GANA KIRITHARAN

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

BY:
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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 DIABETES MELLITUS [T2DM] AND RELATED DISORDERS OF HEALTH.

ABSTRACT

The current explanation of T2DM outlines that increased consumption of high energy food, increased adaptation of a sedentary lifestyle, and urbanization as the cause T2DM is fundamentally wrong. What actually happens is, insulin resistance due to various different reasons causes glucose deficiency inside cells. This deficiency causes body tissue to go into a chronic fasting stage. However, as blood glucose and insulin levels remain high, lipolysis occurs abnormally. As a result, muscle cells begin to breakdown proteins for energy. Elevated blood glucose levels helps tissue get a better supply of glucose by simple diffusion. If insulin is given to bring down blood glucose levels in T2DM, it will force muscles to breakdown more protein in order to maintain high blood glucose level and could also lead to several toxic symptoms related to hyperinsulinemia. Treatment of T2DM should be aimed at identifying and treating the root cause causing a resistance to insulin alongside small frequent meals throughout the day which are low in carbohydrates and have a high protein content.

1. INTRODUCTION

Whilst the medical profession has successfully overcame challenges such as infectious diseases and surgical techniques, new challenges are coming to the front. The three most important challenges for the medical profession currently are:

i) Viral diseases (AIDS, etc.)
ii) Cancer
iii) Metabolic diseases (Diabetes, etc.)

Despite diabetes' comparatively low mortality rate to cancer and viral diseases, it still can cause serious effects not only to human health, but also in an economic capacity. It is estimated that 415 million people has diabetes worldwide, equal to 8.3 % of the world's adult population and it is predicted the incidence of diabetes will continue to rise. Why is this the situation? Is there a mistake in our understanding the disease?

I was diagnosed with Type 2 Diabetes Mellitus (T2DM) in 2005 (when I was 37 years old) and started treatment for it. From the beginning, I was confused on the course of my disease at times. My blood sugar levels unexpectedly went up or down on several occasions. The most important discovery came in 2010 (as a 42 years old). In that year I had realized that I had a chronic type of toxic metal toxicity, possibly due to criminal

intention and started treatment for it. Based on several observations that I have made during the treatment of my chronic toxic metal toxicity and T2DM problem, my parents experience of their chronic diseases and based on research that I have carried regarding the diseases, I believe that the present explanation T2DM is fundamentally wrong and need to be redefined.

2. PRESENT EXPLANATION OF DIABETES MELLITUS AND T2DM.

Currently diabetes mellitus is defined as a group of metabolic diseases characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both. Diabetes mellitus can then be further divided into two major groups; type 1 and type 2. There are also less common types of diabetic disease. This article mainly talks about type 2 diabetes mellitus (T2DM) which make up 95% of diagnosed diabetic patients. T2DM remains asymptomatic for several years as a result in some cases it remains undetected for several years, overall it remains undetected in nearly 50% of people thought to have T2DM. Currently, the medical profession has conducted several studies focusing on the molecular mechanism underlying T2DM without much success.

Present medical knowledge blames increased consumption of high energy foods, increased adaptation of a sedentary lifestyle, and urbanization as the cause of increased incidence of T2DM. I believe that these conclusions are fundamentally wrong due to my experience of my personal health problems and my parents' health problems which leads to a different explanation.

3. GANA KIRITHARAN'S EXPLANATION OF T2DM.

In a T2DM patient, the first event to occur is the development of a resistance to insulin. Although I do not completely disagree with the argument that high energy food, a sedentary lifestyle, and urbanization causes obesity which leads to a resistance to insulin, I do not believe that this is the main cause of a resistance to insulin. I believe that the main cause of a resistance to insulin is probably due to toxins, chronic form of viral, bacterial and/or fungal infections of internal organs. The way in which these factors cause a resistance to insulin is another subject. My previous article "Chronic Toxic Metal Toxicity and other Chronic Medical Problems" touches on heavy metals toxicity causes a resistance to insulin.

After a resistance to insulin has been developed, glucose deficiency inside cells occurs. This glucose deficiency pushes cells to the metabolic stage of chronic starvation. However, starvation due to insulin resistance differs from starvation due to a food. In normal starvation blood levels of glucose and insulin decrease and goes on to cause lipolysis of adipose tissue which will lead to an energy supply due to it. In insulin resistance, as blood level glucose and insulin do not decrease, so that lipolysis does not occur. As there is not enough glucose in muscle cells, this leads to the breakdown of protein in muscle cells. The catabolism of muscle cells not only supplies glucose for muscle cells, but also for nerve cells, red cells, and kidney tissue as well. While insulin

resistance hinders getting enough of a supply of glucose for energy requirements, elevated blood sugar levels help improve the supply of glucose in tissues via the simple diffusion of glucose across the cell membrane. The level of blood glucose that supplies enough glucose for cell energy requirements, will be the blood glucose levels. When insulin resistance is severe, the blood glucose level will increase; when insulin resistance is mild, the blood glucose level will decrease. My above explanation of T2DM needs to be expanded in more detail, I will fulfill this responsibility in coming weeks. For now allow me to explain some associated issues of my explanation of T2DM.

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

Almost all research conducted so far on the topic of T2DM have shown a direct relationship between elevated blood sugar levels and pathological damage to tissue. Why is this? If you agree with my explanation you may accept that the elevated blood sugar level is directly proportional to insulin resistance and glucose deficiency inside cells. So pathological damage to tissue is caused by an increased resistance to insulin and increased glucose deficiency intracellularly, not because of the increased blood glucose levels. Increased blood glucose levels is a 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 levels, then what is the treatment for T2DM? First we should identify the root cause of the resistance to insulin and treat the problem. Secondly we should have small meals throughout the day which are low in carbohydrates and have a high protein content. This will aid glucose supply to tissue and will attempt to decrease the breakdown of protein. A meal that has a large amount of carbohydrate will exhaust the available insulin pumping mechanism and will increase the blood insulin levels, which will lead to pumping of more glucose into fat cells. A meal that has a high fat content will also cause adipose tissue to grow, which won't be used in a T2DM patient. The most important thing may be that T2DM patients should not fast, as if they do their adipose tissue won't be utilized but the protein will be broken down to create energy.

4. WHAT WILL CHANGE? WHAT WILL NOT CHANGE?

Let us look into how my explanation of T2DM will or will not change the present way of managing the disease. I have previously explained that glucose deficiency intracellularly is the root course of T2DM. However, is it possible to measure the glucose deficiency intracellularly? It is not for two reasons. Firstly, preparing and utilizing micro needles to measure glucose deficiency intracellularly is not possible in a normal clinical setup. Secondly, in a T2DM patient glucose deficiency intracellularly may be corrected by the mechanism explained above. So to determine the glucose deficiency intracellularly, one possible way is to measure blood sugar levels. But when interpreting the results we

should worry about glucose deficiency intracellularly, not necessarily the elevated blood sugar levels.

However when it comes to treatment, several things will change. Are we going to give insulin to bring down the blood glucose value? Even though I believe that insulin is contraindicated in T2DM, only a detailed clinical study in the future will determine which kind of insulin would be useful and in what type of situation it should be used in. In general, if you accept my explanation that when insulin is given in order to bring down blood glucose levels, muscle will attempt to breakdown protein to try to keep the blood glucose levels up. This will lead to muscle wasting and high blood pressure-like toxic symptoms. Diabetic ketoacidosis 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 the appropriate medical societies and will conduct several detailed scientific studies and other forms of intellectual discussions which will decide and evolve the proper patient care for T2DM patients.

5. TIME NEEDED FOR CHANGE AND WARNINGS.

William Edward Deming, a man who is considered the father of Quality Management concepts says "A big ship traveling in full speed needs distance and time to turn it around.". Today, T2DM is a ship carrying more than 400 million people and is traveling in the wrong direction. Nobody can expect a change in treatment of T2DM in a short period of time. Everybody may have to wait three or more months for medical professionals to reach a conclusion about my explanation of T2DM.

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

I also want to warn any body or institution or organization who attempt to take my explanation in their hand without my permission or participation and try to develop it 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 concluding, let me talk about the methodology I followed and other scientific details of my explanation. To reach the above conclusion about T2DM, I followed the methodology of giving logical organization of some observations I have made about T2DM. The same methodology may have been followed by Charles Robert Darwin on his theory of evolution.

The following are observations I have made about T2DM which lead me to my conclusion:

i) I experienced a high degree of fluctuation of my fasting glucose levels on a daily basis.

ii) The previous day, I consumed 25 grams of coconut syrup to help reduce my fasting glucose levels of the next day.
iii) Attempting to protect myself from poisoning attempts from toxic metals and proper detox protocols aimed at reducing the fasting glucose levels.
iv) When I tried to take insulin to control my blood glucose levels, it failed to bring any control but I experienced an increase of toxic symptoms which include a substantial increase in my blood pressure as well as increased muscle and joint pain.
v) Several times I found that the longer the duration of fasting, my blood glucose levels started to increase.
vi) On 25th of October 2010, I experienced an increased fasting glucose level than the previous night post prandial blood glucose level.
vii) My father who was a T2DM patient, never took Insulin, did regular exercise, and lived a normal life without any diabetic complications and lived up to 87 years of age.

In addition, following already established scientific theories about glucose metabolism also helped me to lead to my conclusions.

i) Daily blood levels of insulin increase or decrease based on blood glucose levels but blood level of glucagon level stays the same most of the time.
ii) In chronic fasting, muscles breakdown protein and convert it into glucose

6.1 Discovery vs Invention

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

In the same way, my explanation of T2DM is also based on established facts, so that it can be accepted without spending time on scientific experiments or research.

7. CONCLUSION

In conclusion I want to make the following statements about T2DM and my explanation of the disease.

- The present explanation of T2DM is fundamentally wrong.
- T2DM is caused by factors causing barriers for glucose entry into cells.
- In T2DM, the body goes into modified type of chronic fasting, where in which the body breaks down protein and converts it into glucose.
- Present treatment like giving insulin to T2DM patients makes the pathological damage worse.
- I invite medical professionals (WHO, India and Singapore) to work with me to work out detailed treatment protocols based on my explanations.

- In addition to the intellectual property rights claim, I also make a royalty claim of 75% of the money going to be saved as a result of my explanation of T2DM for the next 20 years.
- I request WHO to appoint a panel of experts to verify whether the Tamil Community (or any others) poisoned using toxic metals or any other toxins with criminal intention and to take necessary actions to treat and protect such victims, and to also handover the findings to the International Criminal Judicial system for further action.

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.
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4. Trotora, Gerald J: Anatomy and Physiology : Wiley India Pvt Ltd; Indian Edition: 2016.
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7. Article about William Edward Daming : Wikipedia: 2017

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

- How Metformin Works -

BY:

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

- How Metformin Works. -

One of the first line treatments in the management of diabetes is Metformin (also known as Dimethylbiguanide). Biguanides come from the guanide rich herb "Galega *officinalis*" (also known as goat rue or French lilac) and was originally used as a traditional treatment in Europe. Metformin is now the most prescribed glucose lowering agent world wide.

Current medical knowledge outlines that Metformin act in numerous mechanisms that counter insulin resistance and as a result lower blood glucose levels. The drug also offers some protection against vascular complications alongside its anti hyperglycemic effect. During my private study to understand the relationship between chronic toxic metal toxicity and T2DM, I developed a suspicion that Metformin also works in the same way that other chelation agents work. When I stopped and subsequently restarted Metformin for a CAT scan, it brought down my blood glucose levels in a similar way in comparison to other chelators. I waited for an invitation as well as permission to do a scientific study at a proper medical premises. As acquiring an invitation and permission has been timely, I conducted a limited experiment on myself.

I halted all of my medication, including Metformin for 24 hours [I still took Ramipril, Atorvastatin and Metoprolol during this time]. I then collected urine samples from myself for the next 24 hours. After this, I restarted my Metformin (1000 mg b.d.) and then subsequently collected urine samples from myself for 24 hours. I then sent both urine samples to a 'Doctors Data' laboratory to measure levels of toxic metals in the urine samples. I also conducted a challenged urine test 2 weeks before taking these samples. Results are on following page:

When analyzing the results the following conclusions can be made;

- Chelators like CaEDTA and DMPS both have a superior effect of removing toxic metals from the body.
- Metformin helps remove toxic metals to some extent (Arsenic excretion increased by 20 times.)

This scientific study shows that Metformin may work by removing toxic metals from the body. I would like to invite medical professionals to conduct a similar scientific study to see if any other medications used in chronic medical problems work in a similar way.

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Date	17 Sep 20018	18 Sep 2018	4 Sep 2018
Provocation Agent / Toxic Metal Scale μg/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

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Attachments: Toxic Metal Reports.

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LAB #: U180918-2064-1
PATIENT: Gana Kiritharan
ID: KIRITHARAN-G-00002
SEX: Male
AGE: 51

CLIENT #: 34074
DOCTOR: Wendy Pitblado

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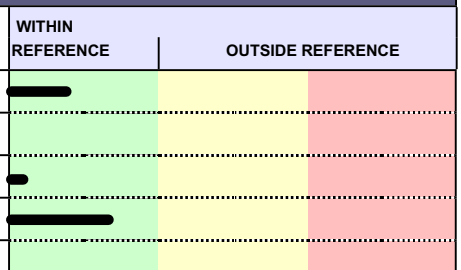
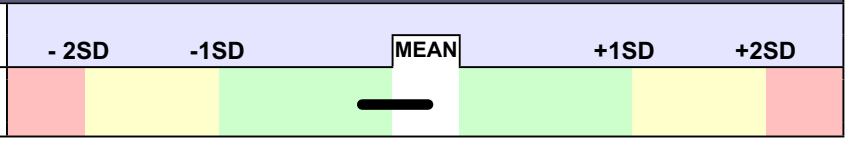
Cambridge, ON N1R 5E2 CANADA

Toxic Metals; Urine 24 hour

TOXIC METALS PER CREATININE		RESULT µg/g creat	REFERENCE INTERVAL	TOXIC METALS PER 24 HOURS RESULT µg/24 HOUR	REFERENCE INTERVAL
Aluminum	(Al)	6.4	< 25	11	< 30
Antimony	(Sb)	< dl	< 0.2	< dl	< 0.3
Arsenic	(As)	6.7	< 75	12	< 150
Barium	(Ba)	3	< 7	5.2	< 8
Beryllium	(Be)	< dl	< 1	< dl	< 1
Bismuth	(Bi)	< dl	< 2	< dl	< 5
Cadmium	(Cd)	0.2	< 0.8	0.4	< 1.5
Cesium	(Cs)	7.2	< 9	13	< 10
Gadolinium	(Gd)	< dl	< 0.5	< dl	< 1
Lead	(Pb)	1.2	< 2	2.1	< 2.5
Mercury	(Hg)	3.1	< 3	5.3	< 5
Nickel	(Ni)	4.4	< 8	7.6	< 13
Palladium	(Pd)	< dl	< 0.3	< dl	< 0.3
Platinum	(Pt)	< dl	< 0.1	< dl	< 0.2
Tellurium	(Te)	< dl	< 0.5	< dl	< 0.5
Thallium	(Tl)	0.3	< 0.5	0.5	< 0.6
Thorium	(Th)	< dl	< 0.03	< dl	< 0.03
Tin	(Sn)	0.2	< 4	0.3	< 6
Tungsten	(W)	0.1	< 0.4	0.2	< 0.5
Uranium	(U)	< dl	< 0.03	< dl	< 0.03



