

# Diabetes

## Case Study

*A New Perspective on Type 1 and Type 2*

By Paul J. Tubiana

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<http://www.diabetescasestudy.com>

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## Preface

It is a privilege for me to add a parental note to Paul's diabetic case study.

As Paul's father, it broke my heart when my wife first called me from the hospital to inform me that Paul, who was seven years old at that time, was diagnosed with juvenile diabetes. When we came to the United States, Paul was very young and very active and he had a very strong will that no one could take from him. Until this day, when he decides on doing something, he will not rest until it's done. From the first days in the hospital he was objecting to the treatment the team of doctors had prescribed for him.

What child at Paul's age would have understood that he had to take daily insulin shots? Paul was never convinced that the treatment was necessary; nevertheless he succeeded to cope with the new situation without losing his hope that sooner or later the truth would emerge. Since then he kept a close eye on each fluctuation of his blood glucose. He seemed to manage his insulin dose without having any medical guidance.

Honestly, I never understood what diabetes was, even when I myself developed type 2 diabetes at age 67. Reading this study has helped me to better understand my actual condition.

As I tried to understand what diabetes was, my wife handed me a brochure to read. Unfortunately the content showed the worse case scenario of diabetes complications without explaining much and without giving us any hope. We as parents didn't have any guidance on how to handle this new situation, except that every day was a guess of how much insulin and how much food we should give him.

We suffered as much as he did when he felt lousy. Every night was a worry because of the possibility of hyperglycemia or hypoglycemia occurring. Paul's determination kept him aware of the state of his health to the point that this preoccupation took center stage, overshadowing any other interest.

I am grateful that at this point in his life he seems to see the light at the end of the tunnel. Knowing of his multiple talents it is our sincere hope that his newly found improved health condition will open a new horizon and new opportunities for him.

Emile Tubiana

September 2003

My special thanks to my endocrinologist, who freed me from the bondage of hopelessness that Type 1 diabetes, brings, and who enabled me to reach these conclusions. Also my special thanks to the makers of Lantus® insulin, for improving the quality of my life.

I dedicate this case study to my parents, my family, the countless people who have helped me along the way, and especially to the children and adults, who were once children, who may benefit from it.

## Introduction

I have been living with insulin dependent diabetes (type 1) for 21 years. I was diagnosed at age 7. The reason that I have been an insulin dependent diabetic is in large part due to the fact that on initial diagnosis the pediatric endocrinologists at a major hospital did not do two things:

1. A C-Peptide test.
2. Administer antibiotics.

I really have type 2 diabetes and that is what I had at age 7, just like older people who get type 2 diabetes. This is an important fact because type 2 diabetes is a curable condition, but instead of investigating the cause of my high blood sugars, I was put on insulin and told that I would have to take shots for the rest of my life. This is where the vicious cycle of insulin dependency began. One thing is certain: I have only been overweight while on insulin. There was no interest on the part of the doctors to find the truth and grossly unscientific assumptions were made about me to arrive at this diagnosis. This simple misdiagnosis cost me my entire childhood and made my life a nightmare. The only explanations that I received from doctors were:

Your pancreas does not work.  
We don't know what causes this.  
You should just accept what you have and live with it.  
You are not doing enough to control your blood sugars.

I knew from day one that the doctors were wrong, but the burden of proof was on me. And so it took me 21 years to prove it, by solving this mystery. I knew something was wrong because I felt something that went against my feeling of well-being. They were the experts. They failed to do their jobs properly and scientifically to arrive at an accurate objective conclusion, given the tools they had available to work with at the time. And they being the experts meant that I had nothing of value to contribute to my own well-being. They never talked with me as if I were a human being. They really did not ask me anything, nor did they discuss my condition with me. I felt excluded. I was treated like an object without a life or a soul. They were free to do as they pleased with my body. They knew it all. Who was I at age 7 to argue with these intelligent, well-educated, long schooled professionals, who claimed to know my body better than I did?

After 9 years of visiting these doctors, I got fed up and decided not to see them anymore, because they were more concerned about whether or not I had a girlfriend in my freshman year of high school than finding the truth about my diabetic condition. I saw many other doctors subsequently, but the original misdiagnosis stuck to me and no one bothered looking any further except me.

## The Cause of Diabetes

Diabetes is a condition of glucose toxicity and not of insulin deficiency. Diabetes is caused by sugars, carbohydrates, elevated blood glucose (sugar), and infections. Diabetes is very much a condition of imbalances with multiple layers of causes, which constitute a vicious circle.

Sugar is like a powerful “drug”; it gives a quick rush, an initial boost of energy that quickly diminishes after 2-3 hours, resulting in a withdrawal effect on the body, and causing a craving for more.

Another way of looking at it is that sugars increase blood glucose concentration quickly, triggering a large insulin release from the pancreas, which then lowers the blood glucose just as quickly, even beyond the original starting point.

Carbohydrates (e.g., bread and pasta) are the “slow-release” or “extended-release” version of sugar. Their effect on blood glucose is less dramatic but longer-lived. Additional intakes will over layer, compounding the effect, making carbohydrates more potent than sugar.

I know that these are significant factors because I used two to three times more insulin when eating carbohydrates or sugars. Modern life has reduced the amount of physical demand on the body, thereby increasing the effects of carbohydrates and sugars.

Infections (microorganisms, especially bacteria) feed on sugars and blood glucose, which in turn increase the presence of infections in the body. As the microorganism population increases, blood glucose concentrations increase, to high levels that are equal to or exceed that of what sugars and carbohydrates do to blood glucose. I will not try explaining though, how infections increase blood glucose concentrations because I do not know. Through careful observations, I do note that the presence of infections is the most prominent feature that causes me to be hyperglycemic.

The relationship of sugars and carbohydrates to infections in the body forms a loop, or a vicious self-compounding circle, so long as sugar is constantly added to the system, until the infection’s effect on blood glucose becomes self-sustaining and takes a life of its own.

Increased sugar and carbohydrate consumption increases blood glucose concentration, which results in more insulin release, and for longer periods of time. This insulin promotes fat storage. Eventually the fat causes insulin resistance, which prevents the body from regulating blood glucose properly. This in turn invites and provides a perfect medium for — infections, especially bacterial, to flourish. If the population of infectious microorganisms in the body increases to a large enough extent, it compromises the immune system. Also, the capacity of the pancreas to produce enough insulin to lower its effects is exceeded, causing ketosis (breakdown of fat — which increases blood glucose). When blood glucose concentration passes 300 milligrams per deciliter (mg/dl) it becomes ketoacidosis, a life threatening condition that must be treated with exogenous insulin.

There are two main keys to understanding diabetes. The first key is to understand blood glucose levels, and why they do what they do, then to pinpoint the cause. The second key is the C-Peptide test, which will be explained later. A holistic approach must be taken, not ignoring any details in one’s life.

