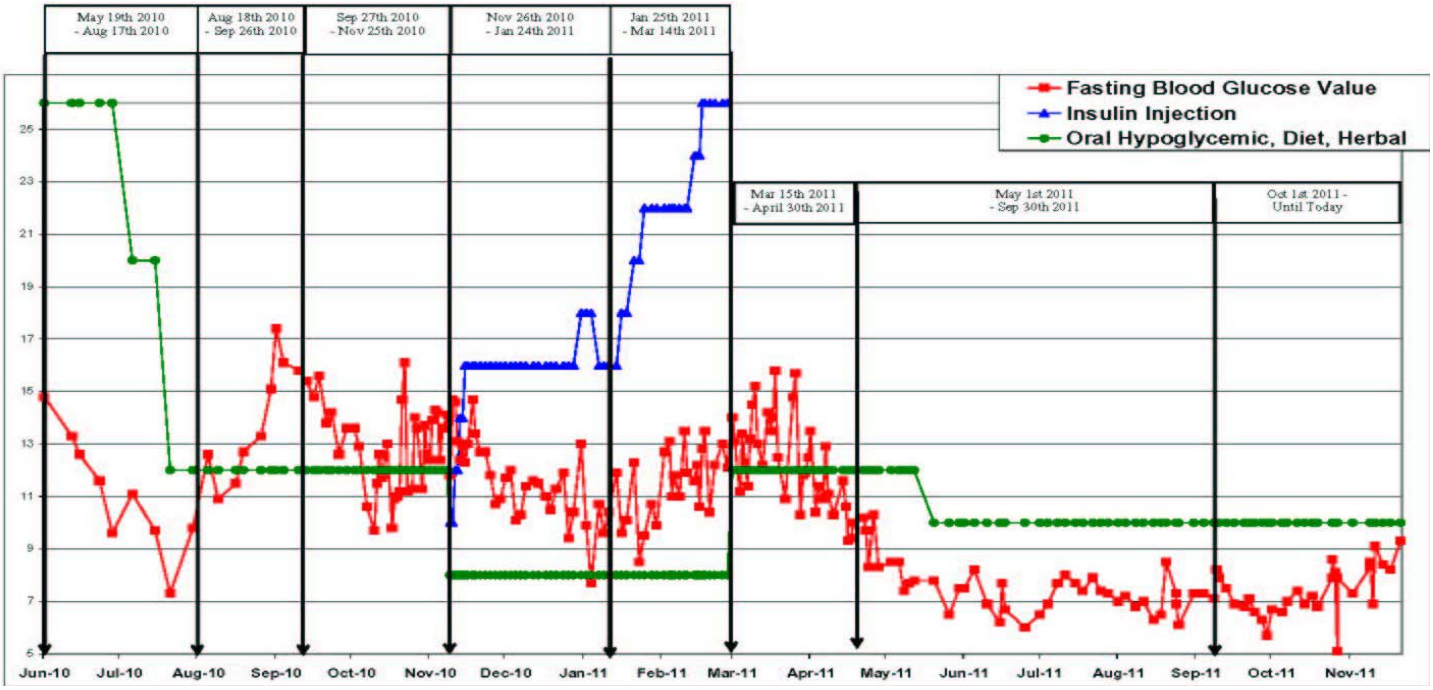


Fig 4: Gana Kiritharan's Battle with Toxic Metal Poisoning during 2010 – 2011.

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Pathology of Toxic Metal Toxicity [1, 2, 3, 4]:

Let try to have an understanding about how toxic metals causes toxicity to human body so that how toxic metals may be the cause of my medical problems may be understood. Toxic metals cause toxicity to human body mainly by interfering in protein synthesis and their functions. Heavy metals react with proteins and forms highly insoluble sulfides[3].

To explain this in details; Proteins are made of 20 or more amino acids. In these amino acids two or more (Methionine, Cysteine) contains sulfur in their composition. The sulfhydryl (-- SH) group of Cysteine is important in two ways. Usually proteins are synthesized as a single long polypeptide chain and then folded into different shapes and develop their functionality. The sulfhydryl group of Cysteine amino acid play important role in the folding of polypeptide chains. Cysteine molecules in two different location of single polypeptide interact and form disulfide bonds (--S—S--) bonds and results in folding of polypeptide chains. The heavy metals are by binding with sulfhydryl groups of Cysteine molecules prevents proper folding of polypeptide chains. Again many enzymes has a one or more free sulfhydryl groups which react with nutritional elements like Zn^{++} or other coenzymes. Heavy metals may interact with this free sulfhydryl groups of enzymes and other proteins, resulting in loss of their functions.

Above explanation may help to understand why I am claiming toxic metals as the important causes of my medical problems. If you look at Insulin or Insulin Receptors both first synthesized as a single polypeptide chain then folded and held in their functional shape using disulfide bonds. Then a potion of this polypeptide chain gets removed and receives functionality. So Cadmium and other Toxic Metals may interfere with this folding of theses proteins one or other way. Fickova M et al, (1) and Lei L J et al, (2) already demonstrated the influence of Cadmium on Insulin and Insulin Receptor synthesis.

In their research Lei L J et al, (2) demonstrated cadmium do not affect serum insulin level. The reason may be even when cadmium interfere with Insulin synthesis pancreas has a high reserve of production and capable of pumping out normal insulin level. However synthesis and availability of Insulin receptors are markedly determined by Cadmium and other toxic metal poisoning. This was demonstrated by Fickova M et al, (1) in detail in their research. The reason may be two. First there may not be enough reserve of synthesis of Insulin Receptors as Insulin. Secondly the disulfide bond between the first and second pair of alpha and beta chain may be more susceptible for toxic metal toxicity. We can leave the responsibility of finding correct answer to future biochemistry research. My treatment experience also support above research finding. During my treatment, when I tried to take Insulin injection, it not only failed to bring any better control but also gave complications which can be attributed to Hyperinsulinemia.