URINE CREATININE	RESULT mg/24 hr	REFERENCE INTERVAL
Creatinine	1740	900- 3000



SPECIMEN DATA

Comments: results checked

Date Collected: 09/17/2018
Date Received: 09/18/2018
Date Completed: 09/21/2018
Method: ICP-MS

pH Upon Receipt: Acceptable
<dl: less than detection limit
Provoking Agent:
Creatinine by Jaffe Method

Collection Period: 24 hr
Volume: 2700 ml
Provocation: PRE PROVOCATIVE

Results are creatinine corrected to account for urine dilution variations. **Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

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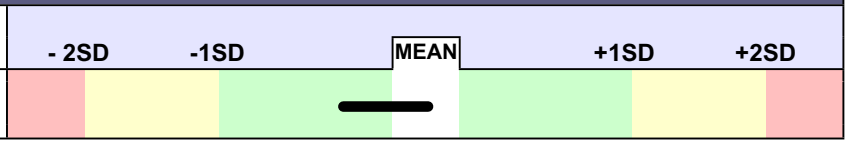
Toxic Metals; Urine 24 hour

TOXIC METALS PER CREATININE		RESULT µg/g creat	REFERENCE INTERVAL	TOXIC METALS PER 24 HOURS RESULT µg/24 HOUR	REFERENCE INTERVAL
Aluminum	(Al)	4.8	< 25	8	< 30
Antimony	(Sb)	< dl	< 0.2	< dl	< 0.3
Arsenic	(As)	140	< 75	230	< 150
Barium	(Ba)	2.7	< 7	4.5	< 8
Beryllium	(Be)	< dl	< 1	< dl	< 1
Bismuth	(Bi)	< dl	< 2	< dl	< 5
Cadmium	(Cd)	0.1	< 0.8	0.2	< 1.5
Cesium	(Cs)	5.9	< 9	9.9	< 10
Gadolinium	(Gd)	< dl	< 0.5	< dl	< 1
Lead	(Pb)	0.7	< 2	1.2	< 2.5
Mercury	(Hg)	0.6	< 3	1	< 5
Nickel	(Ni)	2	< 8	3.3	< 13
Palladium	(Pd)	< dl	< 0.3	< dl	< 0.3
Platinum	(Pt)	< dl	< 0.1	< dl	< 0.2
Tellurium	(Te)	< dl	< 0.5	< dl	< 0.5
Thallium	(Tl)	0.3	< 0.5	0.5	< 0.6
Thorium	(Th)	< dl	< 0.03	< dl	< 0.03
Tin	(Sn)	0.2	< 4	0.3	< 6
Tungsten	(W)	0.08	< 0.4	0.1	< 0.5
Uranium	(U)	< dl	< 0.03	< dl	< 0.03

URINE CREATININE	RESULT mg/24 hr	REFERENCE INTERVAL
Creatinine	1680	900- 3000



SPECIMEN DATA

Comments:

Date Collected: 09/18/2018
Date Received: 09/19/2018
Date Completed: 09/21/2018
Method: ICP-MS

pH Upon Receipt: Acceptable
<dl: less than detection limit
Provoking Agent: METFORMIN 2000MG
Creatinine by Jaffe Method

Collection Period: 24 hr
Volume: 1700 ml
Provocation: POST PROVOCATIVE

Results are creatinine corrected to account for urine dilution variations. **Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements.

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AGE: 50

CLIENT #: 25809
DOCTOR: Fred Hui, MD
The Chelation Center Downtown
421 Bloor St East 202
Toronto, ON M4W 3T1 CANADA

Toxic Metals; Urine

TOXIC METALS		RESULT µg/g creat	REFERENCE INTERVAL	WITHIN REFERENCE / OUTSIDE REFERENCE
Aluminum	(Al)	**8.7**	< 25	
Antimony	(Sb)	**0.4**	< 0.2	
Arsenic	(As)	**29**	< 75	
Barium	(Ba)	**6.4**	< 7	
Beryllium	(Be)	**< dl**	< 1	
Bismuth	(Bi)	**0.6**	< 2	
Cadmium	(Cd)	**0.5**	< 0.8	
Cesium	(Cs)	**5.2**	< 9	
Gadolinium	(Gd)	**< dl**	< 0.5	
Lead	(Pb)	**12**	< 2	
Mercury	(Hg)	**4.7**	< 3	
Nickel	(Ni)	**16**	< 8	
Palladium	(Pd)	**< dl**	< 0.3	
Platinum	(Pt)	**< dl**	< 0.1	
Tellurium	(Te)	**< dl**	< 0.5	
Thallium	(Tl)	**0.4**	< 0.5	
Thorium	(Th)	**< dl**	< 0.03	
Tin	(Sn)	**5.2**	< 4	
Tungsten	(W)	**0.2**	< 0.4	
Uranium	(U)	**< dl**	< 0.03	

URINE CREATININE	RESULT mg/dL	REFERENCE INTERVAL	-2SD -1SD MEAN +1SD +2SD
Creatinine	**54.9**	35- 240	

SPECIMEN DATA

Comments:

Date Collected: 09/04/2018
Date Received: 09/05/2018
Date Completed: 09/06/2018
Method: ICP-MS

pH upon receipt: Acceptable
<dl: less than detection limit
Provoking Agent: DMPS 180MG EDTA 2
Creatinine by Jaffe Method

Collection Period: timed: 2 hours
Volume:
Provocation: POST PROVOCATIVE

Results are creatinine corrected to account for urine dilution variations. **Reference intervals and corresponding graphs are representative of a healthy population under non-provoked conditions.** Chelation (provocation) agents can increase urinary excretion of metals/elements. V13

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

- Preventing Renal Failure in Type 2 Diabetes Mellitus Patients -

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

- Preventing Renal Failure in Type 2 Diabetes Mellitus Patients –

Current medical knowledge states that microvascular damage in T2DM is the cause of renal failure. Even though I do not completely disagree, I believe that renal failure in T2DM is caused by toxic damage due to causatory factors involving insulin resistance and glucose deficiency inside renal tissue. In this article, I will explain how to manage these issues so that renal failure can be prevented in T2DM.

1) Preventing toxic damages due to causatory factors leading to insulin resistance.

An important step in managing T2DM and preventing resulting complications, is identifying the cause of insulin resistance and treating it. Some of the most common causes may be chronic toxic metal toxicity and chronic viral, bacterial and fungal infections of internal organs. If you suspect infection of internal organs, you can do necessary tests in order to identify the responsible microorganisms and treat them with appropriate antibiotics.

Let us talk in details about how to diagnose and treat chronic toxic metal toxicity. Proper treatment of chronic toxic metal toxicity can involve tests such as hair mineral analysis, challenged urine tests, treatment with appropriate chelation agents, and high doses of vitamins, minerals and other naturopathic medications. But this facility is not available for many patients. So, I am outlining the following simplified treatment for chronic toxic metal toxicity with three medications.

a. (Buffered) Vitamin C.

Vitamin C has many positive effects on health. One important function is its antioxidant properties. When Vitamin C buffered with calcium or other minerals, it helps prevent irritation of stomach due to the acidity of Vit C. Buffered Vit C when taken in large amounts helps to prevent pathological damage due to the oxidation effect of toxic metals and other toxins.

b. N-Acetyl Cysteine [NAC]

NAC is the precursor of Glutathione. Glutathione is a natural antioxidant. It not only protects cells from the oxidation damage of toxins, but it also helps excrete toxic metals from body.

c. Chlorella

Chlorella is an algae that has a high nutrient value. It also helps to absorb heavy metals into it. When given in an appropriate dose, it absorbs heavy metals released

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into the gut by the liver and prevent them from being reabsorbed back into the system.

d. How to administer them.

The better order to begin the administration are as follows:

d.1. Start with Buffered Vit C starting at 500 mg once a day, which can then be increased up to 1000 mg three times a day.

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

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

If at any point you begin to experience abdominal discomfort or an allergic reaction – 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.

An important reason as to why renal failure occurs in T2DM patients is that a glucose deficiency inside the renal tissue occurs. The main reason for this is due to the misunderstanding of the disease. Current explanations that outline that elevated blood sugar is the main cause of pathological damage is a mistake. The actual problem is that a glucose deficiency inside the tissue and an elevated blood sugar is a defensive mechanism by the body where in which the body tries to get an adequate supply of glucose. In order to maintain a good supply of glucose to the kidney and other tissues please follow the following advice.

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 an elevated blood pressure and pain in your muscles and joints as your body has started to breakdown protein to keep the blood sugar levels up. The only way to prevent this is to reduce or stop taking insulin.

b. Having food with small a amount of carbohydrate or sugar every 2-3 hours will help to maintain a sustained high blood sugar level so that kidney and other tissues will get an adequate supply of glucose.

By following the above advice, only I, Gana Kiritharan, was able to prevent any serious damage to the kidney. I strongly believe that the above advice will help you to prevent any damage to the kidney and any other tissues.

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

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

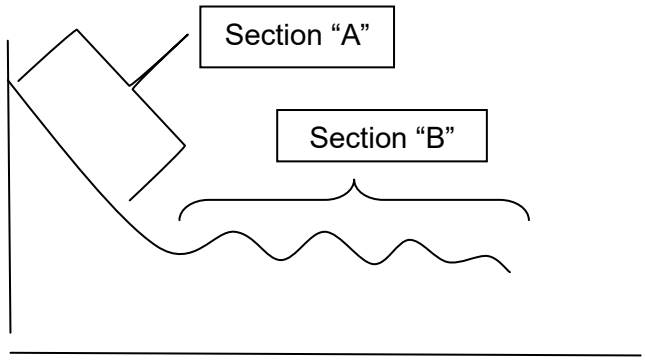
– Continuous Blood Sugar Monitoring –

One of the new facilities available in diabetic management is continuous blood sugar monitoring. The pharmaceutical company, Abbott, in their line of blood sugar monitoring devices called 'Freestyle', have released a new device called 'Freestyle Libre'. Even though it is labelled as a blood sugar monitoring device, it actually monitors the sugar level of interstitial fluid, which may be slightly lower than the blood sugar level.

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

1. **Elevated Blood Sugar Level in the Morning Hours.**
   Elevated blood sugar levels in the morning hours has been observed by medical professionals previously. I also have had similar observations previously and this was mentioned in my article "Gana Kiritharan's explanation of T2DM and related disorder of Human health." Current explanations regarding this phenomena are as follows
   a. High carb bedtime snacks and not enough diabetic medication.
   b. Dawn phenomenon, which states that the body prepares for the next days energy requirement.
   c. 'Somogyi effect' or rebound hyperglycemia. According to this, your blood sugar levels drops so low that this causes the body to release hormones in an attempt to rescue you from dangerously low blood sugar levels.

   According to my observations, a long night fasting blood sugar level will be as follows.



   If you accept my explanation of T2DM, it may be easy to explain the above phenomena. According to my explanation, during the "A" section of the graph, blood sugar levels are high so that cells receive enough of a sugar supply through an insulin pump and additionally via simple diffusion. When, the situation reaches

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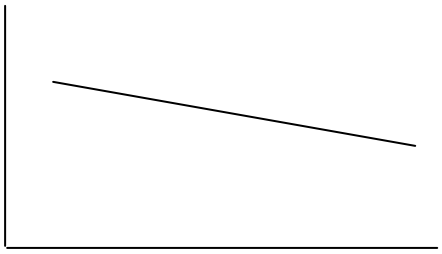
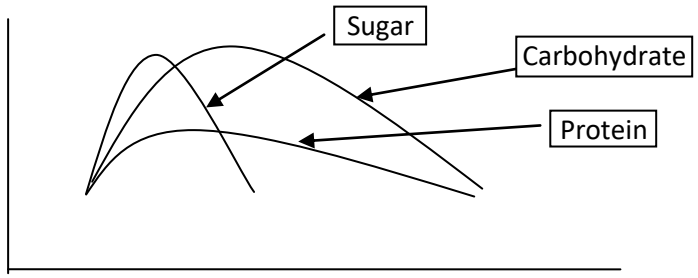
the "B" section of the graph, the sugar supply to tissues from simple diffusion decreases. This leads to stress hormones being released from body tissues. These stress hormones forces the liver to release more sugar and when fasting continues, the body begins to breakdown proteins for energy requirements. But as I have argued previously, lipolysis will happen abnormally as blood sugar and insulin levels do not decrease. This phenomenon will continue up until the next meal. By bringing the next meal closer, you can avoid this phenomenon from happening.

2. **The Effect of Exercise in T2DM Patients' Blood Sugar Level.**
   Even though I was not able to perform heavy exercise during the past few weeks, I was able to do some mild exercise such as moving boxes in a storage room, etc. During exercise, whether it follows a fasting stage or after a meal, blood sugar levels decreases gradually.



3. **Various Types of Meals.**
   During the past few weeks I tried various different types of meals and I observed how they influence my blood sugar graph. My observation as follows:



Whilst foods and drinks containing sugar increases blood sugar levels drastically, they however swiftly return back to normal. A meal with a large amount of carbohydrates increases blood sugar levels drastically however they take a while to come back to normal. A meal with high quantity of protein, increases blood sugar levels up slightly and gradually returns back to normal.

4. **Effects of chelation medication.**
   Another observation of mine was how various chelation medications increase blood sugar levels. I observed that whilst some chelation medications increases blood sugar levels others help decrease it. CaEDTA increases blood sugar level by greatly but Plaquex and DMSA helps to decrease blood sugar levels.

Attachments:
My continuous glucose monitoring graph from 2nd of August 2018.