## The Cause of Hyperglycemia — Elevated Blood Glucose

On average, I monitor my blood glucose 6-10 times a day and I have through careful observation over time concluded that these factors cause blood glucose concentration to increase, in order of strength:

- Infections (especially bacterial) — They feed on sugar in the digestive system and on blood glucose, they increase blood glucose concentrations to very high levels. Elevated and fluctuating blood glucose will feed infections.
- Carbohydrates — The strong extended-release version of sugar which increase blood glucose.
- Sugars — Potent but short lived. They increase blood glucose.
- Lactose (sugar from dairy) — The medium strength extended-release version of sugar. It increases blood glucose.
- Over consumption — Food intakes build up in the digestive system, which continually releases glucose into the blood stream. Blood glucose increases.
- Bowel retention — because one is not allowed or otherwise unable to use the restroom (WC). Creates a condition similar to over consumption. This may increase blood glucose.
- Constipation — Creates a condition similar to over consumption. This can be corrected by adding more fiber to the diet (laxative effect.)
- Glucagons — A hormone that is released from the alpha cells of the pancreas into the blood stream, which increases blood glucose concentration.
- Stress — Bad stress leads to bowel retention, constipation, and glucagon release. Good stress will decrease blood glucose concentration.
- Anger — Causes glucagon release.
- Fear — Causes glucagon release.
- Anxiety — Causes bowel retention, which increases blood glucose concentration and glucagon release.
- Other negative thoughts or feelings — Cause glucagon release.
- The dawn effect — Blood glucose concentration decreases at about 5:00 a.m., and then increases much higher at about 8:00 a.m. This occurs naturally even under fasting conditions.
- Ketosis — Breakdown of fat, that converts to glucose, and increases blood glucose concentrations.
- Ketoacidosis — Ketosis with high blood glucose levels (above 300 milligrams/deciliter) due to the fact that there is not enough insulin present for the glucose load. A great feeding source for infections.
- Lack of physical activity — Excess glucose in the system is not used, increasing blood glucose concentration. Increases presence of infections. Blood glucose is converted and stored as fat, which leads to insulin resistance.
- Some medications — Can increase blood glucose. Usually there is a warning to diabetics on the label.



- Exercise or physical activity — Will lower blood glucose somewhat, but heavy or sporadic exercise will cause a sudden drop in blood glucose and cause glucagon to be released, which will increase blood glucose. Exercise will also result in food cravings, which will result in excessive eating, increasing blood glucose. Depending on the type of physical activity, it may combine with other factors and increase blood glucose levels. Routine and moderate exercise is good for blood glucose control.

The combination and compounding of some or all of the above factors contribute to the vicious self-compounding circle of hyperglycemia (diabetes). Each individual factor is a unique feature that causes the body to be out of balance. The difficulty is to find all the right balance points.

From what I have thus far presented a clear link can be drawn between sugars, carbohydrates, blood glucose, infections, and how children behave. Blood glucose fluctuations do affect the mood. Sudden increases in blood glucose and elevated blood glucose can cause hyperactivity, irritability, discomfort, anger, and sluggishness. Children with these behavioral characteristics may be exhibiting symptoms related to diet and infections.

## Insulin

Hormones are the body's mechanisms for adapting its functions to changes in the environment. Insulin is a hormone. Only the body itself, when in balance, can properly determine the correct amount of hormones to release. One should note the controversies in recent years concerning other hormone replacements.

Insulin does two things:

1. Lowers blood glucose.
2. Promotes fat storage.

Although these two functions are related, it is better to view them as independent and separate, for the sake of understanding diabetes.

When exogenous (bottled) insulin is used in type 1 diabetics it promotes fat storage. When endogenous (human source) insulin is released from the pancreas, through the action of an oral medicine used by type 2 diabetics to stimulate extra insulin release, it promotes fat storage. When endogenous insulin is released due to an increase in blood glucose concentrations from a food intake in normal non-diabetic persons, it promotes fat storage. The more insulin in the system, the more fat is stored. The more fat is stored, the more insulin resistance there is. The more insulin resistance there is, the more insulin is required to lower blood glucose concentration. This is also a vicious self-compounding circle.

I have only been overweight while on insulin. I wonder if overweight people don't have too much insulin in their bodies, which is a sign of a heavy glucose load, the cause of which has not been properly identified yet.

The only reason that the pancreas releases insulin is in response to a glucose (sugar) load, or an increase, or a sudden increase in the amount of glucose (sugar) in the system. Glucose stimulates insulin release. The goal should be to use the least amount of insulin that will keep blood sugar normal.

Therefore, in order to control the amount of insulin released, one needs to control the glucose entering the system, which stimulates insulin release. A long fast should stop most glucose concentration increases in the blood, as well as most insulin release from the pancreas. Fasting may be difficult if not impossible for some bottled insulin users because of insulin peaks (period of time when insulin is more active). Insulin peaks necessitate food intake. Ketosis will occur when fasting, increasing blood glucose levels slightly. Then the cause of hyperglycemia can be narrowed down to other things such as infections.

Frequent fasting to control blood glucose levels is not recommended because it messes up the digestive system and basically makes one sick. Instead, a low glycemic (low sugar content) diet over the long term is a better goal. Always keep blood glucose in the normal range. If necessary gradually decrease the dose of insulin, or oral agent that promotes insulin release, accordingly. When using an oral agent that promotes insulin release, think of it as taking insulin.

If an insulin user stops using insulin suddenly, the absence of that insulin will result in ketosis (breakdown of fat to glucose), then increasing blood glucose levels, resulting in diabetic ketoacidosis, a life threatening condition.

The glucose lowering function of endogenous insulin and of exogenous insulin may or may not lower blood glucose levels efficiently or effectively due to the multiple compounding factors causing blood glucose levels to rise, as listed above. The main factors are sugars, carbohydrates, and infectious diseases, but these are not all inclusive.

What this means is that for a given glucose concentration load, the amount of insulin that is needed to control that blood glucose load to normal levels (80-120 mg/dl) may be either small, large, fixed, variable, routine, or ever-changing, due to the blood sugar raising factors listed above.

Most diabetics on insulin that I have met have reported having had a cold-like infection, and losing a lot of weight prior to diagnosis for diabetes.

I will explain this:

An infection causes the blood glucose concentration to rise above the capacity of the pancreas to produce enough insulin to lower it. This sends the body into a condition of ketosis, which is the breakdown of fat into glucose. This is why weight loss occurs. This glucose (from fat) increases blood glucose levels even further, resulting in diabetic ketoacidosis.

One factor that should be considered in Juvenile Diabetes is growth. A child is growing all the time, the body is always changing, and the amount of fat stored in the body is changing. Think of an older person, who has a poor diet, doesn't exercise, and gains weight: that person becomes a type 2 diabetic. This is because of insulin resistance and perhaps disease. Some pregnant women temporarily get type 2 diabetes. This is attributable to the changes in weight. Stress is also known to be a cause of temporary type 2 diabetes.