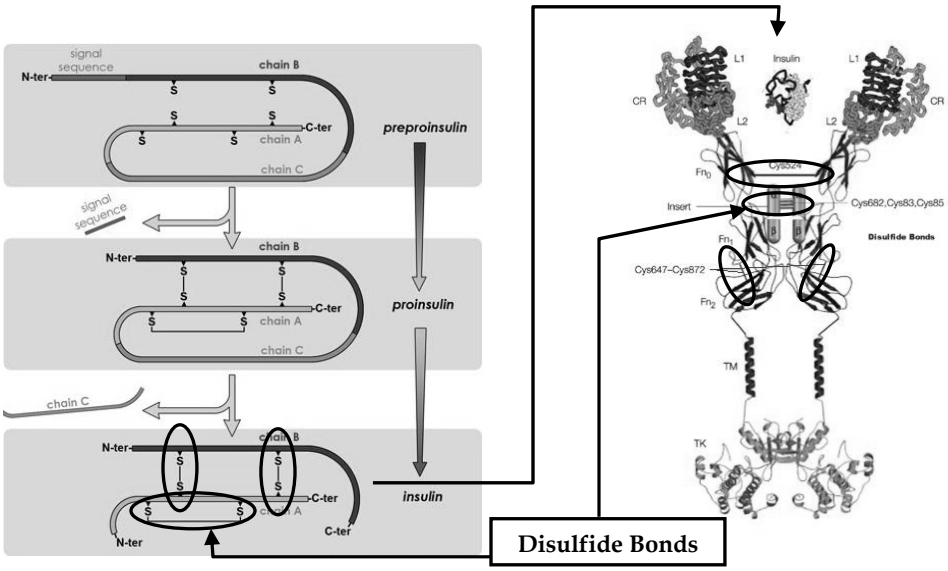

Fig 5: Insulin and Insulin Receptor.

Picture Resource:

1) **Beta Cell Biology Consortium (2004).**
2) **Pierre De Meyts & Jonathan Whittaker : Structural Biology of Insulin and IGF1 receptors : Implication for drug design : Natural Reviews of Drug Discovery 1, 769 – 783 (October 2002).**

Chelation; Treating Toxic Metal Toxicity [6, 7]:

After I started treatment for my Toxic Metal Toxicity I experienced high level of fluctuation of my Fasting Blood Glucose value. During last 1 year my fasting serum glucose value went above 16 mmol/L twice and it went below 8 mmol/L twice. As most of this time period I maintained a stable dose of oral hypoglycemic drugs, I blame level of toxic metal toxicity in my body as the main reason for this fluctuation. To understand this you should have a better understanding of the word Chelation and other antitoxins which helped me to achieve improvements in blood glucose level.

By definition Chelation means "The formation or presence of bonds (or other attractive interactions) between two or more separate binding sites within the same ligand and a single central atom.[9]" Usually this central atom is a metal ion. In medical sense Chelation means removing toxic metals from body by using medical substances. The medical substances used in Chelation form two or more bonds with toxic metals and help to remove them through liver or kidney.

Though I am taking specific Chelation medications which may helped to remove these toxic metals from body, I am also taking several non-specific antitoxins which may help to reduce the general toxic level of my body. I am taking Vitamin E 800 IU or more. A Normal Vitamin tablet contains 50 IU. Also I am taking 3000 mg or more Vitamin C. Normal dose id 90 – 500 mg. Also I am taking high dose of Vitamin A, B and D. I am also taking higher dose of minerals but excluding Fe and Cu. The specific chelating medication I am taking can be divided into two groups. First group is synthetic medical substances. The synthetic medical substance I am taking is DMSA (Meso-2-3-dimercaptosuccinic acid). It has two sulfhydryl (-- SH) groups which form tight bonds with toxic metals and remove them safely through kidney. Important information about these synthetic chelators, they usually do not go into the cells. They only remove toxic metals from interstitial fluids or toxic metals circulating in the blood. The second group is more natural substances. The first one I am taking is NAC (N-Acetylcysteine) which is a precursor of Glutathione. Glutathione is the body's natural chelators. Unlike synthetic chelators, Glutathione go into the cells and bring out the toxic metals. Also liver Glutathione level is important in determining amount of toxic metals being excreted through liver. NAC helps to maintain good liver Glutathione level and helping in excretion of toxic metals through liver.

When I was taking DMSA alone, without NAC the fasting blood glucose value dropped below 10 mmol/L. But when I took DMSA with NAC in proper way the value went below 8 mmol/L twice. When I was taking low dose of DMSA, but tried to take more NAC, I experienced sudden increase of Fasting and Post Prandial blood glucose levels. It may be because NAC mobilized toxic metal stores from intracellular space and caused increased toxicity.

Another naturally occurring substance I am trying to add to my chelating program is ALA (Alpha Liphoic Acid). ALA has a property of crossing blood brain barrier. If taken at early stage of chelating program, it may bring toxic metals into the brain. I started to add it only after 6 months of chelating program of DMSA and NAC. After I started to add ALA to my Chelation therapy I experienced easing of my CNS symptoms of Toxic Metal Toxicity. But my fasting blood glucose level started to go up by small level. It may be due to ALA mobilizing intracellular and CNS stores of these Toxic metals.

Few important questions remain unanswered. Why I am not able to achieve a sustained control of diabetic problem and whether treatment for Toxic Metal Toxicity will give me a long lasting cure from Metabolic syndrome. The answer may depend on two issues. Whether I will get an environment free of these toxic metals to live and whether I will receive necessary financial resources to get best possible treatment for theses toxic metal toxicity. Latest attempt to poisoning me may have happened on March – April 2011; that is almost one year after I discovered I am being poisoned with criminal intention. Many times I left with one or two dollars in my pocket waiting for next pay check. In year 2004 and 2007 I experience interference in my employment opportunity and hours of work as well. Several experts recommend Infrared Sauna as it will help to remove toxic metals through skin. I like to by one of this (may cost around CAD $ 1500.00) but I do not have enough money.

Women's Medical Problems [3,9] :

Why I am claiming Toxic Metal Toxicity is a common but ignored medical problem is my mother's possible long term experience with this toxicity. My mother's first chronic medical problem may be Bronchial Asthma. She probably got this problem in her 30s. Next problem she experienced, sleep disturbances and disturbing dreams which may continue through out her life. Again during her late 30s on one night she experienced a panic attack and family doctor may have to give some thing to make her sleep. My mom was a vegetarian but as doctors advised her, she started to eat non-veg food from early 40s and her symptoms may get eased to some extent. Next medical problem she experienced in her 40s is Hypothyroidism. During her 40s and 50s she developed frozen shoulder and Herpes Zoster. From early 50s she may experienced receding gums. And finally on her 61st year of age she died of (antero septal) myocardial infarction.