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Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

	00:00	06:00	12:00	18:00	00:00	Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 1 Aug	21 mmol/L, 10, 8.3, 0								
Thu 2 Aug	21 mmol/L, 10, 8.3, 0 (● New Sensor ○ Sensor Scan)					14.4 mmol/L			
Fri 3 Aug	21 mmol/L, 10, 8.3, 0					16.2 mmol/L			
Sat 4 Aug	21 mmol/L, 10, 8.3, 0					15.8 mmol/L			
Sun 5 Aug	21 mmol/L, 10, 8.3, 0					15.7 mmol/L			
Mon 6 Aug	21 mmol/L, 10, 8.3, 0					16.4 mmol/L			
Tue 7 Aug	mmol/L, 21, 10, 8.3, 0					mmol/L 17.4			

Chelation with CaEDTA and Plaquex

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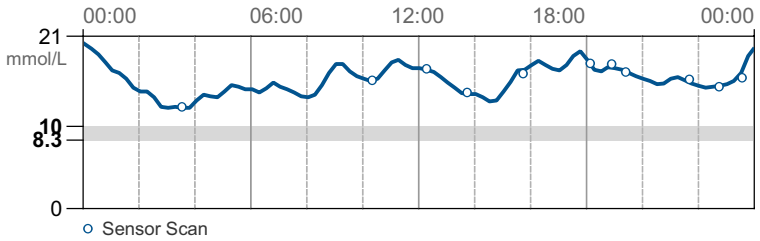
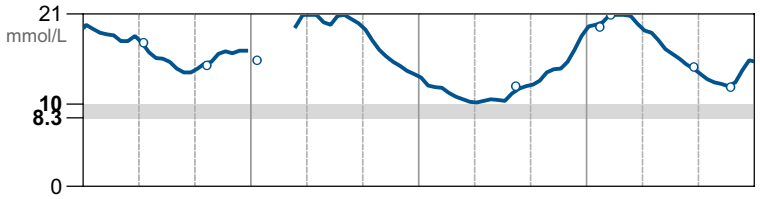
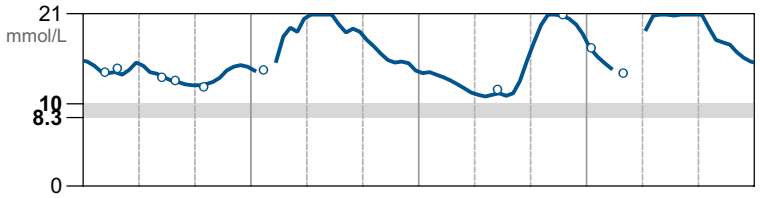
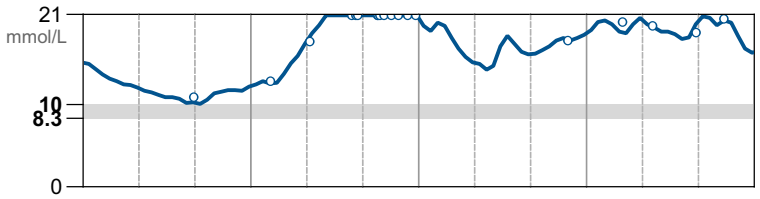
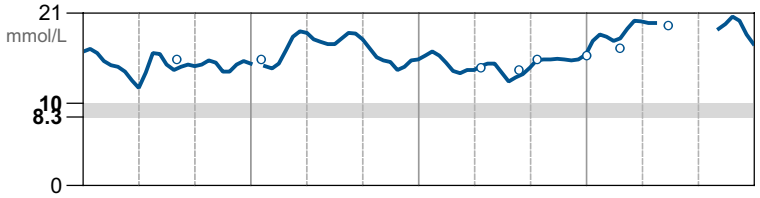
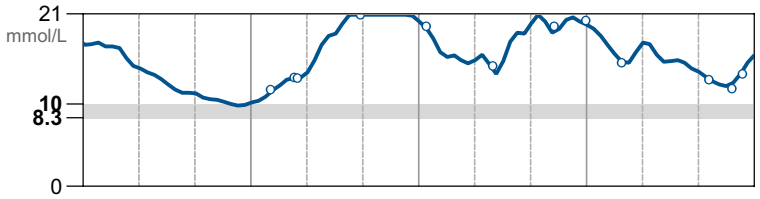
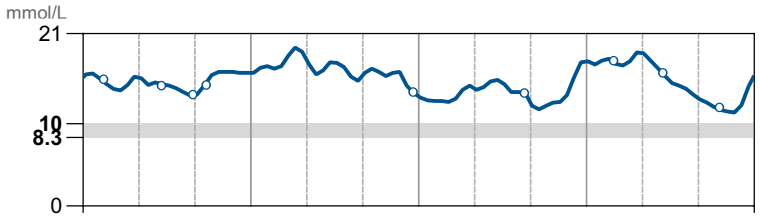
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 8 Aug		15.7 mmol/L			
Thu 9 Aug		16.0 mmol/L			
Fri 10 Aug		16.2 mmol/L			
Sat 11 Aug		17.2 mmol/L	Chelation with CaEDTA and Plaquex		
Sun 12 Aug		16.0 mmol/L			
Mon 13 Aug		16.0 mmol/L			
Tue 14 Aug		15.3 mmol/L			

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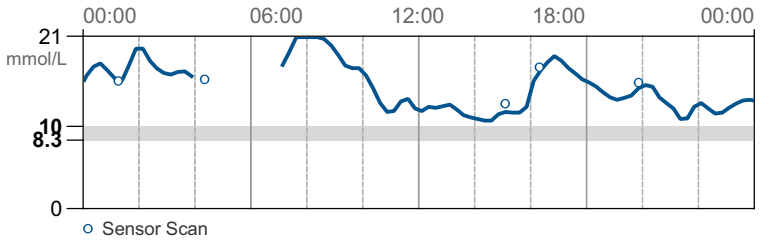
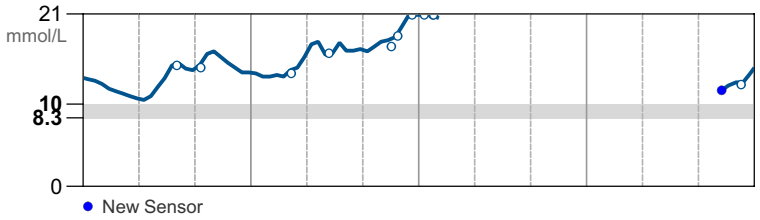
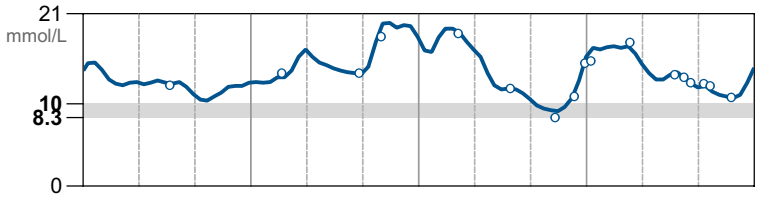
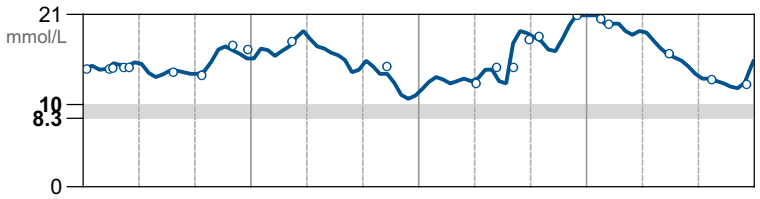
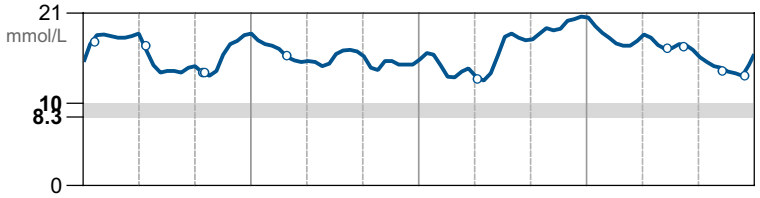
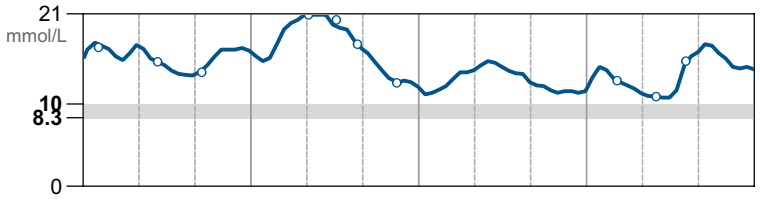
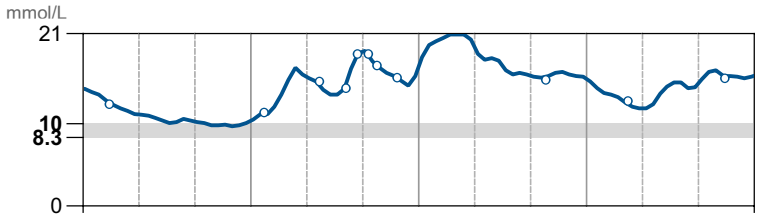
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 15 Aug		14.9 mmol/L			
Thu 16 Aug		15.1 mmol/L	Chelation with Plaquex		
Fri 17 Aug		13.9 mmol/L			
Sat 18 Aug		15.6 mmol/L			
Sun 19 Aug		16.3 mmol/L			
Mon 20 Aug		14.9 mmol/L			
Tue 21 Aug		14.6 mmol/L	Chelation with Plaquex		

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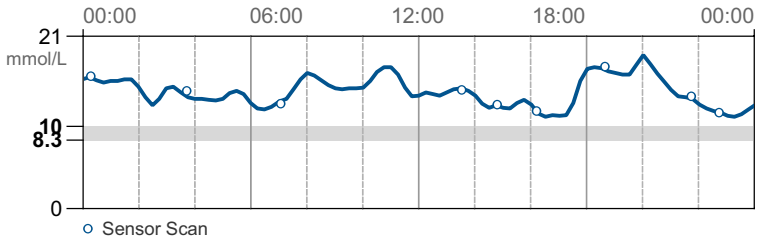
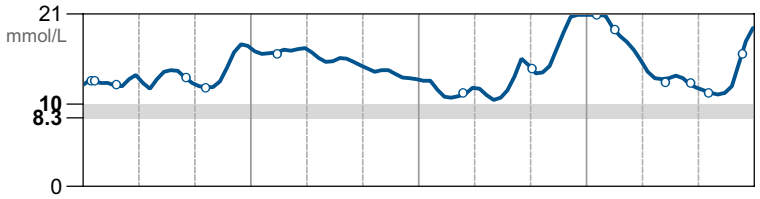
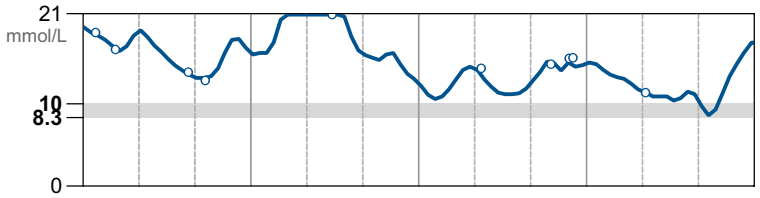
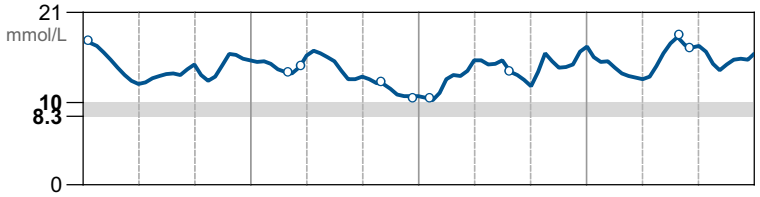
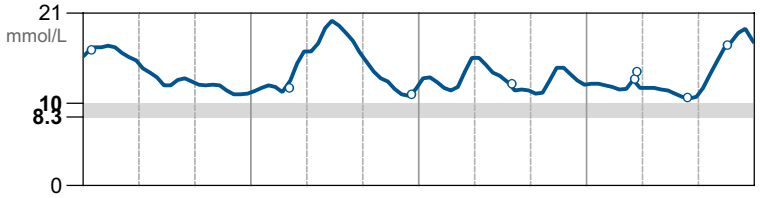
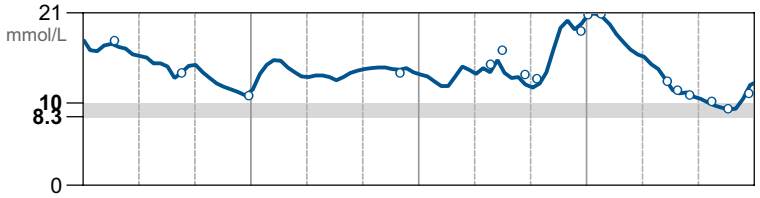
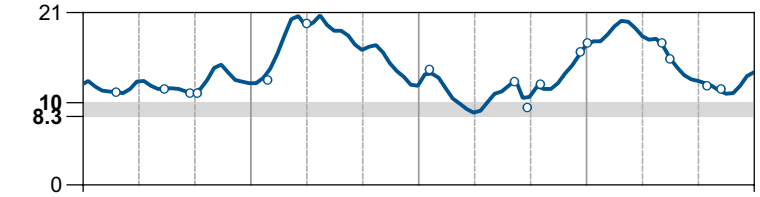
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 22 Aug		14.3 mmol/L			
Thu 23 Aug		14.5 mmol/L			
Fri 24 Aug		15.0 mmol/L			
Sat 25 Aug		14.2 mmol/L			
Sun 26 Aug		13.6 mmol/L			
Mon 27 Aug		14.2 mmol/L			
Tue 28 Aug		14.1			