Why should a child be any different? The insulin producing capacity of the pancreas in relation to the size and fat content of a child's body is always changing. At certain points in the growth development of the child, the system may be taxed to the limit. Sugars, carbohydrates, inactivity, and particularly infections trigger diabetes. Just as in an adult, if a child is started on insulin at a young age, the child's body will develop and adapt itself to the extra insulin in the body. Not all diabetics on insulin are obese, but I would attribute this to diet and to demeanor. A person using insulin who is calm, who is not emotional, who eats conscientiously, routinely, and with moderation will probably have excellent blood glucose control and be a thin diabetic. Insulin use and growth may be the reason why the true nature of type 1 diabetes has eluded doctors and researchers for so long.

The main problem I have had using insulin over the years was that I took two shots of NPH insulin a day. This insulin starts to peak 2 - 3 hours after injection; the peak lasts up to 8 hours, and then drops off somewhat but lasts up to 23 hours. By taking two shots a day, the tail end of activity of this insulin overlapped with the next shot of NPH insulin. I also used Regular insulin for meals, which begins to peak 1 hour after injection and lasts 6-8 hours. Because of this overlapping, slight daily changes in timing of when I took the injections, unforeseeable varied physical activity, changing work load, and ever-changing meal menus, it made the whole scheme unpredictable and therefore unmanageable. It also served to conceal the true nature of my condition.

Every single day of my life for the last 21 years has been a guessing game of how much insulin to take. I have learned how to be right most of the time, but when I was wrong, the cure was either to eat when hypoglycemic or to suffer when hyperglycemic. In the first case, I loved to eat so it was easy. Hypoglycemia causes an instinctive knee-jerk reaction to want to eat more than is necessary to correct the problem, resulting in increased blood glucose. I recall having had hypoglycemia often in the first few years of the condition.

I have rarely had severe hypoglycemia (low blood glucose levels) due to insulin reactions but the few that I had have filled me with vigilance and fear about passing out. I intentionally ran my blood glucose on the high side so as to avoid any chance of public embarrassment. Also low blood glucose makes me feel light-headed and so I ended up saying dumb things. I trained myself to bite my tongue when I suspected hypoglycemia. Occasionally, I have said things that I still regret due to hypoglycemia and also to hyperglycemia.

## The Word Game

Insulin Dependent Diabetes Mellitus (IDDM) — Can't control blood glucose without insulin.

Juvenile Diabetes Mellitus — Refers only to the age when it occurs, as opposed to Adult Diabetes or type 2. Most Juvenile Diabetics are considered to be type 1.

Type I Diabetes Mellitus — Older terminology of Type 1.

Type 1 Diabetes Mellitus — Refers to diabetes where the C-Peptide value was negative or of low value (No or low Beta cell function — requiring insulin) differentiating them from C-Peptide positive, which confirms type 2 diabetes.

These four terms have different definitions, which are important, in order to understand how they are viewed. I have throughout my life been termed by each one of these.

They all have come to mean the same thing now. However, the first two are more vague. Because these definitions with different meanings have been lumped together and interpreted to mean the same thing it has led to more confusion and hiding of the truth.

Type 1 means that a C-Peptide test was performed and the conclusion was that there is low or no beta cell function, therefore, requiring insulin.

## C-Peptide and the C-Peptide Test

The second but most important key to understanding diabetes and to understanding blood glucose levels is to first understand the C-Peptide test.

C-Peptide was discovered in 1967. Other names for it include: Insulin C-Peptide, Proinsulin C-Peptide. C stands for “Connecting” as in Connecting Peptide. C-Peptide comes from the production of the insulin molecule in the beta islet cells of the pancreas and can be measured by a blood test. C-Peptide is released in equal amounts to the amount of natural endogenous (internal source) insulin. Bottled insulin (exogenous – external source), I am told, does not contain C-Peptide. The test for C-Peptide has been around for a long time.

It has been my experience that doctors have been more concerned with blood glucose control than with the cause of elevated blood glucose levels. Therefore I conclude that they unscientifically assume that insulin users have no or few Beta cells or beta cell function in their pancreas. Doctors do not use the C-Peptide test, and when they do, they don't understand the results because of what they assume. Diabetics may have only done one C-Peptide test in their lifetime. This test is simply used infrequently. C-Peptide levels change continually in relation to a blood glucose load.

I have noticed two facts that really troubled me.

1. Whenever I look up any information about diabetes, there is no mention of the C-Peptide test, nowhere, not in bookstores, not in the libraries, not on the web, not even in the medical books on diabetes that I have read. I suspect that others would have similar results. What is going on here?
2. Many sources tell diabetics they should eat carbohydrates. This is wrong. So long as a diabetic maintains a normal blood sugar range of 80-120 mg/dl, and a healthy body weight, and gets proper nutrients, the body is being nourished. Why then bombard the system with extra sugar, which will require more insulin? The body is not a limitless dumping ground where anything can be thrown in without consequences.

A heart doctor checks the heart many different ways. A kidney doctor checks the kidney function through many tests. Why then do doctors and endocrinologists blame diabetes on the pancreas, and not check the pancreas?

## Interpreting the Results of the C-Peptide Test

The C-Peptide test measures how much insulin is released from the pancreas, when stimulated by glucose to do so. According to Mosby's Manual of Diagnostic and Laboratory Tests – Second Edition, "The exogenously administered insulin suppresses endogenous insulin production." More accurately, exogenous insulin controls blood glucose concentrations, thus preventing stimulation of endogenous insulin release, which may in effect suppress endogenous insulin production.

The C-Peptide test does not indicate whether the beta islet cells of the pancreas are working or not, nor whether there are in fact beta cells present. C-Peptide is not a test for insulin production, but a test for insulin released from the pancreas.

To understand how the pancreatic beta cells are working one needs to look at the C-Peptide test and at the blood glucose level at the time of test, and consider all the factors that I have previously listed that contribute to hyperglycemia, to arrive at an objective conclusion.

As a blood glucose load increases, the C-Peptide levels increase.

Of course insulin users (type 1) with good blood glucose control are C-Peptide negative or of low value, let me explain:

A child has a cold-like infection and his blood glucose levels increase to astronomical heights (lets say 500 mg/dl). This is considered an emergency situation (because of ketoacidosis). So after a quick finger test in the emergency room the patient is put on insulin (intravenously) according to standard procedures.

The high blood glucose levels, caused by infection, are lowered to normal levels again with exogenous insulin. The child is told not to eat. 6-10 hours pass and finally an endocrinologist comes around to examine the patient, then he orders a C-Peptide test. And of course it comes back negative or have low value: confirming type 1 diabetes.

This is a false conclusion because the exogenous insulin is controlling the blood glucose and the child has not eaten in 6-10 hours so there is no new glucose entering the blood stream through the intestines. The pancreas has no reason to release insulin (measured by the C-Peptide test). Hence, the result is C-Peptide negative or of low value.

Insulin pump users with good blood glucose control will also have these same misinterpreted results.

The only way for insulin using diabetics to prove in spite of poor or of good blood glucose control and also of the Glycohemoglobin or Hemoglobin A1c test (HbA1c - a test for average long term blood glucose, about three months) that they have in fact Type 2 Diabetes which is a curable condition, and not Type 1 Diabetes, is to stimulate the pancreas to release insulin before taking the C-Peptide test.