When I searched for the possible cause for her toxicity, I realized she may get it from fire woods used in the kitchen. If my conclusion is correct many other women who were exposed to kitchen smoke also suffered toxic metal toxicity and experienced related medical problems. When searched for more evidence I found two evidences. First one is in Tamil Literature. Thruvalluvar; the person considered philosopher of Tamil wisdom; when describing woman leadership uses following words.

"No virtuous deed, no seemly wealth, no pleasure, rests with them who live obedient to their wives' behests."

If you look at Tamil Literature similar comments or stories describing woman psychology can be found in several places. Toxic Metal toxicity can cause several psychological disturbances on victims. Feeling of insecure, feeling of suspicious and panic attacks all associated with Cadmium and other toxic metal toxicity.

Second Evidence I found is menopausal syndrome suffered my woman share several similar clinical features with toxic metal toxicity. The clinical features of Menopausal syndrome, hot flushes and night sweats, difficulty falling asleep, cognitive difficulties, depression, irritability and decalcification of bone all share with toxic metal toxicity. If we can answer how estrogen may protect woman till menopause it may open new intellectual discussion. Estrogen is a steroid synthesized in body from cholesterol. It has a 2 free – OH group. Whether it form oxide with toxic metals and work as a chelators or –CH3 (Methyl) compounds released on synthesis of estrogen give some temporary protection against these toxic metals is intellectual question for future biochemical researches.

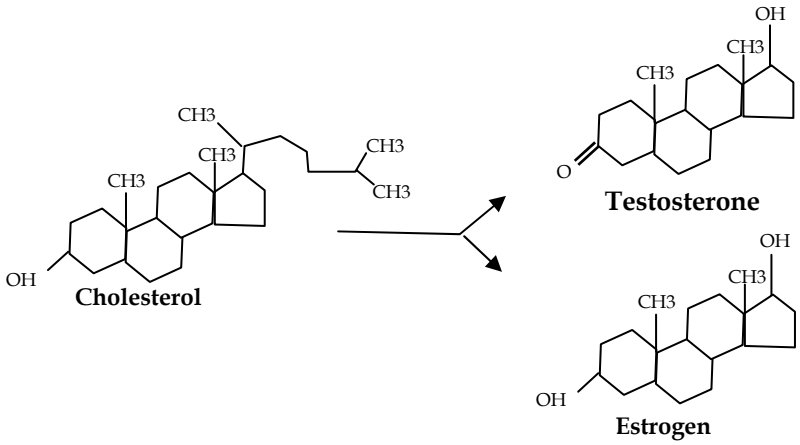

Fig : 6 Estrogen Synthesis

Unacceptable Level of Negligence:

When I realized that I am a victim of poisoning with mercury with criminal intention one of the major challenges I faced on properly diagnosed with this toxic metal toxicity and receiving treatment is unacceptable level of negligence in health care profession regarding this medical situation. When I realized I may be poisoned with mercury with criminal intention I was not able to call police immediately. The well organized nature and powerful authority of people behind this criminal conspiracy has prevented me even expecting an accountable court order from Ontario Judiciary. Even though I tried to make police complaint on 17th August 2010; when I got the first confirming medical report; I was not able to give any big hope on Ontario Judiciary to protect me from these chain of criminal offences I was experiencing. I do not know the reason, whether it is institutionalized racism or high level of corruption, Ontario Judiciary has shown high level of ignorance, irresponsibility and incompetence on protecting me from the chain of criminal activity I am experiencing.

On Medical side, when I called Poison Center at 416 813 5900, the only help (?) I received an advice of talked to

your family doctor. My family doctor may ordered for a wrong medical report (Blood Mercury) to check whether I am suffering from chronic form of mercury poisoning. The received blood mercury report gives value 15.4 nmol/L. This may confirm certain amount of toxicity. Despite several public documents giving a reference value of 0 – 3 nmol/L my report give a value of < 18.0 nmol/L as the reference range. This lead many medical professionals to interpret the report as normal. My argument that mercury is an unwanted toxic metal and 0 nmol/L may be the healthy level was unheard by medical professionals. This forced me to walk away from Ontario Health Care system and spend some of my pocket money and obtain health report analyzing my hair and urine which explained my toxic status.

Even after obtaining the health report confirming my toxicity, the problem may not be solved. A consultant refused to look into the report and described chelating doctors as money makers and my cry is often described as various psychiatric problems. Why is this refusal and denial? If you look back at the history, you may find intellectuals or other forms of social leadership (religious, political and business) had refused to accept the truth told to them on time. Let us discuss the reasons for the challenges faced by scientist to bring out the truth so that not only my medical problem got accepted and I and similar patients receive better treatment but future scientist can improve knowledge with fewer hindrances.

The first challenge is vision. Human vision has a limitation and our explanations about the environment mainly based on our vision. Some people born with gift of seeing beyond the normal vision and give better explanation about our environment and its problems. Another thing increases our vision is technology. For an example telescope helped to have a better look into the universe and give a detailed explanation. What is the vision problem with Toxic Metal Toxicity? The same way Telescope brought better understanding about universe, it is the microscope brought better understanding about smaller objects. There was a time scientist fought with religion to dissect human body and give better explanation for gross anatomy. Then the next level of battle may have happened when scientist tried to examine human body parts under microscope. Today problem we may have to go to the next level of analysis that is biochemical level. The technology we have today has limitation on giving a black and white (or colour full) picture of what is happening at biochemical level inside the cells. This lead to make several conclusions just based on the clinical presentation. Only people with deeper vision can understand and explain what happening at biochemical level inside a cell and help to move medical profession to next level of thinking.

Another challenge is improper distribution of social authority. When a society organized as people with better vision in one corner without much authority and people with fundamental thinking with more authority in another corner, a conflict results and truth got suppressed for a long period of time. The best documented conflict in humanity may have happened between Galileo and Rome Court. Giving the authority to the people with better vision may look like a solution, but even in modern time this rarely happens. In Thailand authority was given for a person with a vision only after the 2004 Tsunami, not before it. If you deeply look in to this problem, nowadays, weather it is mad cow disaster in UK or Tsunami in South Asia it was the fear of loosing business and short term profit motive which may put barriers on scientist bringing out truth on time and education public about an impending disaster. Today religions may have lost the social authority but the authority did not went to the scientist who need it. Instead it went to the major business institution who continuously trying to put barriers for scientist from bringing out truth for several unacceptable reasons and putting humanity in face of disaster again and again.