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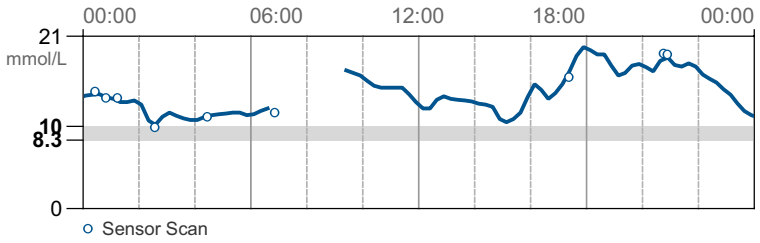
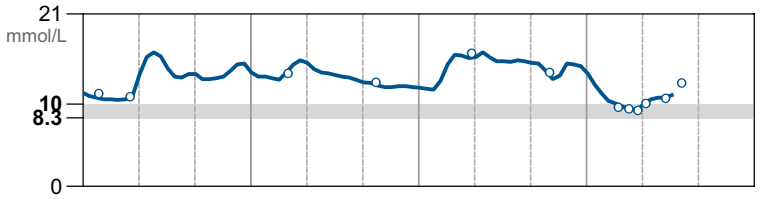
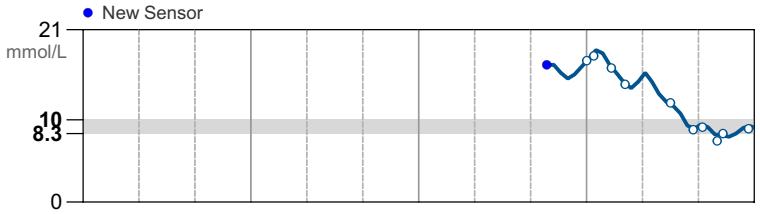
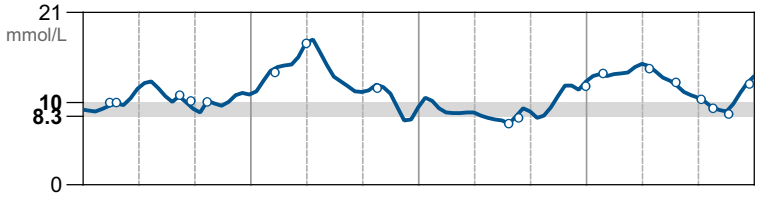
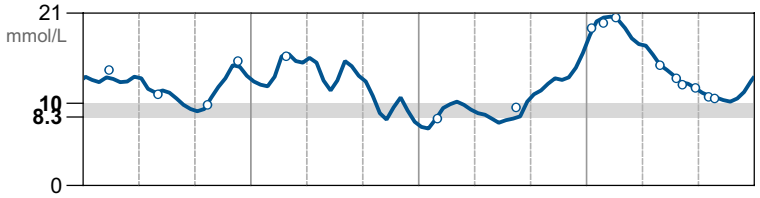
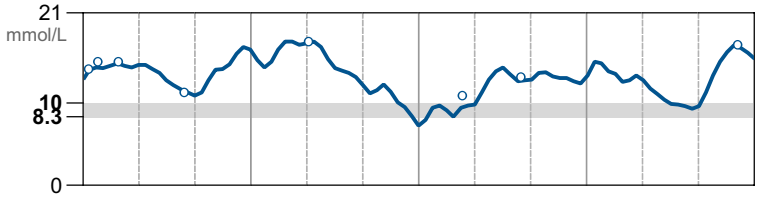
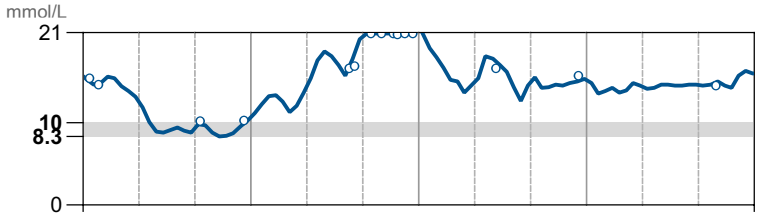
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 29 Aug		14.1 mmol/L			
Thu 30 Aug		13.3 mmol/L			
Fri 31 Aug		13.1 mmol/L			
Sat 1 Sep		11.1 mmol/L			
Sun 2 Sep		12.4 mmol/L			
Mon 3 Sep		13.1 mmol/L			
Tue 4 Sep		14.7 mmol/L			

Challenged Urine Test

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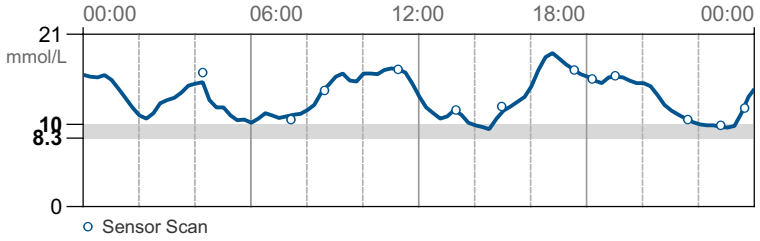
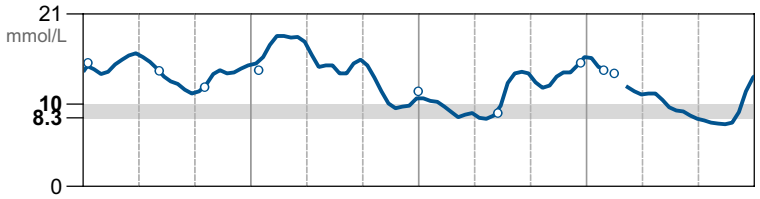
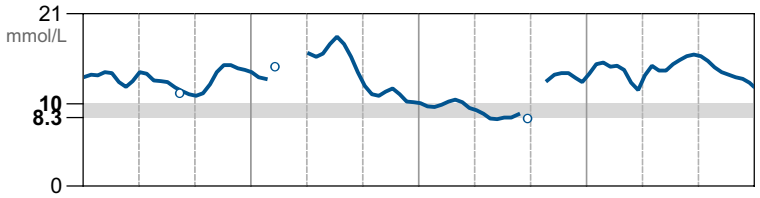
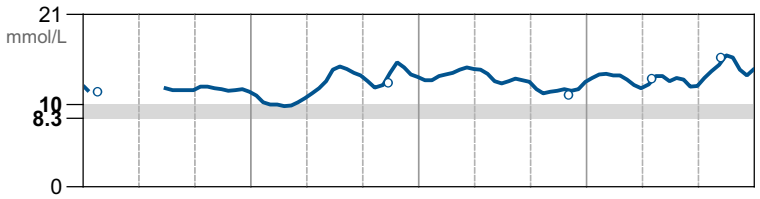
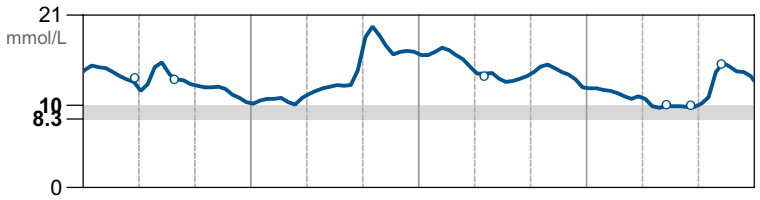
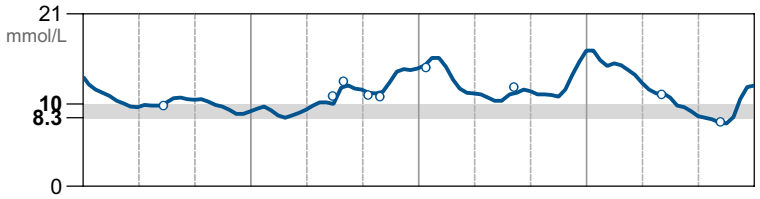
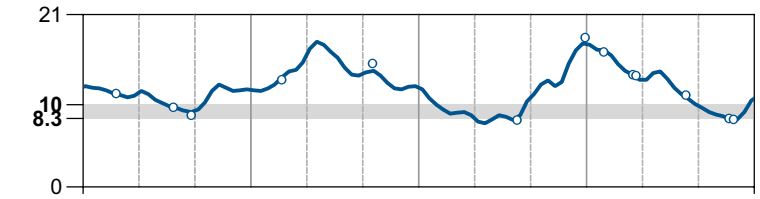
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 5 Sep		13.3 mmol/L			
Thu 6 Sep		12.6 mmol/L			
Fri 7 Sep		12.9 mmol/L			
Sat 8 Sep		12.8 mmol/L			
Sun 9 Sep		13.2 mmol/L			
Mon 10 Sep		11.3 mmol/L			
Tue 11 Sep		12.0 mmol/L			

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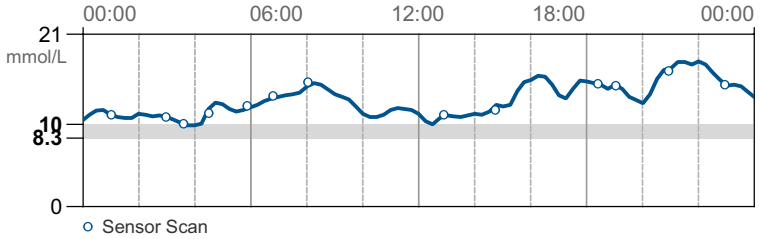
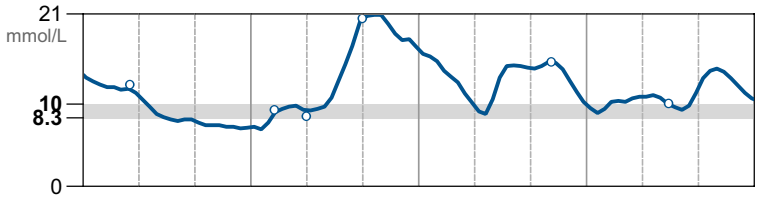
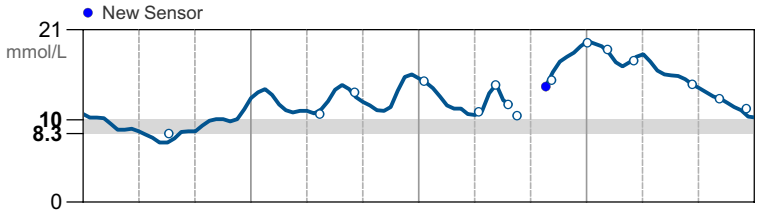
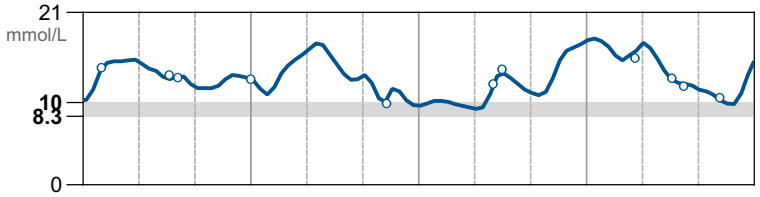
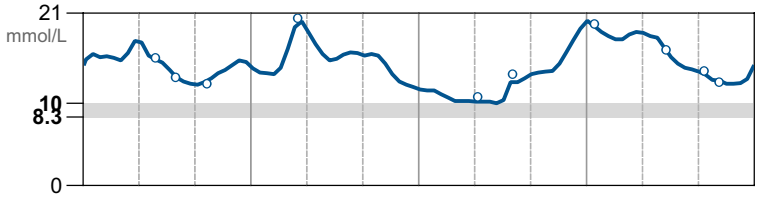
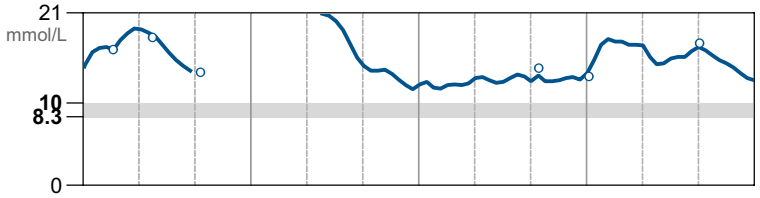
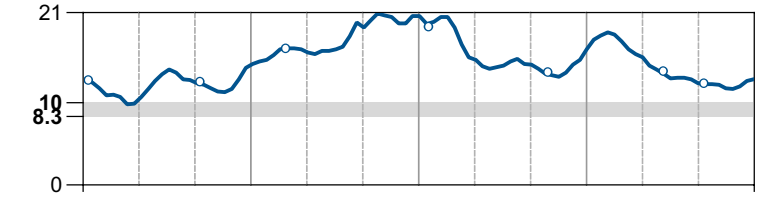
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

FreeStyle Libre

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 12 Sep		13.1 mmol/L			
Thu 13 Sep		11.9 mmol/L			
Fri 14 Sep		12.7 mmol/L			
Sat 15 Sep		13.1 mmol/L			
Sun 16 Sep		14.7 mmol/L	No Metformin		
Mon 17 Sep		15.1 mmol/L	No Metformin		
Tue 18 Sep		15.1			

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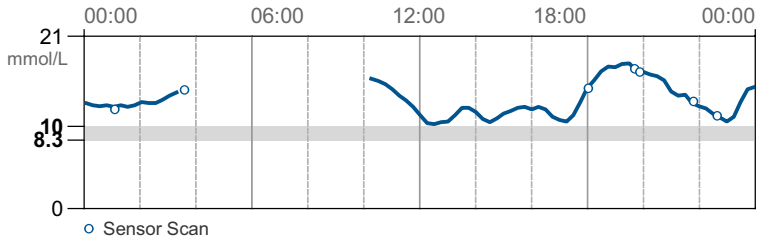
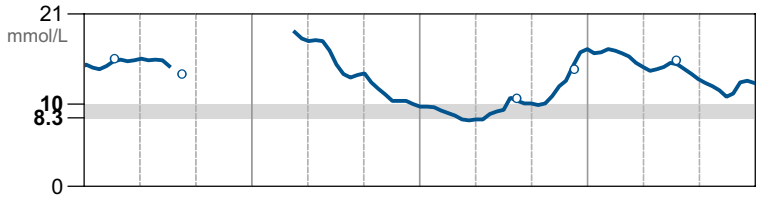
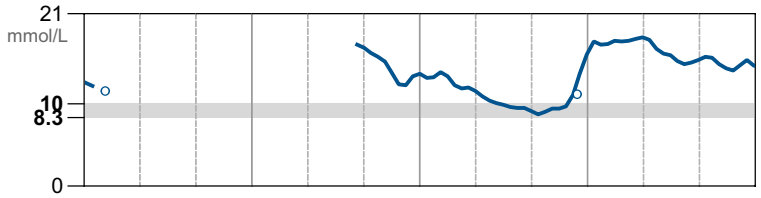
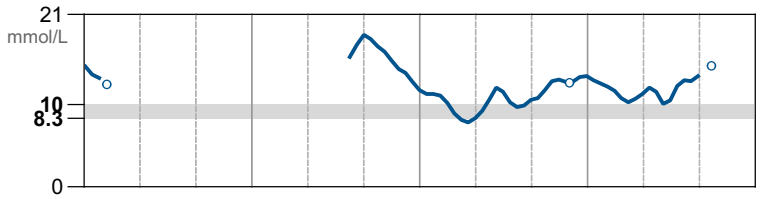
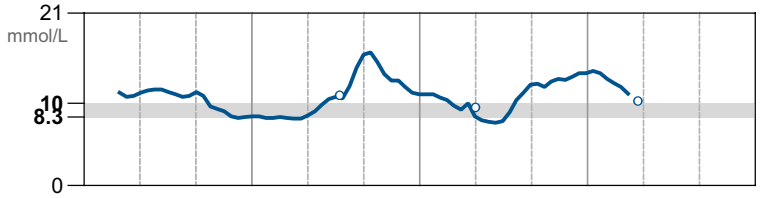
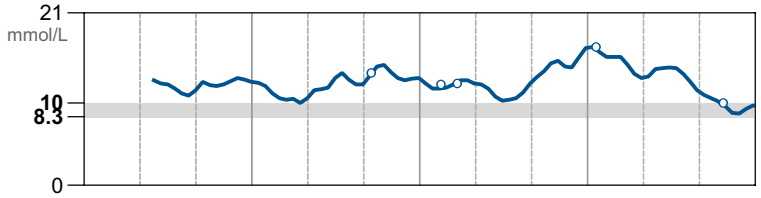
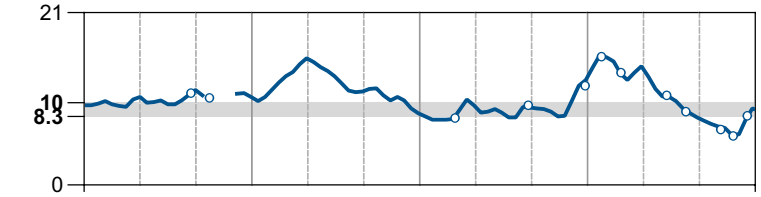
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 19 Sep		13.2 mmol/L			
Thu 20 Sep		13.1 mmol/L			
Fri 21 Sep		13.8 mmol/L			
Sat 22 Sep		12.3 mmol/L			
Sun 23 Sep		10.9 mmol/L			
Mon 24 Sep		12.6 mmol/L			
Tue 25 Sep		10.6			

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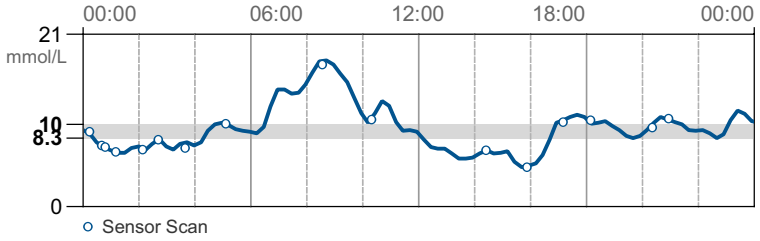
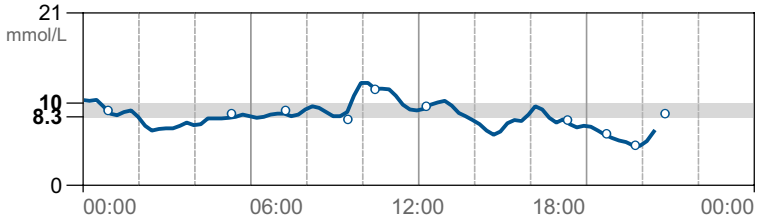
Weekly Summary

1 August 2018 - 27 September 2018 (58 days)

Glucose

		Average Glucose	Carbs	Rapid-Acting Insulin	Long-Acting Insulin
Wed 26 Sep		9.5 mmol/L			
Thu 27 Sep		8.3 mmol/L			

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Chronic Toxic Metal Toxicity and other Chronic Medical Problems.