Increase the blood glucose above normal for at least one hour before taking the blood test.

This should help exculpate the pancreas and prove that maybe the beta cells are there and functioning normally, proving type 2.

I have done the C-Peptide test recently; it was 6.2 nanograms per milliliter (ng/ml). — This is above normal, and proves type 2. My blood glucose at the time of the C-Peptide test was about 230 mg/dl (not intentionally). — I credit this fact for giving me the result that convinced my doctor that I was really a type 2 diabetic.

I was using 70 units of Lantus® 24 hour basal insulin a day and my pancreas was also releasing insulin above the normal range. This was evidence that there was a very heavy glucose load present. Since I was on a no carbohydrate, low calorie diet and my basal insulin was dosed correctly, the heavy glucose load was an unexplainable factor. The sudden need to drop the amount of insulin that I was using right after starting on antibiotics proved that this very heavy glucose load was due in fact to an infection.

The C-Peptide test was done first; the antibiotics were started a week later after the nasal and sinus symptoms came out.

According to Mosby's Manual of Diagnostic and Laboratory Tests - Second Edition  
2002 by Kathleen Deska Pagana, Ph.D., RN & Timothy J. Pagana, MD, FACS - Pages 186-188  
Mosby's Inc. – St. Louis, MO USA  
ISBN 0-323-01609-X

"Fasting range is: 0.78 - 1.89 ng/ml (0.26 - 0.62 nmol/L SI unit)  
Range one hour after a glucose load is: 5.00 -12.00 ng/ml" (During a glucose tolerance test)

According to my lab report, the normal reference range is  
0.6 - 3.2 ng/ml. This reference range may be misleading for doctors because it is incomplete.

I conclude that the complete human C-Peptide range as opposed to the "normal" range is in fact  
0.6 - 12.0 ng/ml, keep in mind that the normal values are for a non-diabetic person.  
The values will be lower in an insulin user and higher in a type 2 oral agent user (agents that promote the  
release of insulin from the pancreas) when blood glucose levels are controlled.

The C-Peptide test is mostly used to diagnose and evaluate patients who are hypoglycemic (produce too  
much insulin) as well as those who have insulinomas (insulin producing tumors). The test is also used to  
see if a normal person is secretly using insulin.

They may ask patients to fast for this test (to reduce glucose levels in the blood) to establish a baseline in  
hypoglycemia cases. This is currently a standard procedure for this test.

In diabetics however the baseline has to be the opposite, to increase glucose levels in the blood, in order  
to prove type 2. Fasting may not yield the intended results. Do not fast.

What I understand from these two sources about my own condition is that if I were normal, non-diabetic,  
these results would mean that my pancreas releases insulin above the normal reference range. However,  
I have a low glucose tolerance threshold. My 6.2 ng/ml C-Peptide falls at the bottom of the 5-12 ng/ml  
glucose test range.

I can only presume that people who can tolerate a high carbohydrate diet would have scored a 12 ng/ml  
on the C-Peptide test for glucose tolerance. Perhaps because they have a higher genetic glucose toler-  
ance (more beta islet cells), and the blood sugars are better controlled under extreme conditions such as  
infection and poor diet, they may in effect have better immunity against disease.

The values of the C-Peptide test and blood glucose separately do not give an absolute picture of what is  
actually happening, they must be considered together. Even when they are considered together they still may  
not present a complete picture of the diabetic condition. Let me add one more factor to the picture, which  
may be relevant: The metabolic rate as controlled by the thyroid gland, which is different in each person.

As I use less exogenous insulin, and more of my own natural insulin I feel a positive change in my mood  
that has been absent for a long time. I attribute this to the fact that more C-peptide is now being released  
along with insulin from my pancreas. Perhaps C-peptide has a physiological effect on mood. Bottled insulin  
does not contain C-Peptide and using it suppresses natural insulin release and probably C-Peptide that is  
released with it. This may explain why I have noticed that my mood was great, even though I had high  
blood glucose at times and a dull mood when having normal glucose levels controlled by bottled insulin.  
The fact that an insulin dependent diabetic feels good after eating a meal may be proof that there is  
endogenous insulin release, because C-Peptide is also being released.

On the subject of diagnostic tests, I found that Mosby's manual contains more complete information about  
the C-Peptide test than other sources that I looked up.

## Bacteria

I am not an expert on bacteria, but several well-known facts about them seem to explain things that I have always been aware of with my condition.

1. Bacteria need oxygen to live. — Their presence would deplete the oxygen supply in the blood and to the brain causing (chronic) fatigue.
2. Bacteria feed on glucose. — Glucose would increase their presence.
3. Bacteria can dissimilate protein molecules by hydrolyzing peptide bonds between amino acids. — Maybe they destroy insulin molecules, regardless of the source of insulin.
4. Bacteria carry out chemical changes. They are used for the production of bottled insulin (synthetic). — They could synthesize glucose or enzymes that help increase glucose concentrations.
5. Bacteria can store compounds in their bodies. Compounds can be released intermittently or when the bacterial bodies die. — Glucose immobilization?
6. Normal body temperature is 98.6 °F (37 °C). The upper limit of most bacterial activity is 99.0 °F (37.2 °C). A decrease in body temperature will increase bacterial activity in the body. When a bacterial infection is present without symptoms, lack of a fever would result in bacteria not being eliminated. — Perhaps further study of the health effects of hot baths and saunas is warranted, as they do raise body temperature.
7. Antibiotics kill bacteria.

I believe that some foods that have a reputation of being beneficial to health simply have anti-bacterial properties.



## Antibiotics

As I have pointed out before, infections increase blood glucose levels drastically. I have noticed through careful observation that my infections are rather chronic and can be present for a long time before they show any major symptoms. I have occasionally had flair-ups of mouth sores, inflammation and joint pain in various parts of my body. I have for a long time suspected latent infections to be the cause. My infections seem to be curable with heavy duty antibiotics, and the antibiotics lower my blood glucose load, thus requiring an immediate drop in the amount of insulin that I use. Overall, I rarely use antibiotics.

Perhaps you have noticed more signs of hypoglycemia than usual when taking antibiotics? In some cases, people are allergic to Penicillin. Could some of these allergic reactions simply be the symptoms of hypoglycemia? Some signs of hypoglycemia include: sweating, dizziness, palpitation, tremor, agitation, hunger, restlessness, headache, seizures, confusion, disorientation, anxiety, fear, death, etc.

I have observed rapid hypoglycemic action when using antibiotics. What I observed was not mentioned on the antibiotics warning labels. The labels did warn of "False positive urine glucose test results on urine test strips." I have observed this phenomenon myself. I have to accept the warning as fact, but it may not be "false" after all. If the antibiotics kill bacteria (which caused the glucose load), and thus rapidly cause hypoglycemia, then where does the glucose load end up? The blood is purged of the excess glucose. Since the blood purge does not occur with insulin by storing it into fat, it may end up in the urine, resulting in a high urine glucose level when using antibiotics.