Another important challenge is, understanding the word science. Important argument put forward by people who disagree with Toxicity due to Toxic Metal Toxicity is, several scientific experiments fail to show any benefit from Chelating. There are several reasons for this misunderstanding. Important one, there is not jet an established and universally accepted way of treating the problem. Today medical profession has successfully established treatment methods for many medical problems. Most of these treatments are last from few days to few months. But treatment for Toxic Metal Toxicity need to followed from several months to few years. Several studies conducted regarding toxic metal toxicity followed a shortened duration of chelating therapy which may failed bring any big benefit.

An important argument I want to put forward here, what we understood by the word 'Science'. Science is a method of analysis the environment and finding solutions for it problems. It disagree any fundamental arguments and accept experiments as the way of finding solutions for the problems. But expecting all the solutions for our problems should come through proper scientific experiments is creating another religion in the name of science. If a simple observations and logical explanation can help us to bring solution for a problem, then waiting for a scientific experiment is just waste of time and money. From Aristotle to Galileo may have conducted several experiments to understand how objects were falling towards earth. But it was Newton's simple observation of a falling apple and logical explanation for it helped to define "Gravity". Today several stories of patients who benefited from treatment of Toxic Metal Toxicity may make any scientific experiments as unnecessary waste of money.

Freya Koss

March' 1998: After Having a Tooth Drilled & Filled with Mercury Amalgam

April' 2002: Four Years After Removal of Amalgam & Detox

Fig 7: Damages due to Mercury Poisoning and Recovery after Chelation. (For More Details Please Visit: http://www.toxicteeth.org)

Do Not Wait for a Disaster:

Important question arises about these toxic metals is, how common this is as a human health problem. Usually modern industrialization is blamed for exposing human health to these toxic metals but I as explained earlier when I searched for logical explanation for my mother's possible toxicity with these toxic metals I had to blame fire wood used in kitchens as the possible source. We have to accept the fact that as how microorganisms are common in our environment, toxic metals also unavoidable reality. But as humanity has established a successful way of preventing, diagnosing and treatment protocols for microorganisms, a preventing, diagnosing and treatment protocols also can be achieved for toxic metal toxicity. All can happen only after medical profession come forward to accept it as a common medical problem. When I searched WHO for correct ICD classification of my medical situation, I may fail to find a disease category saying "Chronic form of Toxic Metal Toxicity".

Important information about Toxic Metal Toxicity is difficulty in detoxifying them. Several harmful substances for human health can be easily detoxified. For an example either TB Bacilli or AIDS Virus can be killed easily by bright sun light. But when talking about detoxifying Toxic Metals, if we use the word impossible it may not be a mistake. As base elements in chemical structure they try to stay in the toxic form forever. Even we dilute them with air and water, several plants, animals and fish has the capacity to concentrate these toxic metals in their body and put it back in to the human food chain. If we really want to protect Human Health from these Toxic Metals, we may need an organized and established long term strategy to decrease the concentration of these toxic metals from our environment.

This may be why a Health Canada document titled "The Risk of Mercury Poisoning" says "The Government of Canada is working in number of areas to reduce the use and release of mercury into the environment." But unknown people are adding unspecified amount of these Toxic Metals to Toronto Eco Environment. I could not find some body, who can ask a question at these people or stop their irresponsible act.

I am calling Health Care professionals and managers not just in Canada but as a whole health care profession to not to wait for a disaster to happen but accept this medical situation and come forward to find out better prevention, diagnosis and treatment protocols.

Further Reading and Reference:

1. Ficková M, Eybl V, Kotyzová D, Micková V, Möstbök S, Brtko J: Long lasting cadmium intake is associated with reduction of insulin receptors in rat adipocytes: Institute of Experimental Endocrinology, Centre of Excellence of EU, 833 06 Bratislava, Slovakia, ueenfick@savba.sk.
   Abstract
   The effects of chronic cadmium exposure on adipose tissue have not been extensively reported. In adult Wistar male rats we investigated in vivo effect of 6 weeks lasting cadmium intake in drinking tap water (CdCl2 9,7 mg/l). Insulin receptors in isolated adipocytes from epididymal fat and glucose transporter protein GLUT4 content in fat tissue plasma membranes were determined. Control and Cd treated rats had similar water intake with subsequent heavy augmentation of Cd content in liver of experimental animals. In comparison with controls, Cd intake did not influence body mass increment and fat cell size, but significantly increased serum glycemia and moderately elevated insulinemia. Cadmium intake significantly reduced (approximately 50%) both, total insulin receptors number and density of the receptors in fat cells. No differences in the content of GLUT4 in crude plasma membranes of adipose tissue were observed. Diminished insulin receptors in adipocytes could account for diabetogenic effect of long lasting cadmium intake.

2. Lei LJ, Jin TY, Zhou YF: Insulin expression in rats exposed to cadmium : Department of Occupational Health, School of Public Health, Fudan University, Shanghai 200032, China.
   Abstract
   Eighteen adult SD rats were administered cadmium subcutaneously (0.5, 1.0, and 2.0 mg/kg x bw). The effects on endocrine of pancreas were assessed. The levels of cadmium and zinc in pancreas, blood and urine glucose, serum insulin and urine NAG (N-acyetyl-beta-glucosaminidase) were determined. The gene expressions of metallothionein (MT) and insulin were also measured, and the oral glucose tolerance tests (OGTT) were carried out. The contents of cadmium in pancreas in cadmium-treated rats were higher than that in the control group, which was associated with slight increase of zinc in pancreas. Cadmium-exposed rats (1.0 and 2.0 mg/kg x bw) demonstrated a marked glucose intolerance. But the levels of serum insulin did not change significantly after cadmium administration, and the UNAG had no change in Cd-treated group. The gene expression of insulin decreased in 1.0 and 2.0 mg/kg x bw cadmium-exposed groups, compared with the control group. The expression of MT-I was higher in the groups exposed to 1.0 and 2.0 mg/kg x bw cadmium while the expression of MT-II was higher in the group exposed to 2.0 mg/kg x bw cadmium. Cadmium may be accumulated in the pancreas, resulting in the change of the expression of insulin, MT-I and MT-II genes. Cadmium can influence the biosynthesis of insulin, but does not induce the release of insulin. The dysfunction of pancreas occurs earlier than that of kidney after administration of cadmium.