Prepared by

Gana Kiritharan

ABSTRACT

I, Gana Kiritharan, have been experiencing chronic medical problems from the year 2002 (age of 34), one of which is known as metabolic syndrome. On May 2010, I discovered that I am a victim of a chronic type of toxic metal toxicity (Mercury, Lead, Cadmium, etc.) possibly due to criminal intention. After I begun treatment for my Toxic Metal Toxicity, I started to experience high levels of fluctuation in my fasting blood glucose levels. During the last 12 months, my fasting blood glucose level went above 16 mmol/L twice and below 8 mmol/L twice. Previously conducted research explains that toxic metals such as Cadmium can cause impairment of glucose tolerance in rats. The paper explains that Cadmium can decrease the number of insulin receptors in fat cells drastically, but can also cause moderate hyperinsulinemia. When I attempted to take insulin injections for my diabetes, it not only failed to bring any control of my blood glucose levels, but it also caused complications which could have been attributed to the hyperinsulinemia. When I recalled my mother's medical problems, I realized she may have suffered from a chronic form of toxic metal toxicity for a long period of time. It is possible that she may have been exposed to the toxic metals from fire wood being used in the kitchen. When I searched for more evidence regarding toxic metal toxicity as a cause of some female health problems, I found that toxic metal toxicity may be a contributing factor for menopausal syndrome and several other psychological problems suffered by the female population. Estrogen or some of it byproducts in synthesis may give protection from the toxic metals up until menopause. Through this article I want to call on the medical profession to abandon its current attitude of denial and refusal as well as to come forward and establish a proper preventive, diagnostic, and treatment protocols for this complex medical problem.

Introduction:

I was born in September 1967 and was selected to undertake Medical Studies in 1987 in Jaffna (Sri Lanka) medical school up until 1995. In our medical school, as our part of studies, we carried out several lab experiments on ourselves. Most of the lab experiments conducted on my body (WBC/DC to ESR) brought normal results. As a part of small medical research, I took a Glucose Tolerance Test in 1989 - 1990 (When I was 22-23). Out of 3-4 medical students who also undertook the, mine was the one displayed the greatest intolerance to glucose, however it was far beyond for diagnosis of diabetes mellitus. I was part of an experiment conducted in August 1999 in Ottawa, Canada. On that report the random serum glucose was 5.6 mmol/L with all other indices being within normal range.

The first medical report displayed some chronic medical problems, 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 me some dietary advice 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

The doctor the started Fenofibrate as a medication to treat my high triglyceride and cholesterol levels 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 2005 (Age of 37) my fasting blood glucose level increased and doctors begun treatment for diabetes. Important information about my chronic medical problems came to light on the 18th or 19th of May 2010. On that date, whilst I was cooking some curry at home, I saw a small amount of glittering fluid that was running on the cooking utensil. On the suspicion that the glittering fluid may be toxic metals such as mercury, I carried out appropriate laboratory experiments and found several toxic metals have been accumulating in high amounts in my body.

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 a 3rd cesarean section for my mother. The first child for my mother was a stillbirth and I do not know the year of this first cesarean section. Then my eldest brother was born in October 1964 through a second cesarean section and three years following this I came into this world through a third cesarean section. As my parents already had a baby boy, they were expecting a baby girl. Unfortunately I came out as a boy and this may have lead to some unwanted treatment in my early childhood. Anyhow, I received much better care than any other average child in my community. One example is when my brother was preparing for his grade 5 (10 years old) IQ test; I was 7 years old and was able to solve most of the problems. When my turn came, my parents arranged for me to have the best private tuition available in our village and I scored 157 for 200, that was the highest score in my school.

The following table may give some important medical incidents in my life.

Age	Incident
5 – 10	I may be a very weak person. Once one of my friends described my body as a good model to teach bones in the body. A normal upper respiratory tract infection would take more than two weeks to heal. Once family doctor suggested that I may be suffering from TB. When I started to grow up, I started to eat and any nutritional problems disappeared.
Around 12	I developed quadriceps tendonitis and it healed through rest and appropriate medication.
18 - 19	Around my high school exams, I developed sinusitis and took ampicillin antibiotics throughout my exam period.
21	When I went to medical school, for the first time I took food outside of my home for a long period of time and I was having vegetarian food as well. After three months or so, I developed a urinary tract 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 a war injured casualty, I got trapped in crossfire and received a minor injury in my right upper arm. Minor surgery was required to remove the fragment, which happened the day after the injury. During the treatment for this injury, I developed a malaria infection for the first time and this was managed with one full dose of treatment.
21 – 28	During my time at medical school, I developed upper respiratory tract infections frequently every two months. Also I developed viral warts and intermittently. I also started to experience some weight gain.
29 – 31	When I was about to leave Sri Lanka for India, I contracted a Malaria infection. Whilst in India, I contracted malaria 3 – 4 times. To treat this, I took some broad spectrum anti-malarial drugs and consequently I did not develop a 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 was rheumatic fever. He developed this medical problem when he was 15 years old and was given a monthly Benzathine Penicillin injection and has not suffered any long-term complications.

My dad is a diabetic, this started in his late 30s to early 40s. This was managed by him taking oral hypoglycemic drugs, and he never took insulin injections.

He did regular exercise (he went to work on a bicycle and he managed our two acre plot of land mostly without any additional help). He did not worry about his diet much. He ate an unrestricted diet but he avoided sugar. I can confirm that his abdominal circumference was greater than 40 inches. His triglyceride levels was never checked and I do not recalled him being diagnosed with metabolic syndrome. My mother had many medical problems and I will discuss this in detail later.

Details Medical History:

The following table gives lab experiments conducted during the last 10 years alongside any change in environment and 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 was enough to suspect that I may be have metabolic syndrome. Further detailed tests have helped to correct the original diagnosis. When I started to take insulin, it not only brought no big improvement but it also caused my BP started to increase. This led me to search for an explanation about my medical problem and to help me to find out about the words insulin resistance, hyperinsulinemia and metabolic syndrome. The above details are presented in the following graph for a graphical representation of the fluctuations mentioned previously.

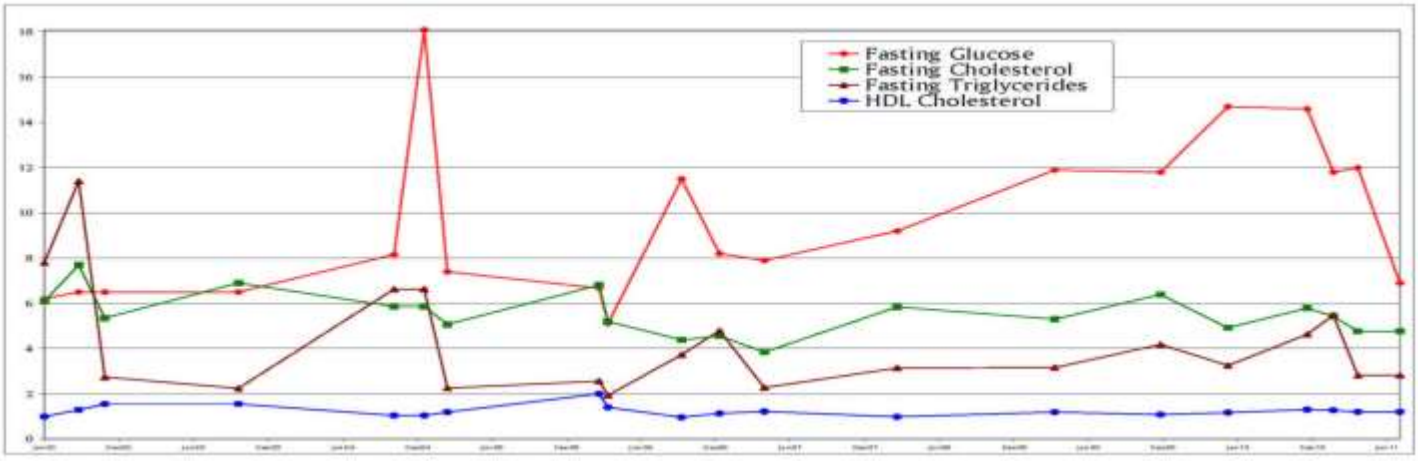

Fig 1: Fluctuation of Gana Kiritharan's Fasting Blood Glucose, Cholesterol/HDL and Triglyceride Value.

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The above graph explains how some values have been fluctuating over the past few years. Even though medication, food, and a change in exercise habits may be responsible for some improvement, the fact of whether or not I was being exposed to toxic metals through the environment may be the important factor.

When I realized that I may be suffering from chronic toxic metal toxicity, I was quite confused. Firstly, the reason for the toxicity may have been due to criminal intentions. Based on previous experiences, I was unable to call the police immediately. Secondly, this was the first time I have heard about mercury poisoning. Even though I have come across several poisoning materials during my medical studies before, I do not remember being taught about mercury poisoning in detail. When I approached a medical professional who was treating me, I experienced several denials and refusals. My family doctor told me that in his 20 year career, he has never seen a patient with mercury poisoning. When the Ontario healthcare system failed to order the proper lab reports, I went on my own and obtained the following reports which might explain the situation.

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

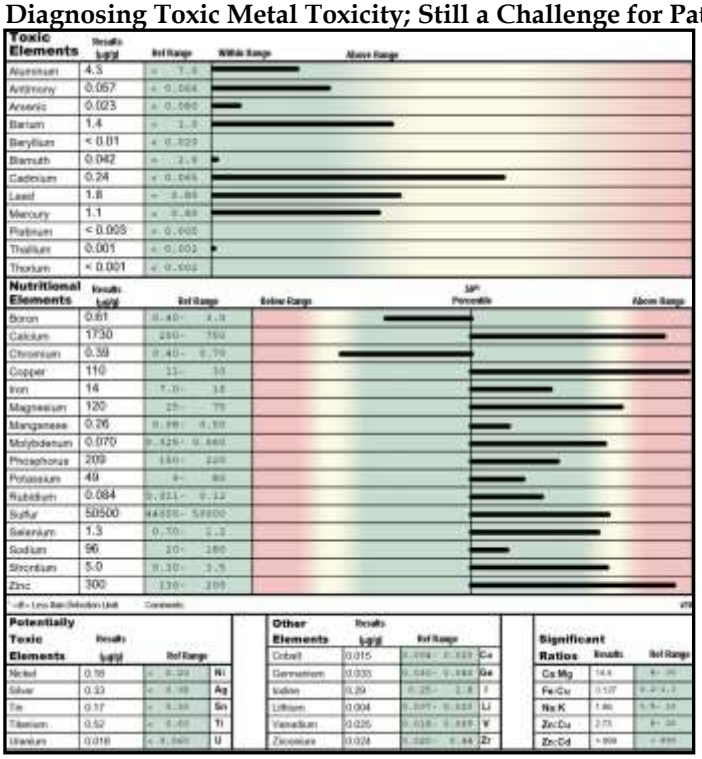


Fig 2.1: Test Conducted in July 2010

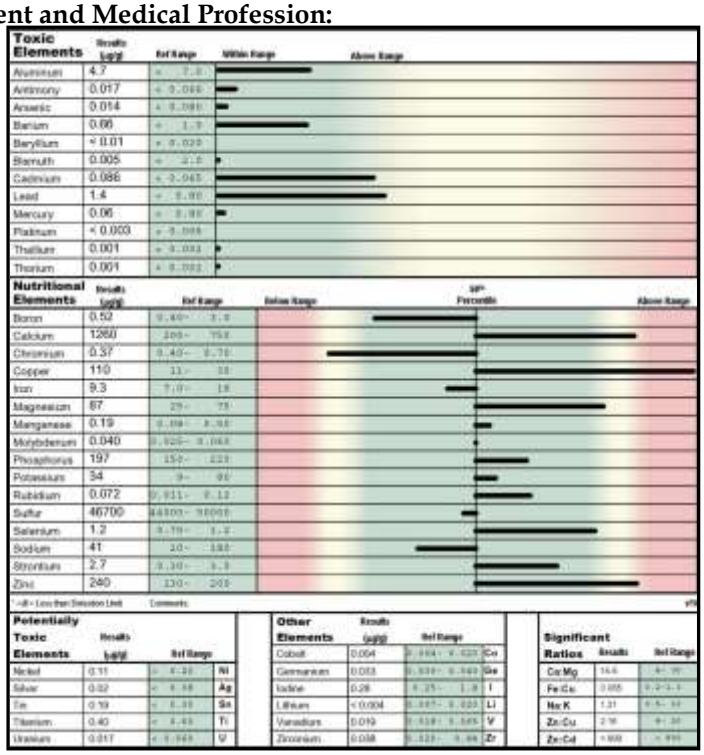


Fig 2.2: Test Conducted in July 2011 (After 1 Year)

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

When a person is to be checked for chronic mercury or other toxic metal toxicity, measuring either blood or random urine metal concentrations has little use. 24 hour urine samples looking at metal concentrations may give some information, however these are rarely performed. Checking fecal concentrations of toxic metals may explain how much is being excreted, again this test is rarely performed. The most useful test is analyzing mineral composition of hair. In a normal adult, hair grows 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 a month. In addition to this, hair may contain 5% or more sulfur in it. Toxic metals in the body usually circulates attached to this sulfur get concentrated in hair. This will happen even the amount circulating is small.