Only after the diet and healthy lifestyle issues have been fully tackled should antibiotics be considered as the next step to curing diabetes. Antibiotics are already being over prescribed, when a better diet and a healthy lifestyle would be a better prescription for the prevention and curing of disease. There are real dangers to using, misusing, and overusing antibiotics though they may be necessary to cure diabetes, it should be a seldom if not a one time event. Antibiotics should be treated with the utmost respect, and only used when prescribed by qualified medical professionals with consultation and under supervision.

Antibiotics do not kill all of a particular infection, what survives will grow back later (with the proper conditions) and be resistant to antibiotics.

To use antibiotics without permanently correcting the diet and lifestyle issues will only guarantee that the diabetic condition will come back and be much worse later. Uncontrolled blood glucose will bring it back. I have explained all this in the relationship between sugars, carbohydrates, blood glucose, and infections.

## **Fever and Extreme Hyperglycemia**

I have noticed when I was younger, when my blood glucose control was poor, that when I got a cold or infection, I usually got a fever. As I got older and my blood glucose control improved I have noticed that I would get sick but not get a fever. I do not remember having had a fever for at least ten years. In the past year my blood glucose control has been near normal most of the time (this will be explained later). When I am sick I just feel tired and out of it, major symptoms are less pronounced and fevers are not there.

Type 1 diabetics have blood glucose controlled by exogenous insulin and may therefore not experience spikes in blood glucose levels. I speculate that perhaps hyperglycemia is an important natural phenomenon that triggers an immune response and fevers, that non-diabetics would have while they have an infection, without even being aware of it (because they don't check their blood glucose.) Could ketoacidosis have been the likely cause of many deaths attributed to fevers?

## **My Life before Diabetes**

My family moved to the United States when I was 4½ years old. Before this move my diet consisted of small regular balanced meals. I only drank water and unsweetened herbal teas (such as peppermint, chamomile, and lemon verbena) as a child.

I can remember feeling tired and sluggish after eating bread for breakfast. Lunch had no effect (because of physical activity). Dinner always made me feel sluggish (hyperglycemia), but I attributed this to fatigue after a long day. I do note that I would often wake up late at night feeling weak or hungry (hyperglycemia triggering insulin release leading to hypoglycemia).

When we moved to the USA, many things changed. I had to adapt myself to a new world. It was hard for me to cope with this new life. Emotionally it was difficult. I experienced many negative feelings, and there were also turbulence and lack of a stable routine. Things were just changing very quickly, all the time. My parents were working hard to succeed and it was very difficult for them.

The changes in my diet included more and more carbohydrates and sugars such as: bagels with cream cheese, breakfast cereals, and orange juice for breakfast, as well as white bread, peanut butter and jelly sandwiches, pasta, pizza, and cookies. I began drinking sugared soda. All of these were influences from school and popular culture. I did not like drinking tap water because of the chlorine taste. Purchasing bottled water was also not as widespread as it is today. Besides soda had more appeal and I had a sweet tooth. Eventually I was drinking only soda. I was eating more and more bread and cookies. I was eating ice cream. With Halloween came the introduction to junk foods. I spent a lot of time watching television because my parents were busy. I learned English by watching television. And when I watched television I ate. I ate bread and cookies, I ate ice cream, and I drank soda.

In 1981, during summer vacation from school I spent most of my time watching television, eating and drinking soda. As a foreign kid not speaking English, I did not make many friends. I felt compelled to learn English, in order to gain acceptance. For the first few years I was experiencing culture shock. I was also missing my family abroad. I was sad and I withdrew.

During that summer I became more and more thirsty. The weather was very hot and humid; I was not used to this. I was drinking a 2-liter bottle of soda each day to the amazement of my family. In September, I went to my family doctor, he performed a urinalysis in his office, and it was negative. Remember what I said about the relationship between sugar, carbohydrates, and disease.

In December, I became sick with a cold-like infection accompanied by fever and nasal congestion. My mother did not allow me to drink soda, only hot chamomile tea. After a week I began to feel better. Then I began to nag my mother about allowing me to drink soda. She said O.K., but insisted that our family doctor should have a look at me. I began to drink soda again.

My family doctor did a urinalysis in his office and it was positive. He recommended that I go right to the emergency room of the local hospital. He did not prescribe antibiotics as he had done in the past when I was sick, instead deferring my treatment to the experts at the hospital who could better figure out what was wrong with me and determine an appropriate corrective course. Also I was over most of my physical symptoms at the time of examination; it seemed that I was getting better. The rest is stated in the record.

I was still sniffing when I was in the hospital. A nurse noticed that. I remember my head feeling slightly feverish for a brief period. After they gave me the bad news, which I never believed was really that serious, I thought to myself, maybe I was still sick. My family doctor didn't give me pills. Surely I would have preferred taking pills than shots.

I became confident that the problem was my cold and I informed the doctor responsible for my care, saying to him: "What about pills for my cold?" I was trying to draw his attention to the fact that I had a cold and to change his focus to that.

“Oh pills, no your family doctor gave you that”, he replied.

He assumed that I had seen my family doctor just as I was coming down with the cold. The fact is that I always became carsick when I was healthy. So when I came down with the cold and already felt nauseated, there was no way I would get into a car to see the family doctor, that wonderful inhumane car upholstery smell.

I believe that this endocrinologist assumed that because my family doctor had said “virus,” he must have thought that he was observing “beta cell destruction” in progress. This would have been a wrong assumption and unscientific.

Each time I visited the endocrinologists for routine check-ups, I was required to fast and take a blood test the following morning. After the blood test I would go up to the hospital’s pediatric endocrinology – diabetes center. There they would offer me a high carbohydrate breakfast, either bagels or cereal and orange juice right around the time when the “dawn effect” was naturally increasing my blood glucose. Then they would reinforce the fact that I should not eat sugars, as if carbohydrates do not affect blood glucose, and then blame me for my non-compliance. The “take home” message I received was “go ahead, eat your carbohydrates.” Then I would go to school. By noon I would be very hyperglycemic, to the point that I would feel sick. In school I was often taught about the “Food Guide Pyramid”, that emphasizes high carbohydrate consumption. These messages were dishonest and very damaging to me.

## My Life with Diabetes Now

I was using Humulin® N and Humulin® R synthetic insulins. I had switched from beef-pork insulins to synthetic insulins when the former was discontinued in 1999.

I would like to make these observations known about my changeover from beef-pork (animal source) insulin to Humulin® (synthetic) insulin.

1. I used a few units less insulin.
2. On beef-pork insulin I could tell what my blood glucose was within a range of 20-40 (mg/dl) points.
3. Humulin® necessitated more frequent glucose monitoring because:
  - a. Fluctuations in blood glucose were more extreme
  - b. They were less pronounced
4. Overall glucose control was better but fluctuations were worse than on beef-pork.
5. Although I felt better on Humulin®, there was an unexplainable, permanent change in my mood (dulling), as if I had been happier on beef-pork (almost like I was missing it.)

My primary care doctor dismissed all of these observations in 1999. He did suggest referring me to an endocrinology specialist.