3. Bhagavan, N.V : Medical Biochemistry – Fourth Edition : Harcourt/Academic Press : 2001.

4. Lieberman, Michael ; Marks, Allan : Illustrations by Chansky, Matthew : Marks' Basic Medical Biochemistry – Third Edition : Lippincott Williams & Wilkins, Wolters Kluwer Health : 2009.

5. Silverthorn, Dee Unglaub : Human Physiology – An Integrated Approach – Fourth Edition : Pearson Benjamin Cummings : 2009.

6. Cutler, Andrew Hall : Amalgam Illness – Diagnosis and Treatment : 1999.

7. PAC : 1994 : 66 : 1077 (Glossary of Terms Used in Physical Organic Chemistry (IUPAC Recommendations 1994)) on page 1094.

8. Pierre De Meyts & Jonathan Whittaker : Structural Biology of Insulin and IGF1 receptors : Implication for drug design : Natural Reviews of Drug Discovery 1, 769 – 783 (October 2002).

9. Mazza, Danielle : Woman's health in general practice – Second Edition : Churchill Livingston : 2011.

Details of Distribution of Articles "Chronic Form of Toxic Metal Toxicity and other Chronic Medical Problems" and "Gana Kiritharan's Explanation of Type 2 Diabetic Mellitus and Related Disorders of Human Health"

S. No	Date	To Whom	Title	Mode	Response
Article "Chronic Form of Toxic Metal Toxicity and other Chronic Medical Problems."					
1	Year 2011	Ministry of Health Ontario and Canada	Health Service Administrators	Registered Post	Minister of Health Canada Responded Advising to Talk to Minister of Ontario; No response from minister of Ontario.
2	Year 2011	More than 5 Medical Professionals	Tamil Community Family Doctors working in Scarborough Area	Drop at Reception of their Clinics	No Serious response.
3	Year 2011	Tamil One and CTR	Tamil Media Organizations in Toronto area	Drop at their Reception	No Response
Articles "GK's Explanation of T2DM" and "Chronic Form of Toxic Metal Toxicity and other Chronic Medical Problems."					
1	21st Dec 2017	Prof, Benjamin Ong	Registrar, Singapore Medical Council	Fax	Email on January 4th Advising to Communicate with Singapore Ministry of Health
2	20th Dec 2017	Dr Jayshree Mehta Dr C V Bhirmanandam Prof. Siri Bhawan Siwach CC: Vice Chancellor MGR	Office Bearers of Indian Medical Council	Email	No Response Received
3	21st Dec 2017	Dr.Tedros Adhanom Ghebreyesus	WHO Director General	WHO Web Site	No Response Received
4	21st Dec 2017	Dr. Pirapakaran	Private Practitioner – Theni (India)	Local Indian Courier	Informed First Response Should Come from A Medical Council (India or Singapore) then Only We can move further.
5	23rd Dec 2017	Dr Jayshree Mehta	President Indian Medical Council	Speed Post (Indian Postal Services)	Initially Able to Track the Package but after Returning to Canada unable to track the package.
6	23rd Dec 2017	Dr.Tedros Adhanom	WHO Director General	DHL India	Package Received By: Able Martinez (No Other Response)
7	27th Dec 2017	Dr. Manokaran	Neurologist Mathurai	Personally Hand over	Will give it to his Endocrinologist Friend. No Response so far.
8	4th Jan 2018	Dr. Geethalaxmi	Vice Chancellor Tamil Nadu MGR Medical University.	Speed Post (Indian Postal Service)	On Tracking Package Received By (XX). No other response.
9	3rd Jan 2018	12 Medical Professionals	Endocrinologists, Deans of Medical Colleges in Tamil Nadu, India.	Speed Post (Indian Postal Service)	Confirmed Packages Received on Tracking. No other response.
10	9th Feb 2018	Dr, Mansula Manogaran	Gana Kiritharan's personal Nephrologist.	Personally Hand over	Personally hand over on a clinic visit, On next visit did not give any positive opinion.
11	10th Feb 2018	Dr. M. Selvanathan	Gana Kiritharan's personal Family Doctor	Drop at reception of his clinic	Drop at reception of his clinic, Next visit only said good presentation.
12	12th March 2018	Dr. Fred Hui	Gana Kiritharan's Personal Doctor looking after his Toxic Metal Problem.	Showed the article on a clinic visit.	Refused to completely agree with Gana Kiritharan's explanation of T2DM.
13	27th March 2018	Ms. Linda Palmer	Association Manager Cana Soc of Endo & Meta Ottawa Canada	Registered Post	No Response
14	27th March 2018	Ms. Lori Clawges	Dir Publi and Commu Ame Ass Cli Endo Florida US	Xpresspost	No Response
15	27th March 2018	Press	Society of Endo Bristol UK	Registered Post	No Response
16	14th May 2018	South Africa Medical Society	South Africa	Registered Post	Letter Sent to Minister of Health got returned For Others No response.

Tracking Details Available; If Necessary will be provided.