In my hair mineral analysis, toxic metals such as Cadmium, Lead and Mercury can be found at high concentrations in the hair samples. After one year, chelation of some of the toxic metals had caused the levels to decrease. Also my hair mineral analysis confirms some deficiencies in my nutritional status. Whilst some nutritional elements are present in excess, some others I am deficient in. This may be due to the secondary effect of toxic metal toxicity and the chelation therapy that followed.

When hair mineral analysis confirms that an abnormality is present, the next useful test is a challenged urine toxic metal report. This test, if performed carelessly, can cause serious damage to the body. If a decision is made to perform a challenged urine toxic metal test, it should be done under proper medical supervision.

Following pictures show the various challenged urine tests that I have undertaken.

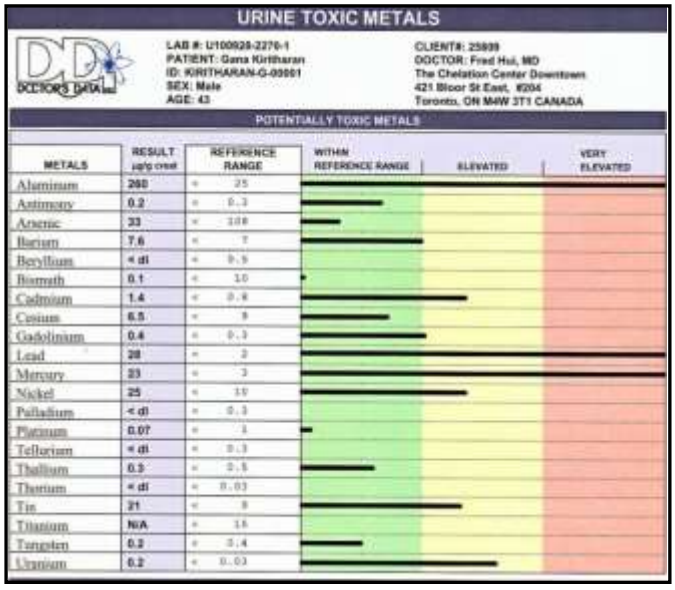


Fig 3.1: Challenged (DMPS & CaEDTA) Urine Test on 27th September 2010

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

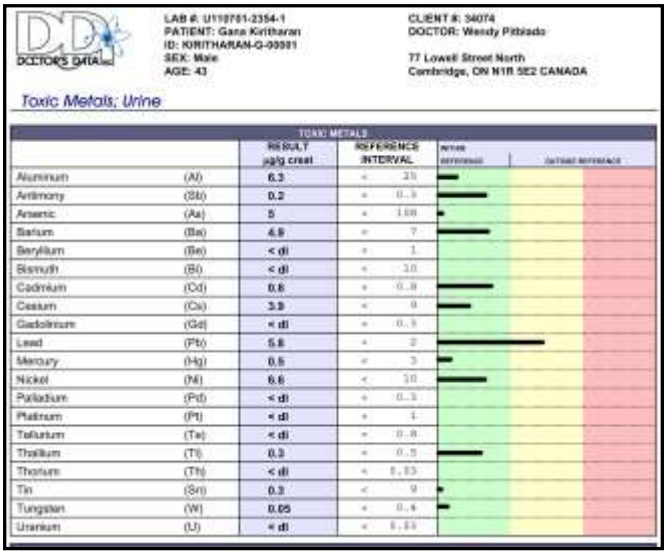


Fig 3.3: Challenged (DMSA) Urine Test on 30th June 2011 (After Two Weeks)

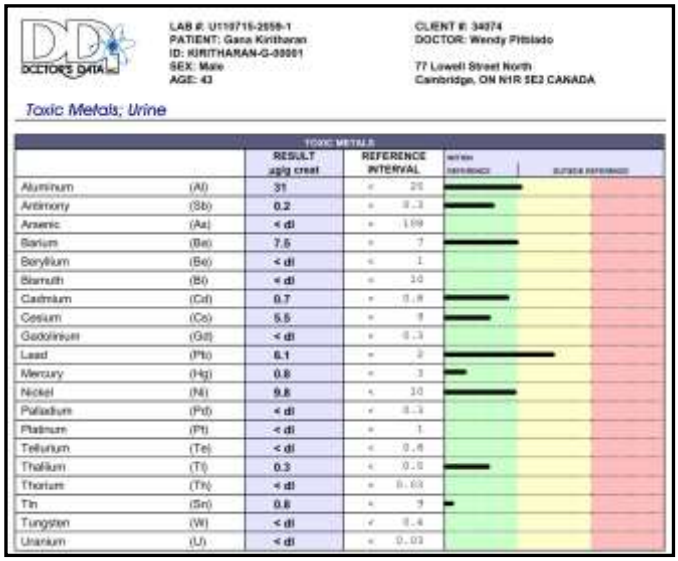


Fig 3.4: Challenged (ALA & DMSA) Urine Test on 14th July 2011 (After Two Weeks)

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

Fig 3.1 shows the challenged urine test conducted at the beginning of treatment. There is a large amount of toxic metals being excreted out via urine. This could be due to three reasons. 1) As I had just begun treatment, toxic metals were only just starting to be excreted. 2) It is possible that I was severely poisoned just before this period. 3) As I did not take a large amount of NAC, the kidneys might have been the only route of excretion of toxic metals (only a small amount via the Liver). Fig 3.2 and 3.3 shows challenged urine tests being conducted after 10 months of chelation (one by IV medication (DMPS & CaEDTA) and the other by oral medication(DMSA)). It could possibly explain how toxic metal excretion was reduced due to the chelation program involving a large amount of NAC (which could possibly act by helping to remove toxic metals through the liver). These tables also help to explain the difference between IV and oral chelation therapy. Even though IV therapy can help to excrete toxic metals rapidly, oral chelation is also equally as effective. Fig 3.4 displays a challenged urine test conducted after taking alpha liphoic acid (ALA) for 24 hours before undertaking the test. This test also shows how aluminum-like toxic metal excretion was increased by ALA.

My Battle with Toxic Metal Poisoning:

I do not know how long I have been poisoned toxic metals and what possibly caused it. During the past year, as the Ontario Medical and Judicial system have failed to protect me, I am now fighting a personal battle against a series of poisoning attempts with underlying criminal intent.

The possible attempts of poisoning, changes in treatment of my chronic toxic metal toxicity and diabetes, and changes in my fasting blood glucose values between 2010 - 2011 are displayed on the table and graph below. As shown, there is a high amount of fluctuations in my fasting blood glucose values. Various factors in the treatment of diabetes and toxic metal toxicity could have possibly caused this, the most logical explanation is that I may have been exposed to toxic metals.

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 food that I suspected was poisoned of which there could have been a couple of attempts. On 5th of August 2010, I received the first confirmatory medical report (Hair Mineral Analysis). On August 17th 2010, I tried to make a police complaint to the Toronto Police Services (TPS) however they failed to accept my complaint	I started supportive medication on 22nd June 2010 and started DMSA from 6th July 2010. I added NAC to my treatment regimen one week after. The DMSA dose was about 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 it decreased to 7.4 mmol/L on
Aug 18 2010 – Sep 26 2010	As I was ignored by the TPS, there may have been several more severe attempts to poison me through various different means..	Continuous supportive medication. I continued to take DMSA and NAC till 13th Sep 2010.	Maintained **Metformin 2000mg Glyburide 60 mg**	Increased up to 17.4 mmol/L
Sep 27 2010 - Nov 25 2010	When I went into formal medical care, the number of attempts to poison me may have decreased but there could have possibly been one or more attempts.	A challenged urine test was conducted on 27th Sep (DMPS, CaEDTA). I restarted DMSA 3 days after this test .	Maintained **Metformin 2000mg Glyburide 60 mg**	Decreased to below 10 mmol/L but it increased soon after.
Nov 26 2010 – Jan 24 2011	The number of attempts to poison me could have possibly decreased, but is also possible that a few attempts were made.	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. My DMSA supply was also interrupted for two weeks. The attempts to poison could have increased.	Skipped one week of DMSA. High doses of NAC. I started to add ALA to the treating regimine.	**Metformin 2000mg Insulin 20 - 26 Units/Day**	Increased and stabilized between 10 – 14 mmol/L
Mar 15 2011 – April 30 2011	There may have been more than one severe attempt. As a result I took extra precautions (sealed the ventilation duct of my room)	I took DMSA and ALA.. Increased the dose of Buffered Vit C and NAC.	**Metformin 2000mg Diet, Exercise, Herbal**	Stabilized between 10. – 16 mmol/L
May 1 2011 - Sep 30 2011	The poisoning attempts could have decreased or even possibly had stopped.	I maintained taking DMSA. High doses 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 have been an attempt to poison me with Arsenic.	DMSA, NAC, Vit C, ALA, and Coriander	**Metformin 2000 mg Diet, Exercise.**	Increased above 9 mmol/L

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

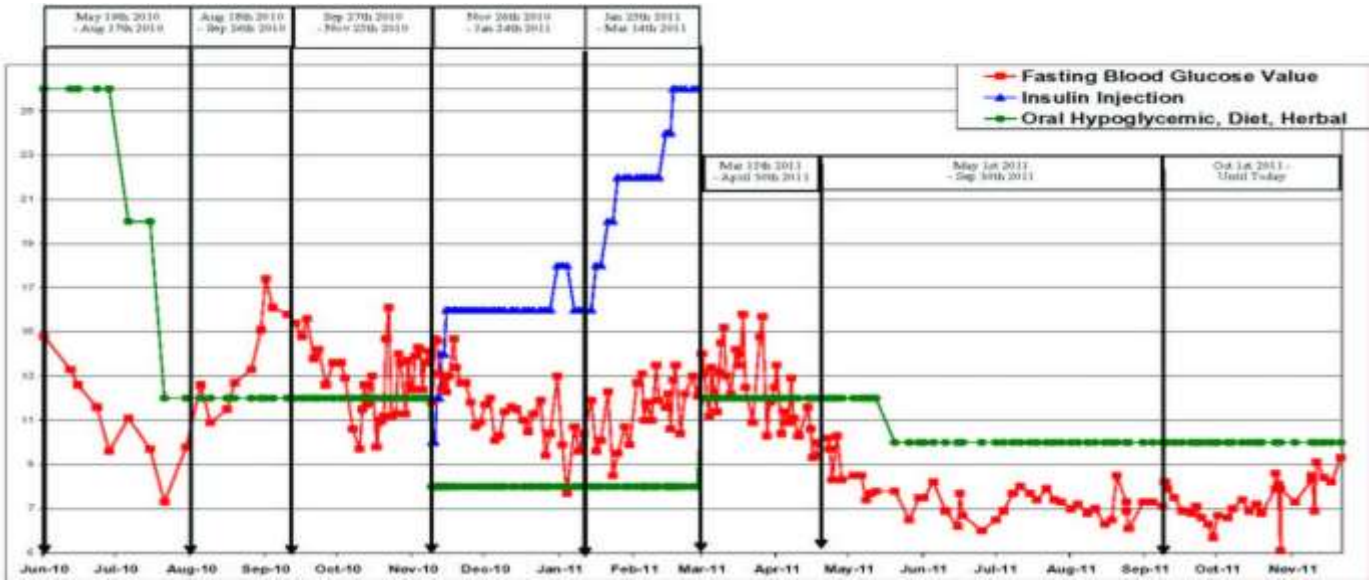


Fig 4: Gana Kiritharan's Battle with Toxic Metal Poisoning during 2010 – 2011.

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

In order to better understand how the exposure of toxic metals may have been a cause of my chronic medical problems, it is important to understand how toxic metals can cause damage to the human body. Toxic metals causes damage primarily by interfering with protein synthesis and its functions. Heavy metals interact with proteins which then goes onto forms highly insoluble sulfides [3].

In more detail, proteins are made up of 20 or more amino acids. Amongst the amino acids, two or more of them (methionine and cysteine) contains sulfur in their molecules. The sulfhydryl (-- SH) group of cysteine is important in different two ways. Usually proteins are synthesized as a single long polypeptide chain and then folded into varying different shapes and which then goes onto to develop their functionality. The sulfhydryl group of cysteine plays an important role in the folding of the polypeptide chains. Cysteine molecules, in two seperate locations of a single polypeptide chain, interact and form disulfide bonds (--S—S--), which results in the folding of polypeptide chains. Heavy metals disrupt this mechanism by binding with sulfhydryl groups of cysteine molecules, thus preventing the proper folding of polypeptide chains. Again, numerous enzymes contains one or more free sulfhydryl groups which react with nutritional elements such as Zn^{++} or other coenzymes. Heavy metals interact with the free sulfhydryl groups with the molecules of enzymes and other proteins, leading to a loss of their functionality.

The above explanation helps to understand why I believe that toxic metals are an important cause of my chronic medical problems. If you look at insulin and insulin receptors, both are first synthesized as a single polypeptide chain, which are then subsequently folded as a result of disulfide bonds. Then a portion of the polypeptide chain is removed and receives its functionality. Cadmium and other toxic metals interferes with the folding of theses proteins. Fickova M et al, [(1)] and Lei L J et al, [(2)] have already demonstrated the influence of cadmium on insulin and insulin receptor synthesis.

In their research, Lei L J et al, [(2)] demonstrated that cadmium does not affect serum insulin levels. The reason could possibly that even when cadmium interferes with insulin synthesis, the pancreas has a high reserve of already produced insulin and is capable of maintaining a normal insulin level. However, synthesis and the availability of insulin receptors are markedly affected by cadmium and other toxic metals. This has been demonstrated by Fickova M et al, [(1)] in detail in their research. There is two possible reasons for this. Firstly, there is not enough of a reserve in the synthesis of insulin receptors as compared to insulin. Secondly, the disulfide bond between the first and second pair of alpha and beta chains may be more susceptible to a toxic metal interaction. A more definitive answer requires more research however. My experience through treatment also supports the above research findings. During my treatment when I trialled insulin injections, it not only failed to bring better control but it also brought about side effects which can be attributed to hyperinsulinemia.

