

HOW TO GET THE RIGHT MEAL TIME INSULIN DOSE WITH TYPE 1 DIABETES



IMAGINE IF YOU COULD CHOSE AN INSULIN DOSING STRATEGY THAT
WAS PERFECT FOR THE FOOD YOU WANT TO EAT RATHER THAN
CHANGING YOUR FOOD CHOICES TO SUIT YOUR INSULIN

DREAM NO LONGER — DISCOVER **HOW TO MOVE PAST CARB COUNTING**
WITHIN THESE PAGES

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ABBREVIATIONS

AA = Amino Acid
ADP = Adenosine Di Phosphate
ATP = Adenosine Tri Phosphate
AUC = Area Under the Curve
BCAA = Branches Chain Amino Acids
Ca = Calcium
CGM = Continuous Glucose Monitoring
FFA = Free Fatty Acid
FPU - Fat and Protein Units
GDH = Glutamate Dehydrogenase
GI = Glycaemic Index
GL = Glycaemic Load
GLP-1 = Glucagon-like peptide-1
GIP = Gastric Inhibitory Polypeptide
GPCR = G-Protein Coupled Receptor
I:G = Insulin to Glucagon ratio
ICR = Insulin to carbohydrate ratio
MDI = Multiple Daily Injections
MUFA = Monounsaturated Fatty Acids
KATP = ATP-regulated potassium channel
mTOR = mammalian target of rapamycin
SFA = Saturated Fatty Acid
TCA = Tri-Carboxyl-Acid cycle

INTRODUCTION

After being diagnosed with Type 1 diabetes, your first job is to get over the initial shock, because a month ago you felt in great shape.

You're then told your B-Cells have been attacked by your own immune system. Nobody knows exactly why, but your pancreas will never produce sufficient insulin again.

Then the big bomb. You need to inject or pump insulin into your body. Not just that, but in the exact amounts your B-Cells would have delivered, if they were working effectively!

Why?

To keep your blood glucose level between 3.5 - 7.0mmol/l (65 - 120mg/dl), so that you stay healthy now, and in the future.

Then it dawns on you:

"To deliver the right amount of insulin, my decisions must match that of my B-Cells."

This realisation seems daunting at first, but then you get a lot of education (hopefully) to help you make those decisions.

Education such as:

- Carbohydrate counting
- How to use an insulin to carbohydrate ratio (ICR)
- Exercise management
- Maybe some information on Glycaemic Index (GI)
- Maybe some information on meal planning

The education does the trick. Your blood glucose levels stay in target, and you are super motivated. Then Boom, reality hits! The remaining B-cells are killed off, and your motivation waivers. The honeymoon is over, and you start to notice a few things:

- Carbohydrate counting does not always work. Despite counting to the gram, your blood glucose goes really high when eating:
 - Pizza
 - Cheesy pasta dishes
 - Traditional English fry ups (toast, sausage, beans, bacon, egg)
 - Takeaways and fast foods meals
 - Three course meals with a big tasty dessert at the end
- When you have salad with a large protein source, your blood glucose level goes high despite having no carbohydrate on the plate.
- When you have carbohydrate only meals, such as fruit, you go hypo

What do you do next?

Ask your diabetes team, Dr. Google, or the diabetes online community?

A mixture of all three most likely. This leads to a variety of different answers and solutions. Some of these sound familiar?

- You must not be counting your carbs correctly, get weighing again. Most often from your Diabetes Team. This is not disrespecting diabetes teams, as I am diabetes dietitian by trade, and I USED to say that all the time!
- You need to count every gram of protein and fat and adjust insulin according. Have you not heard of the Warsaw method?
- You need to match your insulin delivery to the insulin demand for the whole meal. Have you not heard of the Food Insulin Index (FII) and Food Insulin Demand (FID) system?
- Take the low carbohydrate approach, which comes in different guises:
 - Cut your carbohydrate to a lower amount, 50-200g per day, and up your protein and fat.
 - The Dr Bernstein approach. Cut your carbs right down to under 50g (ideally no more than 24g), up the protein considerably, and have moderate fat.
 - Stop being a sugar burner, become a fat burner, **Keto is the only way!** Cut carbs to less than 50g per day, small to moderate protein, and fat should make up 70-80% of total energy intake.