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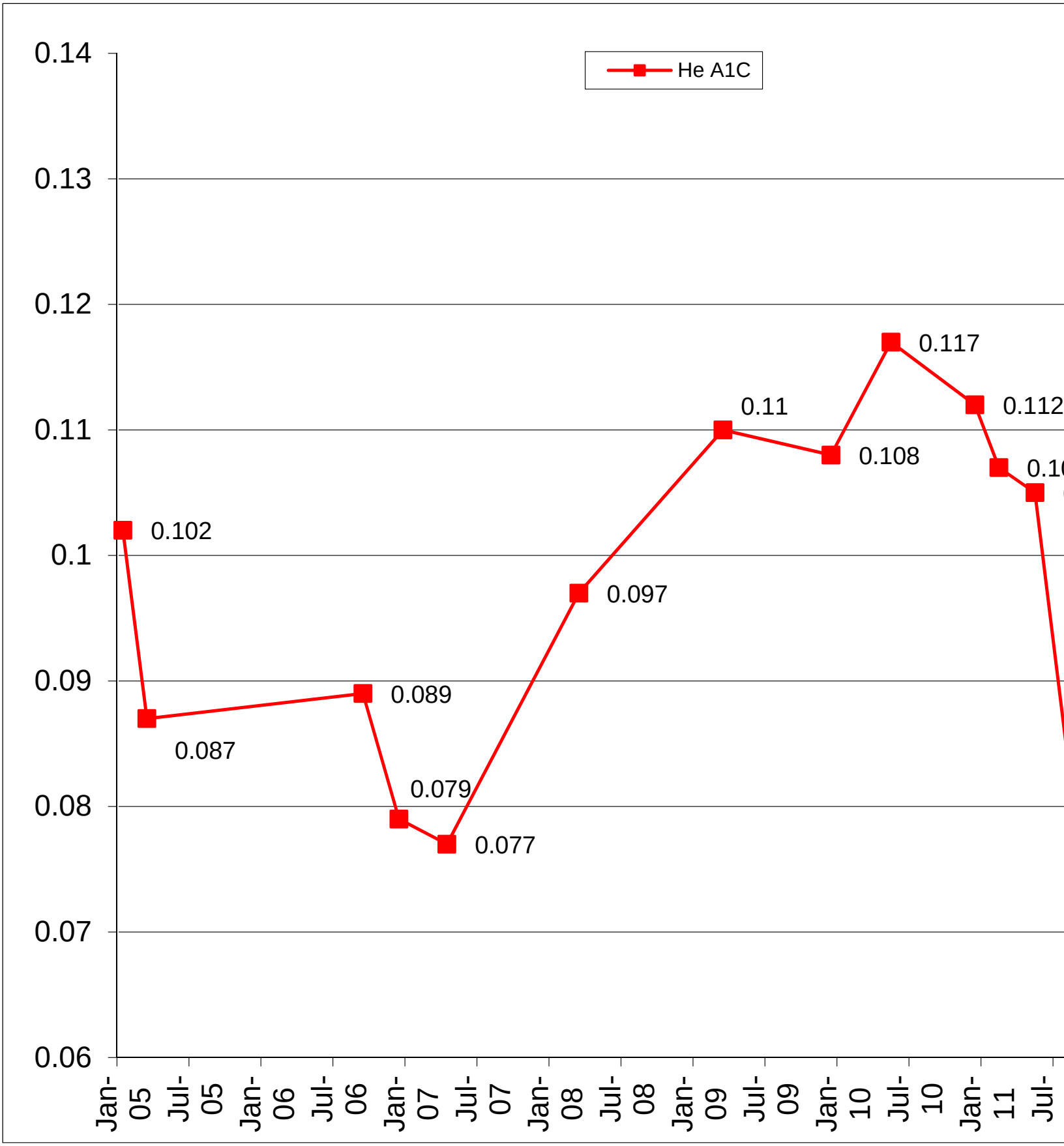

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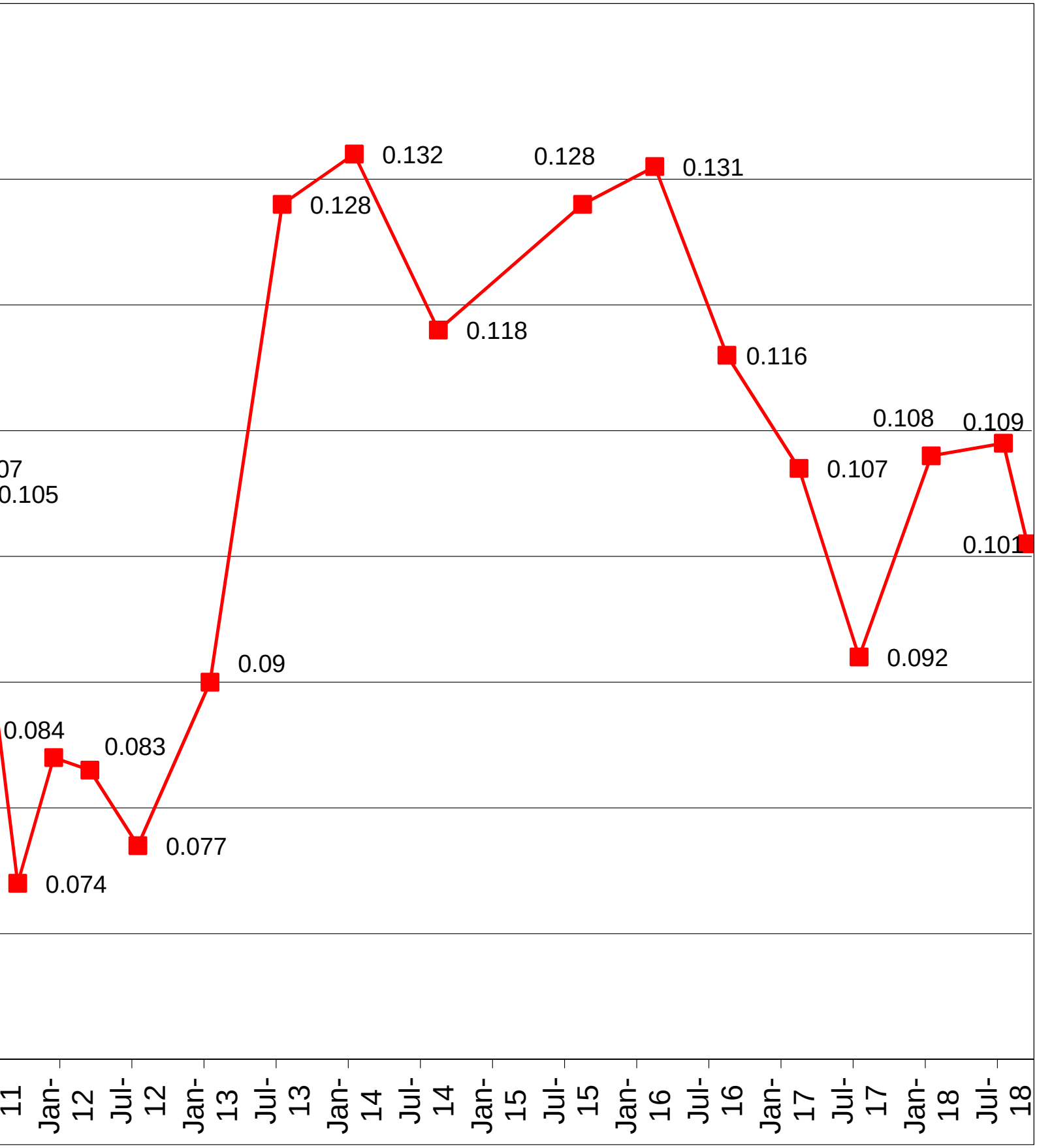