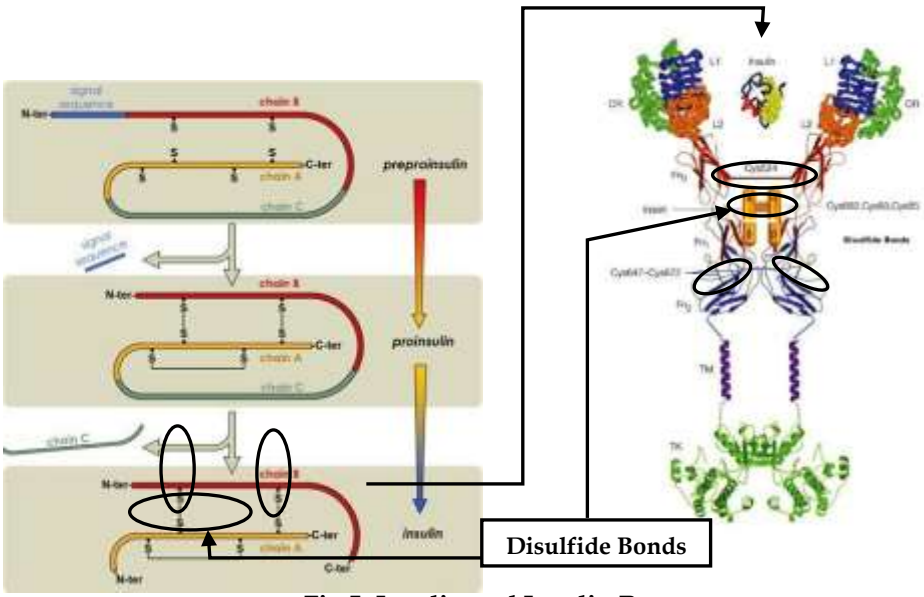


Fig 5: Insulin and Insulin Receptor.

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 begun to experience a high level of fluctuation in my fasting blood glucose values. During the past year, my fasting serum glucose value increased above 16 mmol/L on two occasions and it also decreased down to 8 mmol/L twice. During the most part of this period, I maintained taking a stable dose of oral hypoglycemic drugs. I therefore believe that levels of toxic metals in my body was the main reason for this fluctuation. In order to better understand how the exposure of toxic metals may have been a cause of this fluctuation, we need to have a better understanding of the word chelation and other anti-toxins that helped me to achieve an improvement over the control of my blood glucose levels.

The definition of chelation is; "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 a medical sense, chelation means removing the toxic metals from the body using medicinal substances. The medicinal substances used in chelation form two or more bonds with toxic metals which help to remove them via the liver or through the kidneys.

Although I am taking specific chelation medications which could possibly be acting to remove these toxic metals from my body, I am also taking several non-specific anti-toxins which help to reduce the general toxic level of my body. I am taking vitamin E at a dose of 800 IU or more. A normal vitamin E tablet contains 50 IU. Also I am taking 3000 mg or more of vitamin C, its normal dose is 90 – 500 mg. Alongside this, I am also taking a high dose of Vitamin A, B and D. I am also taking high doses of minerals (not including Fe and Cu). The chelating medication that I am taking can be divided into two different groups. The first group is made up of synthetic medical substances which include; DMSA (Meso-2-3-dimercaptosuccinic acid). It has two sulfhydryl (-- SH) groups which form tight bonds with toxic metals and removes them safely through the kidneys. An important property of these synthetic chelators is that they usually do not go intracellularly. They are only able to remove toxic metals from interstitial fluids or toxic metals circulating in the blood. The second group is made up of more natural substances, such as NAC (N-Acetylcysteine). It is a precursor of glutathione which is the body's natural chelators. Unlike synthetic chelators, glutathione is able to act intracellularly and remove toxic metals. Also, liver glutathione levels are important in determining the levels of toxic metals being excreted via the liver. NAC also helps to maintain a normal liver glutathione level, thus allowing for the excretion of toxic metals via the liver.

When I was taking DMSA alone, my fasting blood glucose levels dropped below to 10 mmol/L. However, when I took DMSA with NAC, my fasting blood glucose levels decreased to 8 mmol/L on two occasions. When I was taking a lower dose of DMSA with a higher dose of NAC, I experienced a drastic increase in my fasting and postprandial blood glucose levels. This may be due to NAC mobilizing toxic metal stores from the intracellular space and thus causing an increased toxicity.

Another naturally occurring substance I am trying to add into my chelating program is ALA (alpha liphoic acid). ALA is able to cross the blood brain barrier. If taken at the early stages of the chelating program, it could potentially cause toxic metal deposits into the brain. I started to add it after six months of taking DMSA and NAC. This is when I started to experience an easing of my CNS symptoms which were brought about as a result of my chronic toxic metal toxicity, however my fasting blood glucose level started slightly increase. This may have been due to ALA mobilizing intracellular and CNS stores of toxic metals.

A few important questions remain unanswered. Why am I unable to achieve sustained control over my diabetic problem and whether treatment for toxic metal toxicity will give me a long lasting cure from metabolic syndrome? The answer depends on two issues. Whether I will be able to achieve an environment free of these toxic metals and whether I will be able to receive the necessary financial resources to get the best possible treatment it. The latest attempt to poison me may have happened between March – April 2011, this was almost a year after I discovered that I was being poisoned with an underlying criminal intent. Many times, I am left with one or two dollars in my pocket whilst waiting for the next paycheck. In 2004-2007, I experienced an interference in my opportunities for employment and my hours of work suffered as well. Several experts recommend an infrared sauna, as this helps to remove toxic metals through the skin. I would like to buy one (costs around CAD $ 1500.00) however I am unable to afford it.

Women's Medical Problems [3,9]:

The reason as to why I am claiming that toxic metal toxicity is a common but disregarded medical problem is due to my mother's possible long term experience with it. My mother's first chronic medical problem was bronchial asthma. She possibly first had this in her 30s. Following this, the next problem she begun to experience was sleep

disturbances and disturbing dreams, which continued throughout her life. During her late 30s she experienced a panic attack one night which led to the family doctor to give her something to aid her sleep. My mom was a vegetarian, but on doctors advice she started to eat non-vegetarian food in her early 40s and she found that her symptoms eased to some extent. In her 40s she was diagnosed with hypothyroidism. During her 40s and 50s she developed a frozen shoulder and a herpes zoster infection. From her early 50s she experienced gingival hypoplasia. And finally on her 61st year of age she passed away due to a (anteroseptal) myocardial infarction.

When I searched for the possible cause of her toxicity, I realized that she may get have gotten it from firewood used in the kitchen. If my thoughts are correct the many other women who are exposed to kitchen smoke also could possibly suffer from toxic metal toxicity and could go onto experience related medical problems. When I searched for more evidence regarding this, I found two pieces of literature around this topic. The first one is in Tamil literature. Thruvalluvar, a person who is considered a philosopher of Tamil wisdom, described female leadership as the following.

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

If you look further into Tamil literature, similar comments and stories describing female psychology can be found several times. Toxic metal toxicity can cause several psychological disturbances on victims such as; feelings of insecurity, feelings of suspicion, and panic attacks are all associated with cadmium and other toxic metals in toxic doses.

The second piece of evidence I found is that menopausal syndrome shares several similar clinical features with toxic metal toxicity. The clinical features of menopausal syndrome are; hot flushes and night sweats, difficulty falling asleep, cognitive difficulties, depression, irritability, and decalcification of bone are all characteristic of toxic metal toxicity as well. If we can answer how estrogen protects women up until menopause, this could possibly open a new intellectual debate. Estrogen is a steroid synthesized in the body from cholesterol. It has a free – OH group. Whether it forms an oxide with toxic metals and work as a chelator or that –CH3 (Methyl) compounds are released during the synthesis of estrogen to give some temporary protection against these toxic metals is a question for future research.

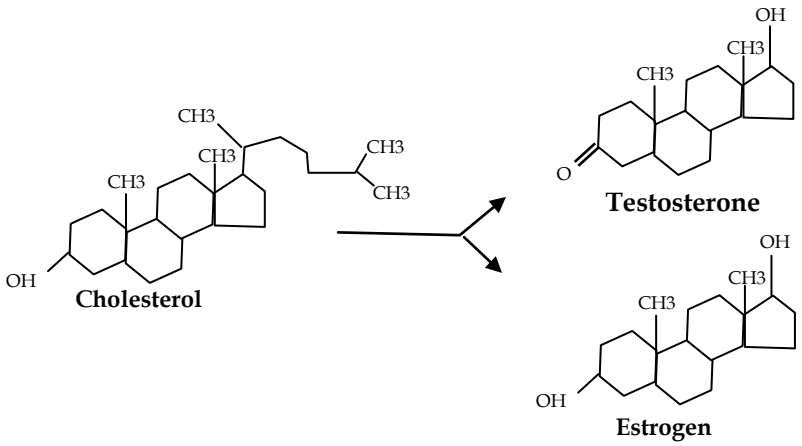


Fig : 6 Estrogen Synthesis

An unacceptable Level of Negligence:

When I realized that I was a victim of being poisoned with mercury with an underlying criminal intent, one of the major challenges I faced was being properly diagnosed and receiving treatment, this was and still is an unacceptable level of negligence in the healthcare profession regarding my medical situation. When I realized I might have been poisoned, I was unable to call the police immediately. The well organized nature and powerful authority of the people behind this criminal conspiracy has prevented me expecting an accountable court order from the Ontario Judiciary. Even though I attempted to make an official police complaint on 17th August 2010, when I got the first confirming medical report, I did not have hope that the Ontario Judiciary would have protected me from the sequence of criminal offences that I was a victim of. I do not know the reason as to why this is, whether it is institutionalized racism or corruption at the highest level, the Ontario Judiciary has showed a high level of ignorance, irresponsibility, and incompetence in protecting from the sequence of criminal offences that I was a victim of.

Medically speak, when I called the Poison Center at 416 813 5900, the only help I received was some advice to talk to my family doctor. My family doctor ordered the wrong test (Blood Mercury) to check whether I am suffering from a chronic form of mercury poisoning. The

result was a blood mercury value of 15.4 nmol/L. This could possibly confirm that I was suffering from a certain level of toxicity. Despite several public documents giving a reference value of 0 – 3 nmol/L, the report gave a value of < 18.0 nmol/L as the reference range. This lead to many medical professionals to interpret the result as normal. My argument that mercury is an unwanted toxic metal and that 0 nmol/L may should be seen as the healthy level was ignored by medical professionals. This forced me to walk away from the Ontario healthcare system and to spend out of my own pocket to obtain report analyzing my hair and urine to have a closer look of the toxic metals present.

Even after obtaining the health report, which confirmed toxic levels of toxic metals being present, the problem was still yet to be solved. A consultant refused to look into the report and described the chelating doctors as money makers and that my efforts is often described as a result of various psychiatric problems. What was the reason for this refusal and denial? If you look back at the history, you find that intellectuals and other forms of social leadership (religious, political, and business) refused to accept the truth told to them at that point in time. Let us discuss the reasons for the challenges that are faced by scientists to bring out the truth so that not only does my medical problem becomes accepted and that I and similar patients receive better treatment for it, but also that future scientists are able to improve their knowledge fewer hindrances.

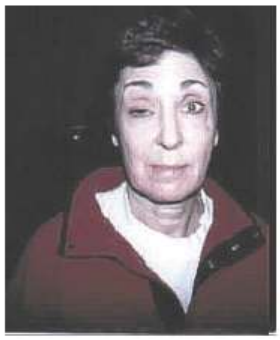
The first challenge is vision. Human vision has limitations and our explanations about the environment is mainly based on our vision. Some people are born with the gift of seeing better than normal vision and thus give better a explanation about our environment and its problems. Another thing that increases our vision is technology. For example, the telescope helps to give a better view into the universe and helps to give a more detailed explanation about it. What is the visual problem in toxic metal toxicity? The same way that the telescope brought about a better understanding about the universe, the microscope brought about a better understanding of smaller objects. There was a time that scientists fought with religion to dissect the human body in order to give a better explanation of anatomy. The next level of this battle happened when scientists tried to examine human body parts under the microscope. Today the problem that we may have is to go to the next level, which is the biochemical level. The technology we have today has its limitations in giving a black and white (or colourful) picture of what is happening at the biochemical level intracellularly. This lead to the making of several conclusions based only on its clinical presentation. Only people with a deeper vision can understand and explain what is happening at a biochemical level inside cells to help to move the medical profession into next level of thinking.

Another challenge is the improper distribution of social authority. When a society is organized with people with better vision without much authority in one corner and people with fundamental ways of thinking with more authority in another corner, a conflict results and the truth gets suppressed for a long period of time. The best documented conflict in humanity happened between Galileo and the Rome Court. Giving the authority to the people with better vision may look like a solution, but even in modern times this rarely happens. In Thailand, authority was given to people's with vision only after the 2004 Tsunami, not before it. If you look into this problem deeper, nowadays, whether it is mad cow disaster in the UK or a tsunami in South Asia, it was the fear of losing businesses and short term profit motive put up barriers on scientists bringing out the truth on time and educating the public about an impending disaster. Today religions may have lost social authority but this authority did not go to the scientists who needed it. Instead it went to the major business institutions who continuously try to put up barriers for scientist to prevent them from bringing out the truth for several unacceptable reasons and putting humanity in face of disaster again and again.

Another important challenge is understanding the word science. An important argument that is put forward by people who disagree with toxicity due to toxic metals is that several scientific experiments fail to show any benefit from chelating. There are several reasons for this misunderstanding. An important one is that there is not yet an established and universally accepted way of treating this problem. Today, the medical profession has successfully established treatment methods for many different medical problems. Most of these treatments last from a few days to a few months. But treatment for toxic metal toxicity needs to be followed anywhere from several months to a few years. Several studies that have been conducted regarding toxic metal toxicity follows a short duration of chelating therapy which may have failed to bring about any measurable benefit.

An important argument that I want to put forward here is what do we understand about the word 'Science'. Science is a method of analysis of the environment and finding solutions for its problems. It disagrees with any fundamental arguments and accepts experiments are a way of finding solutions for problems. But expecting that all the solutions for our problems should come through proper scientific experiments is likeable to creating another religion in the name of science. If simple observations and logical explanation can help us to come to a solution for a problem, then waiting for a scientific experiment is just a waste of time and money. From Aristotle to Galileo, many have conducted several experiments to understand how and why objects were fall

towards earth. But it was Newton's simple observation of a falling apple and the logical explanation for it that helped to define "Gravity". Today, several stories of patients who benefited from the treatment of toxic metal toxicity might make any scientific experiments on the topic an 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:

An important question that arises about toxic metals is; how common is it as a human health problem? Usually modern industrialization is blamed for exposing human health to these toxic metals but as I explained earlier when I searched for a logical explanation for my mother's possible toxicity firewood was a possible source. We have to accept the fact that as how microorganisms are common in our environment, toxic metals are also an unavoidable reality. But as humanity has established a successful way of preventing, diagnosing, and treating microorganisms; preventing, diagnosing and treating toxic metal toxicity can also be achieved. All of which can only happen after the medical profession comes forward to accept it as a common medical problem. When I searched the WHO for an ICD classification of my medical situation, I failed to find a disease category similar to "Chronic form of Toxic Metal Toxicity".