What advice should you take?

This is the million dollar question. Unfortunately there is no simple answer that applies to everyone.

Some questions that are good to ask yourself;

- What impact will a certain type of diet have on my overall health in the long-term, even if it does improve my diabetes control?
- Does the diet provide my body with all the vitamins and minerals it needs to sustain my overall health?
- Can I honestly see myself sticking to the suggested food choices?
- Can I follow this when I socialise with friends?

- What insulin dosing strategy do I need use to get in target blood glucose levels with this type of diet?
- If I choose one diet today, does that mean I need to follow that for the rest of my life?
- Can I switch between different types of nutritional intake from meal to meal, day to day, week to week?

Today there is a wealth of information on the internet, and no shortage of people telling you there is only one way, THEIR WAY.

Some of the approaches are first class, some are money making scams, and others are promoted by people who have had enormous personal success, and think everyone should follow their approach.

The challenge is deciphering the Wheat from the Chaff.

I posed the above questions to myself nearly ten years ago. This led me to trying all approaches to see what works. It was a fun and challenging journey, but looking back, I realise I missed the most important questions:

- How much insulin do the B-Cells release after eating different combinations and amounts of carbohydrate, fat and protein?
- How much insulin is needed after eating for key metabolic processes, such as growth and repair?
- How much insulin is needed after eating to balance the effect of Glucagon, which is released from the A-Cells?
- Does it matter that I inject insulin, rather than having it delivered direct from the B-Cells?
- Are there any other hormones released from the B-Cells that need to be considered?

Answering these questions gave me the ability to match what my B-Cells would have done, if they were fully operational. If you invest the time to gain this knowledge, you can be very flexible with your dietary approach, whilst still achieving first class diabetes control.

Within these pages I have condensed ten years of research, clinical practice and experimentation to document:

- How the pancreas behaves after eating to keep blood glucose levels in target.
- What is different for people with Type 1 Diabetes.
- How the gap can be bridged with novel insulin dosing strategies.

Once you're comfortable with this, we will move on to critique the commonly used insulin dosing strategies.

Finally and most importantly, we will discuss which insulin dosing strategies best fit the different nutritional approaches. **This flips the current way of thinking on its head.** The traditional approach is;

“Here is your insulin dosing strategy, carbohydrate counting and an ICR, now fit your diet around it.”

Imagine if it was the other way round;

“Choose the diet that best suits you and your goals, then select an insulin dosing strategy that matches it.”

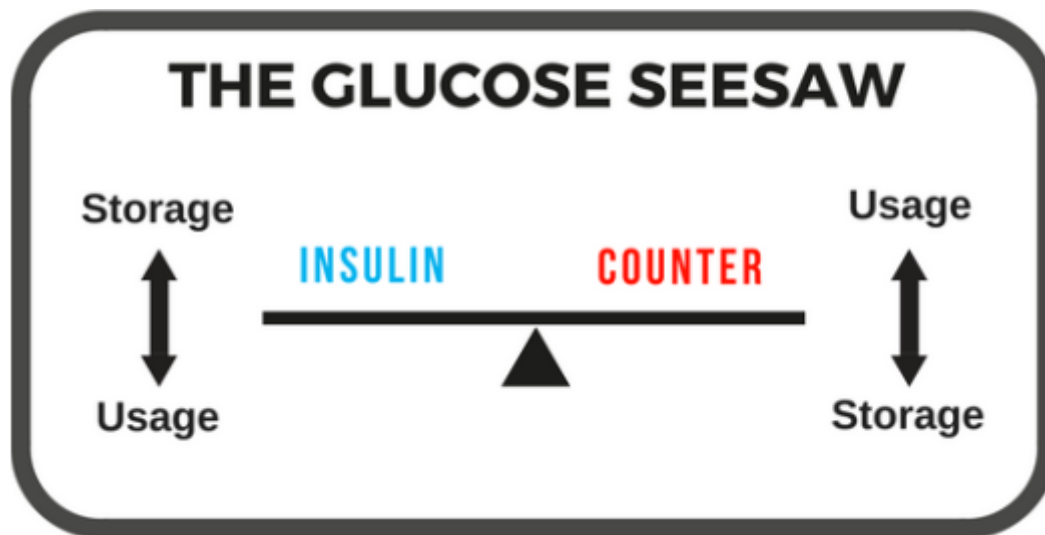
Before we get stuck in, I have to tell you that what you are about to read is a summary version of a much more detailed report. The complete 160 page report that has over 80 references details the physiology, biochemistry and has in-depth critique of the different dosing strategies.