In March 2001 I changed insurance plans. I now had a no referral insurance plan that made it easy for me to choose and change doctors. I could now see any specialist without having to go through a primary care physician. I started to see a new endocrinologist because of new hypothyroidism. At that time we discussed diabetes treatment. This doctor was more open-minded and listened to everything I had to say. He dismissed much of what I would present, but he would still listen to me with interest and respect. He promised me better blood sugar control (HbA1c) with optimism. So he became my diabetes doctor. Without him the mystery of my condition could never have been solved.

He recommended that I go on an insulin pump. I was not excited about this for various reasons. A colleague of mine at the Research Foundation who was diabetic told me that he had perfect blood glucose control. This was the first time I had heard of this from a diabetic. He suggested that there were many treatment options available, that my doctor should tell me about, and that it was up to me to choose the best option for me and not the other way around. So I became more open to working with my doctor. Following my colleague's advice I asked my doctor to come up with other alternatives, and he did.

On May 29, 2002, I began using Lantus®, a 24-hour, no-peak basal insulin. Within three days I knew it would change my life and it would help me demonstrate all the unexplainable events in my hyperglycemic condition that the doctors say do not occur.

I was using 60-65 units of Humulin® N insulin a day. When I started on Lantus®, it worked at 30 units a day. I used to use 20-30 units of Humulin® R per meal, but with Lantus® I used only 3-10 units per meal. After ten days I switched to Humalog®, which uses the same dosing scale, but the peak lasts only 3 hours, the tail effect ends 5 hours after injection. It starts to work in 15 minutes.

Before Lantus® I was averaging about 120 units of insulin a day. With Lantus® it dropped to 40-60 units a day. Because of this, I was able to quickly lose a lot of weight with no exercise. I refrained from exercise so I could observe and learn how to use this new insulin. I had normal blood glucose control for the first time in 20 years. Everyone noticed the change in my mood and personality, as if I was a different person.

For the first time since I first began using insulin, I could now skip a meal or even fast. Basically I could now by choice control how much food enters my body because there are no peaks, and no meal planning is necessary. Blood glucose fluctuations were not pronounced at all and I had to monitor my blood glucose frequently, since otherwise I had no idea. On Lantus®, hypoglycemia was now easily corrected with very small amounts of sugar instead of the large amounts.

As I was losing weight, I had to reduce the amount of insulin I was using. I believed it was possible that if I lost enough weight I could walk off insulin, given that I was using so little of it now.

After two months, something began to go wrong. Mysteriously I had to increase the amount of Lantus® that I was using. There were some conceivable explanations such as the honeymoon effect, but by the end of September I was very concerned that something was defective with this new hi-tech insulin product, which I was the guinea pig for. My decision to stay on this product even though it was obvious that the dose was not stabilizing was on a wait-and-see, almost day-to-day basis. I felt normal again after 20 years. I did not want to change that. Many times I considered switching to the pump, but always hesitated. I really believed in this product, Lantus®, I wanted it to work.

I noticed the dose would stabilize for a while, then after having certain feelings I noticed it would begin to climb again over time. Since August, I was feeling tired and low on energy all the time. I thought it was because of the weight loss and the major changes to my body.

By February the dose was 70 units a day. The way that I dose Lantus® is by taking a blood glucose reading before going to sleep, then taking a blood glucose reading first thing in the morning. By comparing the overnight change in blood glucose levels I would change my dose accordingly.

By March I began to feel seriously out of it, I suspected my thyroid condition, so I went to see my endocrinologist to do a TSH (Thyroid Stimulating Hormone) test. He said my blood glucose was about 230 mg/dl and when blood glucose is elevated it is a good time to do the C-Peptide test.

“What is this test?” I asked.

“It’s a test for natural insulin production. C-Peptide is released in equal amounts to natural insulin. Bottled insulin does not contain C-Peptide.” Then he said, out of experience or earlier hints that I had given him:

“Maybe you have some insulin production, let’s see.” I replied:

“Doctor, you are the first person to do this test.” I had no knowledge about this test, because information is not readily available. Even if I did, I would not have dared to ask about it because I have long felt stigmatized about second-guessing a doctor due to my experiences with doctors and the way they treat diabetes.

I said to him that previously I was using 65 units of Humulin® N a day which was similar to using 70 units of Lantus® a day. I said to him:

“It is amazing that Lantus® worked for two months at the lower 30 unit dose.”

The day after the visit, I started to get really congested, I was reading my original diagnosis (1981) and I noticed a pattern with the way I was feeling. I was feeling bad for a month but had no symptoms until the end of March (2003), just as I was sick three weeks before hospitalization (1981) and most symptoms went away, but I still had something, and I remembered that I received no antibiotics. Normally I tough out cold-like infections, but this time I decided to see a doctor about it. So I did, he said that I had a serious infection in my nose and because I was diabetic I should take care of it. I was prescribed antibiotics. I predicted to myself that this would require a drop in insulin use due to hypoglycemia and within 24 hours of starting on antibiotics I had to. Within the first 6 hours of their effect I could already feel myself becoming hypoglycemic.

A week later, my doctor and I spoke over the phone. He said:

“The TSH test came back normal, but we have a little surprise.” I said:

“What is the surprise?”

“Your insulin production is above normal. You are by definition a type 2 diabetic.”

I said: “That is no surprise to me; I knew something was wrong all along. That changes everything doesn’t it?”

He replied, “Yes, we will have to switch you to oral medication.”

Then I added, “I think I have a reasonable explanation of why I became diabetic. I will tell you about it when I see you.”

During the visit I explained and showed him my graph, which I was using to track my treatment and progress on Lantus®.

After two weeks on antibiotics and by fasting, I was able to be off insulin for a day and have normal blood glucose levels. This was short lived because I was still overweight.

In May I was able to cut my insulin use in half and eventually be off insulin for a day, again, when I started using Glucophage® XR, an insulin sensitizer. Strangely, the dose was creeping back up.

In August I went to see an ear, nose, throat doctor who confirmed that I have a chronic sinus infection. He put me on antibiotics.

I now note that I have always had nasal and sinus problems, but never gave it enough consideration. I just lived with it.

Mystery solved.

Whether the goal is to stabilize blood glucose fluctuations or to cure diabetes, the path remains the same.

If there were a term for what I have lived through, I would call it:

Insulin Induced Diabetes - A condition where the symptoms of hyperglycemia are treated without identifying its cause: By either administering exogenous insulin or increasing endogenous insulin release. This results in higher insulin amounts in the body, which result in more blood glucose fluctuations and more difficult blood glucose control. This does not eliminate the cause, and serves to feed infections: creating a vicious circle which results in a more unresolved diabetic hyperglycemic condition due to more resistant disease.

Hyperglycemia is a glucose load that surpasses normal human insulin capacity.

I hope that in the future doctors will view elevated blood glucose, apart from diet, as an early warning symptom of infection.