More important information about toxic metal toxicity is the difficulty in detoxifying them. Several harmful substances to human health can easily be detoxified. For example, TB bacilli and the AIDS virus can be killed easily by bright sunlight. But detoxifying toxic metals is near impossible. As they are base elements in chemical structure, they try to stay in their toxic form forever. Even if we dilute them with air and water; plants, animals, and fish have the capacity to concentrate these toxic metals and put it back into the food chain. If we really wanted to protect human health from toxic metals, we need an organized long-term strategy to decrease the concentration of toxic metals in our environment.

This may be why a Health Canada document titled "The Risk of Mercury Poisoning" says "the government of Canada is working in a number of areas to reduce the use and release of mercury into the environment.". However, anonymous people are adding unspecified amounts of these toxic metals to the Toronto ecological environment. I was unable to find somebody who could question these people or even to stop their irresponsible acts.

I am calling on healthcare professionals and managers not just in Canada but also worldwide to not to wait for a disaster to happen but to accept this medical situation and to come forward to create better prevention, diagnosis and treatment protocols.

© Gana Kiritharan 2011.

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.

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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.

Following Pages Contains Summary of Gana Kiritharan's Lab Reports:

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Dr. Mansula Manogaran

Dr.Mansula Manogaran, Nephrologist

RVH-Centenary Hospital,2863 Ellesmere Rd, #303
Toronto, Ontario, M1E 5E9

Phone: 647-436-3397
Fax: 647-436-3403

Date:	2018/09/18
To:	
Address:	
Phone:	
Fax:	

Patient:	KIRITHARAN, GANA
Address:	6 Rosebank Dr,#9L
	scarborough, ON M1B0A1
Phone:	416-820-8581
Birthdate:	1967/09/09
Health Card No:	1438520023 AX

Mr.Kiritharan is a 51-year-old gentleman who is known to me since February 2011. He fellow's-up with me regarding chronic kidney disease with proteinurea.
His past medical history's remarkable type II diabetes since 2004, hypertension and dyslipidemia.

He is on Altace 10 mg daily, Metformin 1000mg b.i.d., Metoprolol 25 mg bid and Lipitor 5 mg daily. He has no known drug allergies.

He has never been a smoker or drinker. He works as a security guard. Family history is strongly positive for diabetes and his mother died from myocardial infarction at age of 61.
On physical examination, he appears we. Blood pressure was 135/80. Heart rate was 90 and regular. Head and neck examination was unremarkable. Chest was clear. Cardiac examination reveals regular rhythm and normal heart sounds. Abdomen was benign. There was no edema in the extremities.

July 2018; Fasting blood sugar 15.2. Hemoglobin A1c 10.9. Serum creatinine 57. EGFR 114. Serum sodium 138. Serum potassium 5.1.
July 2017; Fasting blood sugar 12.0. Hemoglobin A1c 9.2. Serum creatinine 57. EGFR 115. Random urine albumin 46 mg/L. Albumin/creatinine ratio 5.4.
August 2016; Fasting blood sugar 17.3. Hemoglobin A1c 11.6. Fasting cholesterol 4.0. Triglycerides 3.6. HDL-cholesterol 1.19. Random urine albumin 74.2 mg/L. Albumin/creatinine ratio 9.6.
August 2015; Fasting blood sugar 19.0. Hemoglobin A1c 12.8. Serum creatinine 67. EGFR 109. Urinalysis was positive for 0.3 g/L. Random urine albumin 80.1.
August 2014; Fasting blood 18.7. Hemoglobin A1c 11.8. Hemoglobin 162. Serum creatinine 65. EGFR 114. Fasting cholesterol 6.6. Triglycerides 8.43. Urinalysis was positive for protein 0.3 g/L. Random urine albumin 96.2 mg/L.
February 2011; Fasting blood sugar 11.8. Hemoglobin A1c 10.7. Hemoglobin 150. Serum creatinine 64. EGFR over 90.Serum calcium 2.62. Serum albumin 50. Fasting cholesterol 5.42. Triglycerides 5.84. HDL-cholesterol 1.27. ALT 29. ANA was negative. Compliments were normal. Serum protein electrophoresis was negative for M protein. Urinalysis was negative for protein, glucose, blood or infection. Random urine albumin was 10 mg/L. Albumin/creatinine ratio less than 2.0.

In summary,Mr. Gana Kiritharan is 51 years old. He has long-standing diabetes. He's known to me since 2011. He was referred with proteinuria.
His blood pressure has been in good control, but blood sugar has been poorly controlled. Lipid profile is better after starting Lipitor.
I believe he has diabetic nephropathy. There was evidence of proteinuria without microhematuria. Serum creatinine has been stable over the last 7 years. There is no significant change in proteinuria either. However, he is on Altace.
He experiences numbness and tingling in his both feet.
It's well-known optimal blood sugar control is important to slow down renal deterioration. He is explained many times, but he wanted to keep his blood sugar higher and he thinks higher blood sugar protects his kidneys.
Best regards;

Dr. Mansula Manogaran Dictated, but not read

M. Mu

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Dr. Fred Hui M.D.
421 Bloor Street East, Toronto, ON
Tel: (416) 920-4200 Fax: (416) 920-4204

October 2, 2018

To Whom It May Concern

GANA KIRITHARAN is a diagnosed case of diabetes mellitus type II. His blood sugar is still not under control and HbA1c is more than 10. His diet also is still not optimal and he is not following a low carb diet.

He refuses to take additional medication other than Metformin to control his sugar levels.

He has his own theory about the role of heavy metals as the main reason for his diabetes.

I disagree with his opinion and am worried that he will suffer from neurological and cardiovascular complications down the road.

Should you require further discussion on this case, please do not hesitate to contact me.

Thank you.

Very truly yours,

Fred Hui, MD

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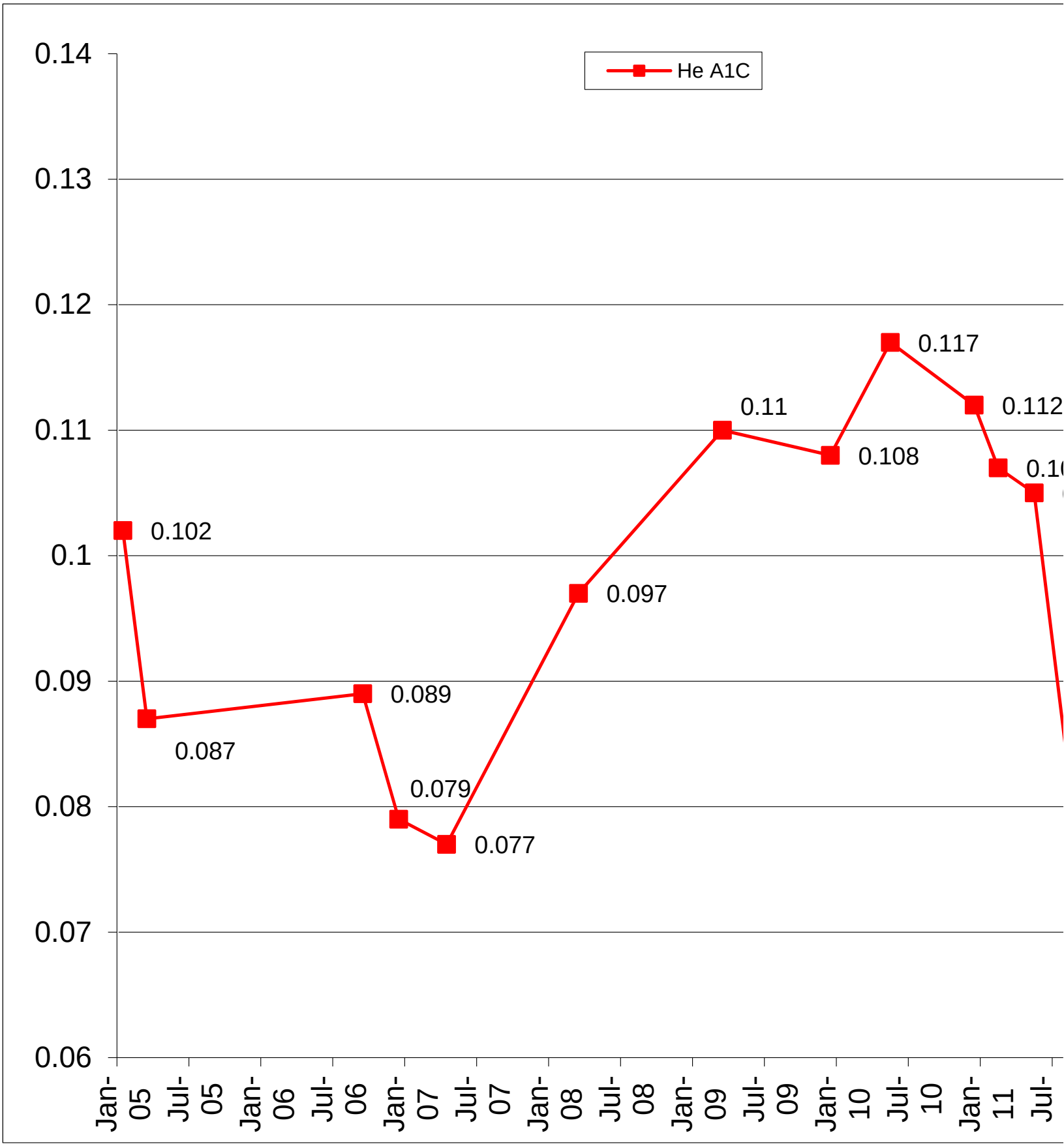

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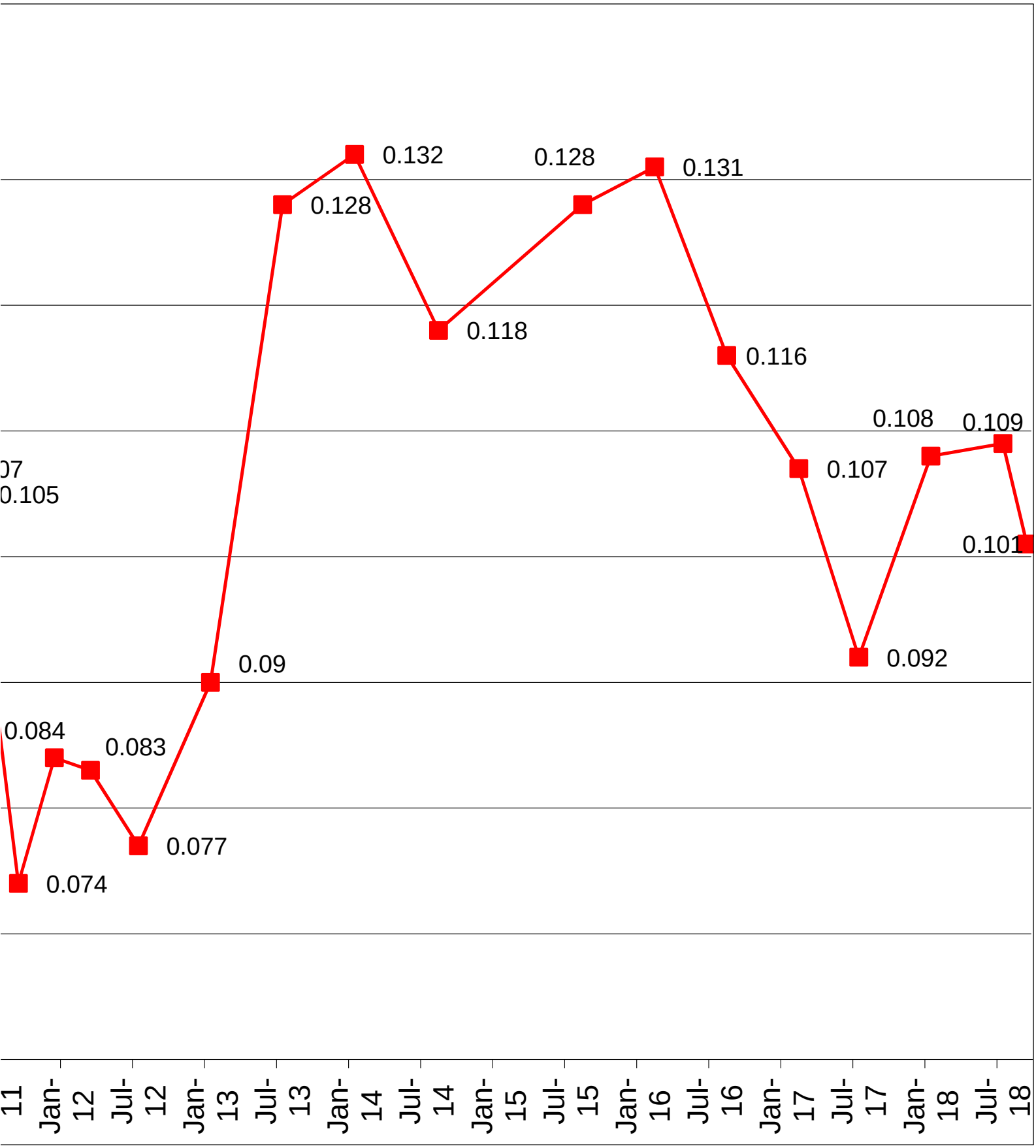

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Type of Report =>	Hair Mineral Analysis			
Report Number =>	**H 1**	**H 2**	**H3**	**H4**
Date of Test =>	July 2010	July 2011	July 2012	July 2013
Time Interval =>	Initial	Af 1 Year	Af 2 year	Af 3 Year
Provoking Agent =>				
Scale => / **Toxic Metals**				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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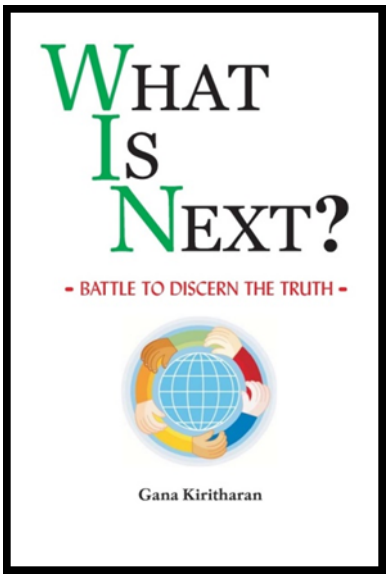
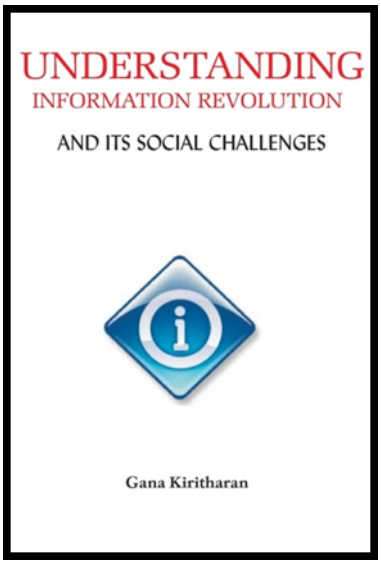
OTHER GANA KIRITHARAN'S INTERLECTUAL WORKS

BOOKS

ARTICLES





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