As you progress through this summary you may want to go deeper into a particular section, to get a comprehensive understanding. To make this possible, the full report has been broken down into easily digestible chunks. At the end of each section of this summary, there is a link to the corresponding full report section that goes much deeper. So if you want to go deep into the biochemistry and physiology of the insulin to glucagon ratio, just follow the link at the end of that section. But if critically evaluating the research evidence on the currently available insulin dosing strategies is your bag, just follow the link at the end of that section.

I hope you enjoy.

THE INSULIN TO GLUCAGON RATIO (I:G)

After eating, your blood glucose level is mainly determined by two hormones that are secreted by the pancreas, Insulin and Glucagon. An easy way to remember the basic principle is to think of a Seesaw. When your **INSULIN** is high and glucagon (glucagon is one of the **COUNTER**-regulatory hormones, along with Cortisol and adrenaline) low, you have a high I:G, and your body is in energy storage mode. When your I:G is low, your body is in energy usage mode.



Our main focus is discovering what happens when the I:G changes, and understanding the blood glucose consequences .

Let's get started with Insulin.

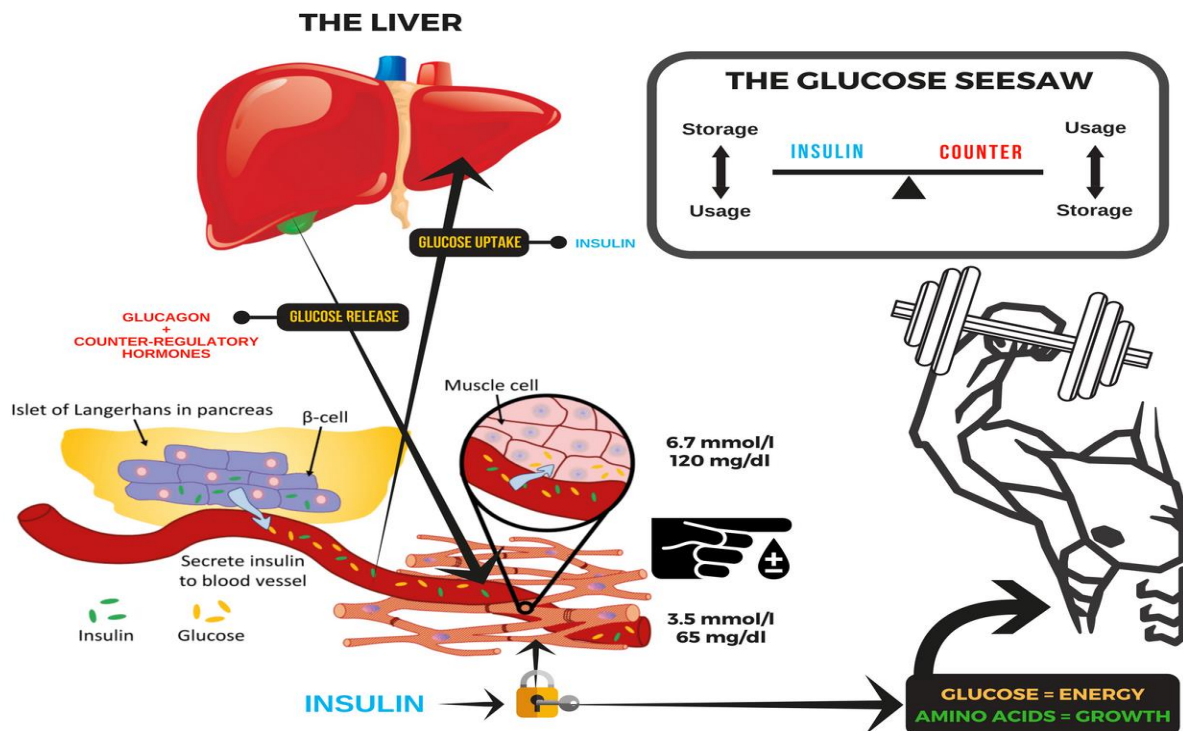
INSULIN

Insulin is the main hormone secreted from the B-Cells of the pancreas. Insulin has an enormous number of direct and indirect effects on metabolism. We shall focus our discussion on the main ones of:

- **Anabolism:** using AA (Amino Acids) for the synthesis of more complex molecules, mostly for growth and repair.
- **Energy storage:**
 - Glucose stored as Glycogen by a process called glycogenesis
 - Fatty Acids (FFAs) stored as Fat.
- **Energy production:** the movement of Glucose into cells to be used burned as fuel by a process called glycolysis.

Insulin moves glucose and AA from the blood into the cells of the body. Consider insulin as a key that unlocks the body's cell doors to allow glucose and AA to enter, a bit like a

bouncer on a club door. See the below diagram for an overview.

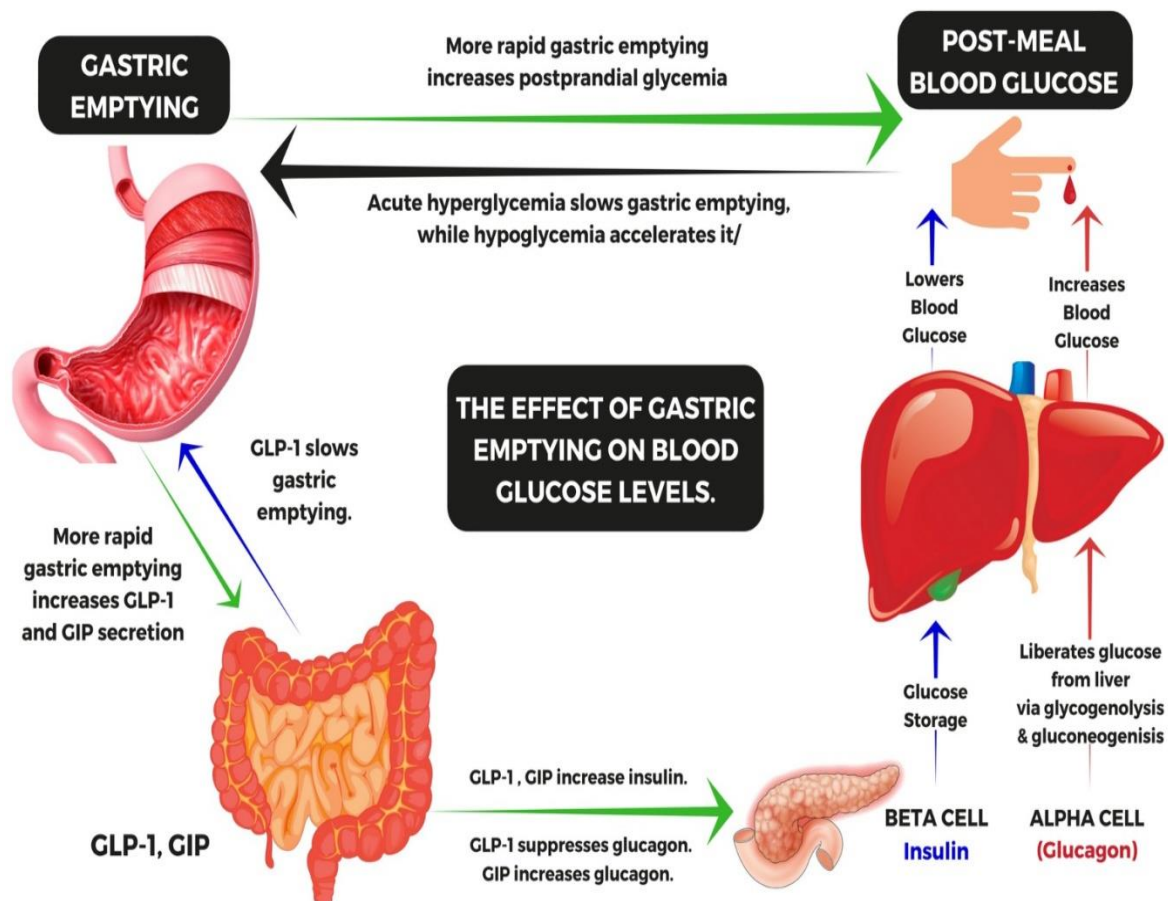


THE REGULATION OF INSULIN SECRETION

There are multiple regulators of insulin secretion from the B-Cells. The key ones stimulating insulin secretion by positive regulation are:

- High energy state of the B-Cell from increasing levels of energy substrates
 - Glucose has the strongest effect
 - AA have a medium effect
 - FFA have a weak effect
- GLP-1 (Glucagon-Like-Peptide-1)

The diagram below shows that GLP-1 plays a major role in the speed of stomach emptying. It also highlights GLP1's positive regulation on the B-Cells to stimulate insulin secretion. The diagram highlights that GIP (Gastric Inhibitory Polypeptide) mildly stimulates insulin secretion, but that it's major role is increasing Glucagon secretion from the A-Cells. We will discuss this in a later section



When the B-Cells no longer function properly, as happens with Type 1 Diabetes, all these signals are lost. Therefore the job of a person with Type 1 Diabetes is to match insulin delivery to what the B-Cells would have secreted in response to the meal composition, so that:

- Glucose can be transported from the blood into the cells for usage via glycolysis, and storage as glycogen.
- AA can be transported into cells for protein synthesis, enzyme re-generation, and to provide substrate for glucose metabolism.

GLUCAGON

Glucagon is the main hormone secreted from the A-Cells of the pancreas. It has many functions in catabolism, the breaking down of more complex molecules, which include:

- Glucose liberation from glycogen stored in the liver and muscles, called glycogenolysis
- Glucose creation in the liver from AA, called gluconeogenesis
- FFA liberation from stored fat supplies

THE REGULATION OF GLUCAGON SECRETION

There are multiple regulators of glucagon secretion from the A-Cells. The key ones stimulating secretion by positive regulation are:

- GIP
- Central Nervous System by releasing adrenaline in response to a falling glucose level.

The key ones inhibiting secretion by negative regulation are:

- Insulin
- GLP-1

When the B-Cells no longer function, as happens with Type 1 Diabetes, the negative regulation signals are lost. Therefore the job of a person with Type 1 Diabetes is to match insulin delivery to what the B-Cells would have secreted in response to the meal composition, so that:

- Glucagon can be suppressed from rising too high after eating high protein and fat meals. If this does not happen Glucagon will increase liver output of glucose too much, causing hyperglycaemia.
- Glucagon is not suppressed too much by delivering a very large bolus of insulin. If this happens glucagon will not be released to prevent hypoglycaemia.

INSULIN AND GLUCAGON SUMMARY

Let's simplify this. Insulin is the **big brother and master** in this hormonal relationship. Insulin trumps the action of other hormones when it comes to regulating Glucagon secretion. A high I:G will prevent glycogenolysis and gluconeogenesis, whilst simultaneously increasing glycolysis, glycogenesis and anabolism (4).

This works beautifully when the B-Cells of the pancreas are in full working order, and the adjustments are automatic. However, when you have Type 1 Diabetes, insulin secretion is not automatic, you must learn to be the master controller!

So it's a big responsibility for the person with type 1 diabetes to use this trump card at the right time, and in the right amounts!

To go deeper into the physiology and biochemistry of the I:G, access that section of the full report.

We have now built a foundational knowledge of the functions of insulin and Glucagon,

and understand their key regulators .Let's move onto seeing how this hormonal relationship works after eating different types of meals. This will provide the model that people with Type 1 diabetes need to follow when delivering insulin for their meals.

To gain a full understanding we must ask very specific questions. What does the pancreas do to maintain blood glucose level, and what is different for the person with Type 1 Diabetes;

- When carbohydrate only is consumed?
- When protein only is consumed?
- When fat only is consumed?
- When carbs and protein are consumed?
- When carbs and fat are consumed?
- When fat and protein are consumed?
- When carbohydrate, fat and protein are consumed?