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Type of Report =>				Hair Mineral Analysis
Report Number =>	**H 1**	**H 2**	**H3**	**H4**
Date of Test =>	July 2010	July 2011	July 2012	July 2013
Time Interval =>	Initial	Af 1 Year	Af 2 year	Af 3 Year
Provoking Agent =>				
Scale => / **Toxic Metals**				µg
Total Toxic Metals	10.181	7.681	7.715	8.337
%	100.00	75.44	75.78	81.89
Arse, Bari, Cadmi,Merc	3.302	1.238	1.419	2.058
Thall, Tita, Uran %	100.00	37.49	42.97	62.33
Reat of the Metals	6.879	6.443	6.296	6.279
%	100.00	93.66	91.52	91.28
Total of Barium, Thallium,	1.419	0.678	0.909	0.842
Uranium %	100.00	47.78	64.06	59.34
Hemoglobin A1C	11.2	10.5	7.7	12.8

Arsenic	0.023	0.014	<.01	<.01
Barium	1.4	0.66	0.88	0.82
Cadmium	0.24	0.086	0.11	0.066
Mercury	1.1	0.06	0.08	0.82
Thallium	0.001	0.001	0.001	0.002
Titanium	0.52	0.4	0.32	0.33
Uranium	0.018	0.017	0.028	0.02

Aluminum	4.3	4.7	4.2	4.5
Antimony	0.057	0.017	0.019	0.012
Beryllium	<.01	<.01	<.01	<.01
Bismuth	0.042	0.005	0.007	0.016
Lead	1.8	1.4	1.7	1.6
Nickel	0.18	0.11	0.18	0.08
Platinum	<.003	<.003	<.003	<.003
Silver	0.33	0.02	0.01	0.02
Thorium	<.001	0.001	<.001	0.001
Tin	0.17	0.19	0.18	0.05
Hemoglobin A1C	0.112	0.105	0.077	0.128

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H5	H6	H7	H8	H9
July 2014	July 2015	July 2016	July 2017	July 2018
Af 4 Year	Af 5 Years	Af 6 Years	Af 7 Years	Af 8 Years
g/g				
5.582 54.83	4.826 47.40	4.244 41.69	7.179 70.51	4.057 39.85
1.298 39.31	1.582 47.91	1.587 48.06	3.055 92.52	1.586 48.03
4.284 62.28	3.244 47.16	2.657 38.62	4.124 59.95	2.471 35.92
0.525 37.00	0.557 39.25	0.998 70.33	1.603 112.97	0.676 47.64
11.8	12.8	11.6	9.2	10.9
0.023	0.026	<.01	0.022	0.02
0.48	0.53	0.97	1.6	0.65
0.39	0.089	0.039	0.24	0.12
0.03	0.45	0.21	0.9	0.44
0.001	0.003	0.003	0.002	0.001
0.33	0.46	0.34	0.29	0.33
0.044	0.024	0.025	0.001	0.025
3	2.2	2	3	2
0.024	0.014	0.017	0.013	0.01
<.01	<.01	<.01	<.01	0.01
<.002	0.01	<.002	<.002	<.002
1.1	0.85	0.42	0.9	0.26
0.08	0.11	0.18	0.11	0.09
<.003	<.003	<.003	<.003	<.003
0.02	0.01	<.006	0.01	0.01
<.001	<.001	<.001	0.001	0.001
0.06	0.05	0.04	0.09	0.09
0.118	0.128	0.116	0.092	10.9

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Type of Report =>				
Report Number =>	**U 1**	**U 2**	**U 4**	**U8**
Date of Test =>	27-Sep-10	16-Jun-11	14-Jul-11	17-Jul-12
Time Interval =>	Initial	After 10 Months	After 11 Months	After 2 Years
Provoking Agent =>	DMPS & CaEDTA	DMPS & CaEDTA	ALA & DMSA	ALA & DMSA
Scale =>				
Toxic Metals				
Total Toxic Metals	406.97	106.6	62.7	44.4
%	100.00	26.19	15.41	10.91
Arse, Bari, Cadmi,Merc	65.5	37.1	9.3	19.6
Thall, Uran %	100.00	56.64	14.20	29.92
Reat of the Metals	341.47	69.5	53.4	24.8
%	100.00	20.35	15.64	7.26
Arsenic	33	18	< dl	7.8
Barium	7.6	14	7.5	11
Cadmium	1.4	0.8	0.7	0.5
Mercury	23	3.4	0.8	< dl
Thallium	0.3	0.9	0.3	0.3
Uranium	0.2	< dl	< dl	< dl
Aluminum	260	16	31	5.2
Antimony	0.2	0.6	0.2	< dl
Beryllium	< dl	< dl	< dl	< dl
Bismuth	0.1	3.5	< dl	< dl
Cesium	6.5	5.4	5.5	4.2
Gadolinium	0.4	< dl	< dl	< dl
Lead	28	15	6.1	7.1
Nickel	25	19	9.8	7.7
Palladium	< dl	< dl	< dl	< dl
Platinum	0.07	< dl	< dl	< dl
Tellurium	< dl	< dl	< dl	< dl
Thorium	< dl	< dl	< dl	< dl
Tin	21	10	0.8	0.4
Tungsten	0.2	< dl	< dl	0.2
Hemoglobin A1C	0.112	0.105	0.105	0.077