## The Evidence

1. My original diagnosis. No mention of a C-Peptide test and no mention of antibiotics. I know that I did not receive any. The word "viral" is inaccurate, because it was passed on by phone by my family doctor as a suggestion, which was not actually verified. The actual cause was not determined; it was some sort of a cold-like infection.
2. The graph showing basal insulin daily use in units of Lantus® per day. A high dosage indicates a high blood glucose load (which is controlled). I am on a no carbohydrate, low glycemic, and low calorie diet. And I do not drink, nor smoke, nor do drugs. Antibiotics demonstrate the cause to be infection related.

Look at the pattern of infections (with or without symptoms) and their treatment on the original diagnosis and on the graph.

3. C-Peptide test result.
4. Thyroid tests.
5. My diabetic food pyramid.



January 20, 1982

New York

Dear Doctor : RE: Paul Tubiana

This letter is to inform you about our aspect of the hospitalization of your patient, Paul Tubiana, a 7 year old white male whom you admitted for treatment of new onset juvenile diabetes mellitus.

Paul was admitted on 12/29/81. He was apparently well until September of 1981 when he developed increased thirst and urination. At that time a urinalysis was normal. About 3 weeks prior to admission he developed a viral illness with fever and vomiting which resolved, and in the past week, there has been a marked increase in drinking, eating and frequency of urination.

Since September there has been a 7 lb. weight loss.

On December 29, 1981 a urinalysis revealed glycosuria and ketonuria and he was referred to the

Hospital Emergency Room for evaluation. His blood sugar was 553 and his serum was acetone negative. Arrangements were made for admission.

Family history is positive for adult onset diabetes.

Paul was the 3 kg product of a full term uncomplicated pregnancy and delivery. Milestones were normal,

On physical examination his weight was 29 kg, height 130 cm, pulse 100/minute, blood pressure 110/70 mmHg, respirations 24/minute - no Kussmaul. HEENT - fundi normal, no otitis, pharyngitis, thyroid non-palpable; chest clear; heart normal; abdomen - no masses or hepatosplenomegaly; GU - prepubertal male; neuro - grossly normal.

Doctor  
Page Two

RE: Paul Tubiana

Laboratory Data:

Hgb 13.5; Hct 38.5; WBC 7.7; U/A 4+ glucose, negative protein; Osmo, serum 266; glucose 556; serum pH 7.41, bi 25.

Our impression was, naturally, diabetes mellitus and that Paul presented in a non-acidotic, non-hyperosmolar, not excessively dehydrated hyperglycemic state. We elected to use the I.M. insulin protocol with I.V. and p.o. hydration. Paul received 0.1 u Regular insulin/kg q 1 hour IM x 3 which brought his blood sugar down to 128. At that point he was given a snack and begun on sliding scale.

On Day #4 (1/2/82) he was changed to one shot/day of an NPH and Regular combination. At this time it became apparent that Paul was having considerable difficulty in dealing with the hospital situation and the routines of diabetic care. Also his insulin dose quickly rose to greater than 1 unit/kg/day without evidence of Somogyi phenomenon.

Also on 1/6/82 the dose was split into a.m. and p.m. NPH and Regular doses. At the time of discharge his evaluation was still in progress.

Other studies undertaken in the hospital revealed:

1. normal a.m. cortisol (13.4)
2. normal thyroid functions -  $T_4$  8.2; TSH 4.2;  $T_3$  140
3. normal BUN (7) and creatinine (0.8)
4. no proteinuria

Also, Mrs. Tubiana was instructed by our nutritionist about a low concentrated sugar diet and our nurse clinician taught the use of 2 drop method urine testing and the use of Glucagon for emergencies.

At the time of discharge, Paul's dose was: 20 NPH and 9 Regular in a.m. and 9 NPH and 4 Regular in the p.m. We plan to see Paul in about 3 weeks in our diabetic center.

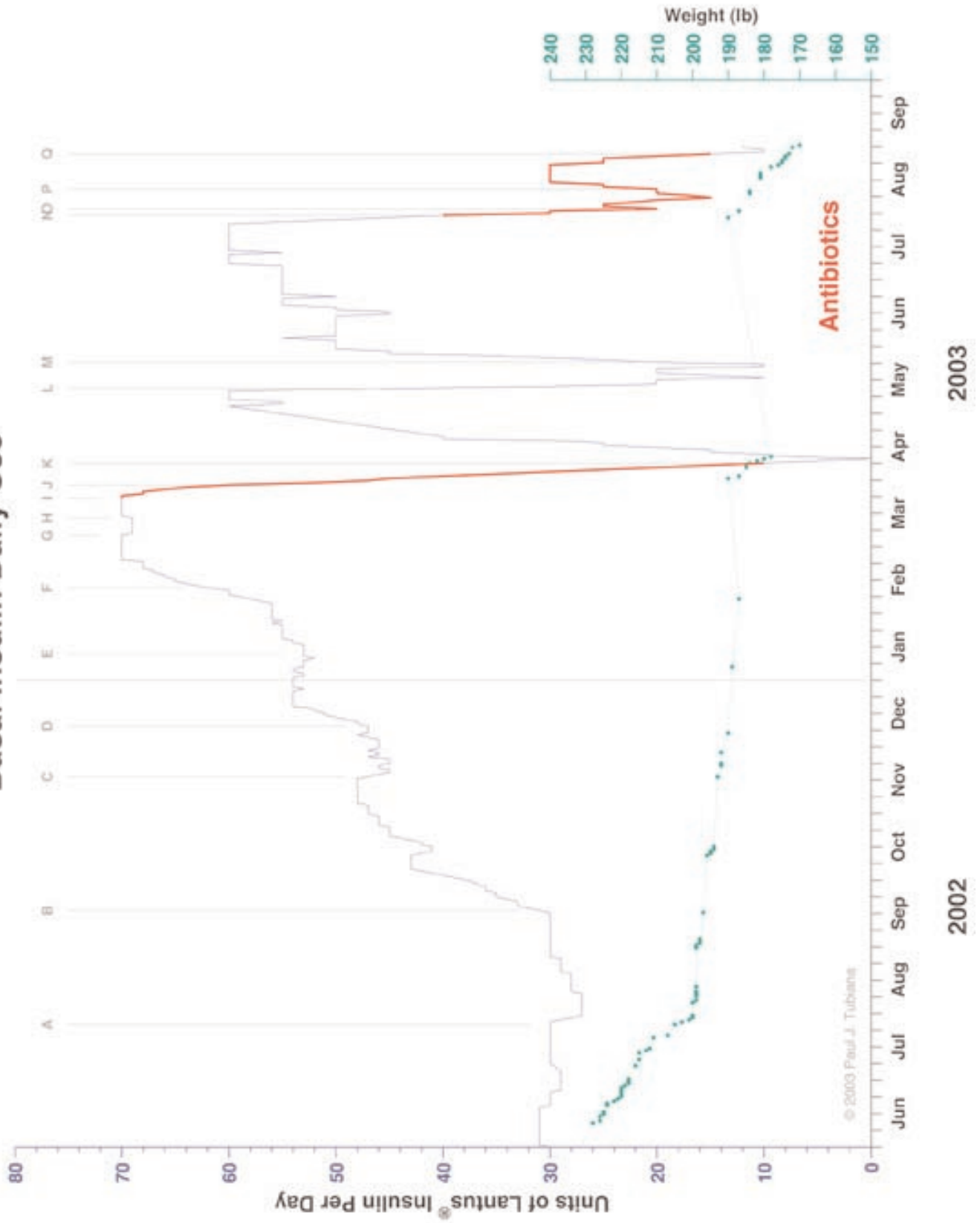
We thank you for allowing us to share in the care of this patient.