Here is where the real fun begins!

WHAT DOES THE PANCREAS DO WHEN CARBOHYDRATE ONLY IS CONSUMED?

INSULIN

Put simply, the B-Cells secrete extra insulin to move the glucose from digested carbohydrate into the liver for storage as glycogen. Glucose not stored as liver glycogen enters the circulation, and insulin moves the glucose from the blood into body's cells for energy production and storage as glycogen.

GLUCAGON

Put simply, the A-Cells reduce secretion of Glucagon when carbohydrate only is consumed. The I:G increases ensuring the body moves into energy storage mode.

This point is essential to consider for a person with type 1 diabetes.

Why?

People with type 1 diabetes do not have the automatic negative regulation of the A-Cell that is provided by B-Cell secretion of insulin. They have to deliver insulin in the correct amount to not only move glucose and AA into cells, but also to elicit the inhibition of Glucagon secretion.

Consider this situation;

Not enough, or no insulin is delivered for a carbohydrate only meal, such as fruit or sweets. There is a double effect;

- Not enough insulin to move glucose from the blood into the cells, therefore the blood glucose level rises.
- Not enough insulin to negatively regulate A-Cells to stop glucagon production. Glucagon slightly increases, liver glycogenolysis increases, blood glucose increases further.

SUMMARY

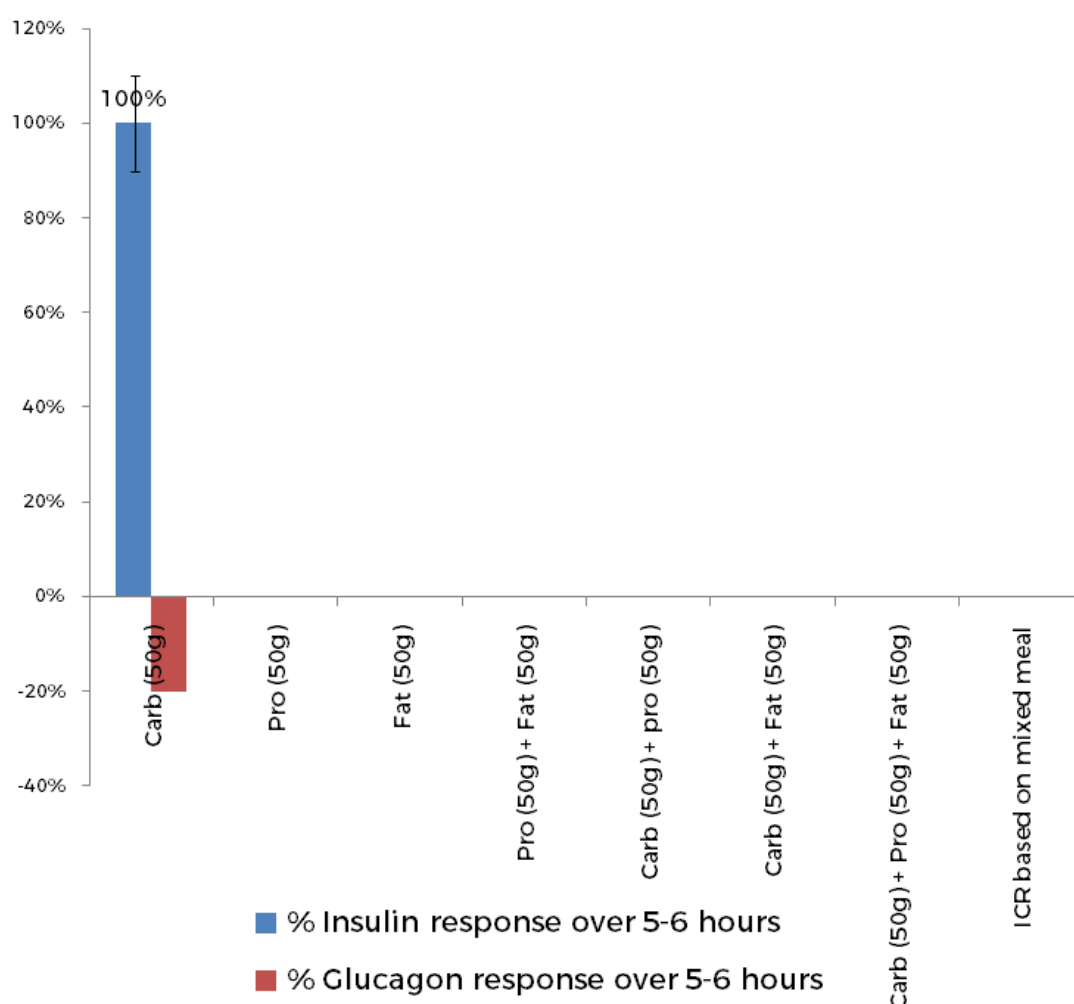
Very simply, when carbohydrate alone is consumed the I:G ratio increases putting the body into storage mode. This is regulated by an increase in GLP-1 and glucose stimulating insulin secretion, and the consequent direct negative regulation of Glucagon release. The job of the person with Type 1 Diabetes is to match the amount of insulin the B-Cells would release for carbohydrate only. Too little leading to hyperglycaemia, too