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Challenged Urine Tests					
U9	**U10**	**U11**	**U12**	**U13**	**U14**
3-Jul-13	16-Jul-14	19-Sep-16	24-Jul-17	12-Feb-18	4-Sep-18
After 3 Years	After 4 Years	After 6 Years 2 Months	After 7 Years.	After 7 Years and 6 months.	After 8 Years and 2 months.
ALA & DMSA	ALA & DMSA	ALA & DMSA	ALA & DMSA	DMPS & CaEDTA	DMPS & CaEDTA
µg/g Creatinine					
92.4 22.70	57.8 14.20	135.8 33.37	149.8 36.81	101.59 24.96	89.3 21.94
51.7 78.93	25.4 38.78	51 77.86	127.7 194.96	45.5 69.47	41 62.60
40.7 11.92	32.4 9.49	84.8 24.83	22.1 6.47	56.09 16.43	48.3 14.14
27	14	39	110	30	29
15	8.8	11	16	9.5	6.4
0.8	0.4	0.6	< dl	0.3	0.5
8.6	1.8	< dl	1.4	5.4	4.7
0.3	0.4	0.4	0.3	0.3	0.4
< dl	< dl	< dl	< dl	< dl	< dl
12	3.3	56	< dl	11	8.7
< dl	0.5	< dl	< dl	1.3	0.4
< dl	< dl	< dl	< dl	< dl	< dl
< dl	0.2	0.3	< dl	0.6	0.6
7.2	4.8	5	6.7	4.4	5.2
	< dl	< dl	< dl	< dl	< dl
11	16	4.1	1.8	10	12
10	6.8	19	13	24	16
< dl	< dl	< dl	< dl	< dl	< dl
< dl	< dl	< dl	< dl	< dl	< dl
< dl	< dl	< dl	< dl	< dl	< dl
< dl	< dl	< dl	< dl	< dl	< dl
0.3	0.6	0.3	0.3	4.7	5.2
0.2	0.2	0.1	0.3	0.09	0.2
0.128	0.118	0.116	0.092	0.108	0.101

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DATE	FASTING GLUCOSE	Hemoglobin A1C	TRIGLYCERIDE	CHOLESTEROL	HDL CHOLESTEROL	HEMOGLOBIN
19-Jun-2002	6.2		7.08	6.09	0.99	149
11-Sep-2002	6.5		11.4	7.69	1.26	
22-Oct-2004	8.16		6.63	4.87	1.04	159
2-Mar-2005	7.4	0.087	2.25	5.07	1.19	149
30-Mar-2006	5.11		5.2	1.93	1.4	
23-Sep-2006	11.5	0.089	3.73	4.38	0.96	152
28-Dec-2006	8.2	0.079	4.79	4.56	1.13	
17-Apr-2007	7.9	0.077	2.27	3.84	1.22	
7-Mar-2008	9.2	0.097	3.14	5.84	0.98	133
28-Mar-2009	11.9	0.11	3.16	5.3	1.19	135
12-Dec-2009	11.8	0.108	4.18	6.39	1.08	133
26-May-2010	14.7	0.117	3.25	4.92	1.17	144
6-Dec-2010	14.6	0.112	4.63	5.79	1.3	153
8-Feb-2011	11.8	0.107	5.48	5.42	1.27	150
9-May-2011	12	0.105	2.81	4.77	1.2	
7-Sep-2011	8.8	0.074				
17-Dec-2011	12.4	0.084	3.74	6.46	1.36	145
17-Mar-2012	8.4	0.083	3.5	5.3	1.31	143
13-Jul-2012	7.8	0.077				
4-Jan-2013	12.5	0.09	4.59	6.58	1.17	
19-Jul-2013		0.128	7.72	5.74	1.12	158
10-Jan-2014	14.6	0.132	8.49	6.4	1.1	150
22-Aug-2014	18.7	0.118	8.43	6.6	1.31	162
21-Aug-2015	19	0.128	5.38	5	1.21	161
19-Feb-2016	17.6	0.131				
22-Aug-2016	17.3	0.116	3.62	4.04	1.19	156
2-Feb-2017	16.4	0.107				144
31-Jul-2017	12	0.092	2.86	4.78	1.47	141
23-Jan-2018		10.8				
10-Sep-2018	12.7	10.1	3.53	4.45	1.3	142

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AST	ALT	Alk Phos	CK	CREATININE	eGFR	MICROALBUMIN (RUR)	CREATININE (RUR)	MicroAlb / Crea
				77				
30	42		487					
28	34		330	95		19.2		
					99			
28	31			70	116	39.6		
24	25			92	83	31.9	12.1	2.6
				81	97	46.3	21.7	2.1
	27					15.6	14.2	1.1
	30					22	15.6	1.4
	37	40	222	72	103	80.3	38.4	2.1
	36			55	>120	250.7	15.3	15.3
25	29		274	64	>90	10	>2	Unab to Cal
25	32	27		75	98			
	26							
	20		215	80	91	48.6	17.4	2.8
	26		194	82	88	40.7	16.7	2..4
		45	160	67	111			
	19		137	55	>120	79.2	21.9	3.6
	29		120	65	114	96.2	15.3	6.3
	28		130	67	109	80	13.4	6
	26			59	114	114.6	12.5	9.2
	31			71	106	74.2	7.7	9.6
		47	81	55	116			
			135	57	115	46	8.5	5.4
				119	110	34	15.5	2.2
		47	238	59	111	94	15	6.3

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ILLEGAL INTERFERENCES

Gana Kiritharan may be a victim of complex Criminal Conspiracy.
Details of incidents related to this intellectual works as follows.

- Gana Kiritharan may get poisoned with Toxic Metals (slow poison) for a long period of time (Problem may started as early as 1993 while he was in Sri Lanka). Gana Kiritharan's attempt to protect him from these poisoning attempts only leads to his discovery of new explanation of T2DM.

- During 2014 – 2015 period there was an attempt to obtain Gana Kiritharan's intellectual works about Diabetics and other illegally in Toronto.

- During 2017 Gana Kiritharan found it difficult to book a lecture hall in India or Singapore to deliver his discovery in a lecture format. An Email communication with a Singapore University Administrator to book a lecture hall in Singapore was interrupted after 3rd Email.

- When Gana Kiritharan visited India some activity of business people in city of Bangalore caused Gana Kiritharan unnecessary additional expenses.

Issues not related this intellectual work.

- During 2007 Gana Kiritharan had filed a complaint to Ontario Civil and Criminal judicial system about some illegal interference into his financial life. But Gana Kiritharan failed to receive Justice.

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