Sincerely,

M.D.

M.D.  
Pediatric Endocrinology

# Basal Insulin Daily Use



© 2003 Paul J. Tubiana

## Key to Basal Insulin Daily Use Graph

Basal Insulin Daily Use Graph delineates presence of infection causing glucose load. Lantus® is a 24 hour, once a day basal insulin, with no peaks.

To convert units to hourly basal rate (Insulin pump), divide units by 24.

Units/24 hours	approx. units/hour
10	.416
20	.833
30	1.250
40	1.666
50	2.083
60	2.500
70	2.916

To convert weight in pound (lb) to kilogram (kg), multiply lbs by 0.454. (Lb x .454 = kg)

A – A personal loss.

B – Feelings of disappointment

C – Beginning a new job.

D – Feelings of disappointment. New job did not work out.

E – Beginning of college spring semester. – Stress.

F – Feelings of stress and anger related to specific events at school.

G through H – Spring break

B through F – Chronic fatigue, feeling tired, feeling low on energy.

F through K – Major sinus and nasal symptoms appear.

I – Begin on Zithromax® 250 mg an antibiotic (once a day for 5 days) for nasal/sinus infection.

J – Begin on Amoxicillin 875 mg an antibiotic (twice a day for 10 days)

K – End of antibiotics course.

L – Begin use of Glucophage® XR, an insulin sensitizer.

M – Maximum daily dose of 2000 mg of Glucophage® XR is reached.

N – Begin on Augmentin® 1000 XR an antibiotic (two, twice daily for 4 weeks) for chronic sinus infection.

O – I was soaked by a downpour of rain.

P – I was awake for 30 hours with little rest in order to complete a project.

Q – End of antibiotics course

## Thyroid Tests

TSH and T4 values may indicate presence of infection. Compare with Basal Insulin Daily Use graph. Infections increase TSH value, which subsequently increases T4 value. Body weight and physical activity also affect these values.

Reference range :		TSH	T4	T3	
		.40-5.50 mIU/L	.8-1.8 ng/dL	60-181 ng/dL	
Note	Date				
a	01/20/82	4.2	<b>8.2</b>	140	
b	06/14/86	2.9			
	12/13/86	<b>8.2</b>		<b>340</b>	
c	04/04/87	2.4	<b>6.5</b>		
d	04/17/89	3.6	1.1		
	11/06/98	1.70	<b>6.4</b>		
	12/19/00	<b>5.81</b>	<b>6.7</b>	145	
Hypothyroidism					Synthroid® dose:
e	03/20/01	<b>7.00</b>	1.09		Begin .05 mg (50mcg)
	11/20/01	5.44	1.3		change to .075 mg (75mcg)
	05/23/02	<b>6.34</b>	1.2		change to .1mg (100mcg)
	08/15/02	2.78			
	09/18/02	1.73	1.4	121	
	12/26/02	1.45	1.4		
f (I/J)*	03/19/03	2.48	1.3		
	05/14/03	1.95	1.5		change to .088 mg (88mcg)
g (N)*	08/20/03	1.63	1.5		change to .075 mg (75 mcg)

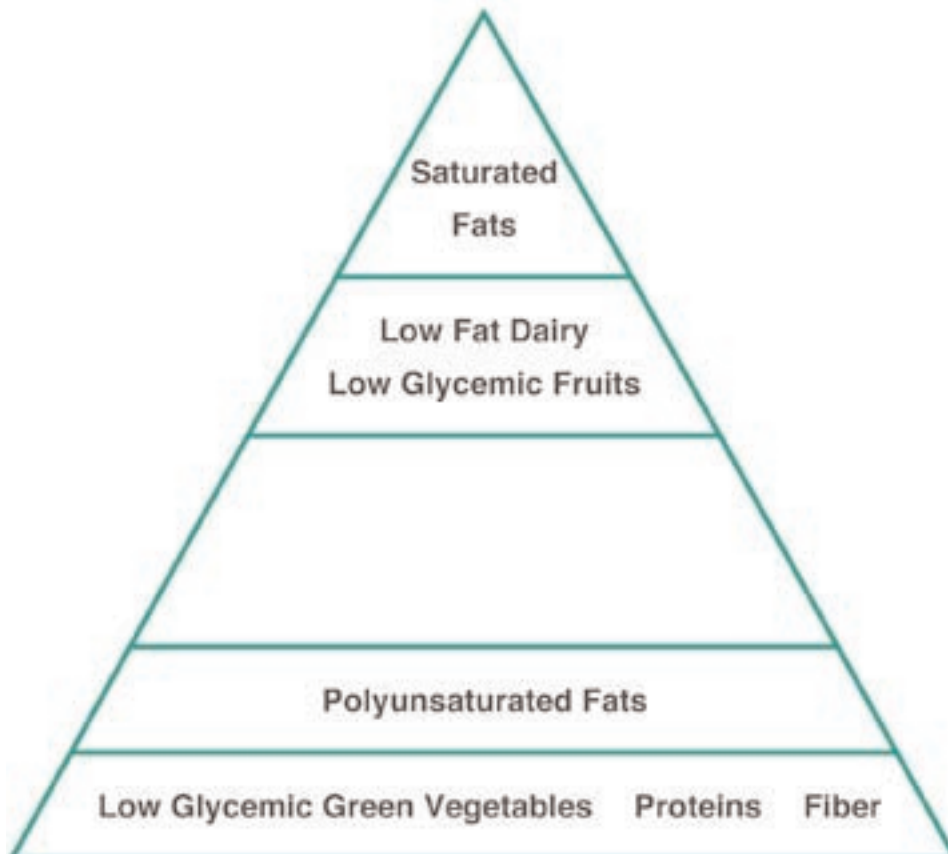
### Notes on infections

a	12/08/81	Cold-like/fever infection	No Treatment
b	06/12/86	Tonsillitis/strep throat	Treated with Erythromycin
	07/23/86	Eye irritation	
	07/23/86	Chicken pox	
c	11/13/87	Tonsillitis	
d	01/23/89	Flu/fever/cough	Treated with Amoxicillin 500mg
e	03/08/01	Ear infection	Treated with Augmentin® 500mg
f (I)*	03/25/03	Sinus/nasal infection	Treated with Zithromax® 250mg
(J)*	03/31/03	Sinus/nasal infection	Treated with Amoxicillin 875mg
g (N)*	07/30/03	Sinus/nasal infection	Treated with Augmentin® 1000 XR

Other infections occurred but are not documented.

\*Letters in parentheses refer to letters on graph.

## My Diabetic Food Pyramid



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