much leading to hypoglycaemia.

From this point forward, I want you to consider the insulin response to 50g of carbohydrate alone as 100% over 5-6 hours. Also consider the Glucagon response as a reduction on 20% over 5-6 hours.

The below graph will be built upon to model the insulin response required for the macronutrients singularly, and in combination. This will help put the insulin and glucagon response into perspective, and identify the challenge a person with type 1 diabetes has to match it. This graph is not perfectly scaled, therefore it is not 100% factually correct, but it will prove very valuable.

The insulin bar on the graph has a 10% error bar. This represents the difference in response based on the glycaemic index (GI). Lots of type 1 diabetes studies have shown that low GI carbohydrates require about 10-20% less insulin than high GI carbohydrates, even though the exact same amount of carbohydrate was consumed (1,2).



To go deeper into the physiology, biochemistry and research for carbohydrate only, access that section of the full report.

WHAT DOES THE PANCREAS DO WHEN PROTEIN ONLY IS CONSUMED?

INSULIN

Put simply, the B-Cells secrete extra insulin into the blood to move the AA from digested protein into the body's cells for anabolism. The increase in insulin also moves glucose from the blood into the cells for usage and storage as glycogen.

The important point to consider for people with Type 1 Diabetes is that the GLP-1 increase will not lead to insulin secretion. Therefore the missing insulin secretion must be matched by the person with Type 1 Diabetes, by delivering the exact amount of insulin required for the protein consumed.

GLUCAGON

Put simply, the A-Cells secrete extra Glucagon into the blood to prevent hypoglycaemia from the concurrent increase in insulin.

It has been shown clearly that an increase in blood AA increases Glucagon secretion, but when this is not accompanied by an increase in insulin, the body moves into a catabolic state rather than the anabolic state (3).

People with Type 1 Diabetes do not have this automatic secretion of insulin from GLP-1 positive regulation when they consume protein. Therefore they have to balance delivering just enough insulin to allow anabolic activity, whilst not delivering too much causing hypoglycaemia.

Consider this situation:

Not enough, or no insulin is delivered for protein only meals, you get a double whammy;

- Not enough insulin to move AA into the cells for anabolism. You miss out on growth and repair.
- Not enough insulin to A-Cells to stop glucagon secretion. Glucagon increases liver glycogenolysis leading to a steady and persistent rise in glucose after eating protein with no insulin.

RESEARCH

In non-diabetic subjects, ingestion of 50 g of protein, such as lean beef, elicited a significant increase in insulin over four hours (4). The amount required was 28% of that required for 50g of pure glucose (4). Similarly Berger found that ingestion of 50-100g protein elicited 21% of the insulin response of 100g glucose (5). Taken together this suggests the insulin requirement for 50 grams of protein is 20-30% of the requirement needed for 50g glucose.

So what does this research mean for people with Type 1 Diabetes, should they give insulin for protein only?

Put simply there are two very good reasons for delivering insulin for pure protein meals:

- First, to ensure the AA's are used for anabolic activity.
- Second, to prevent glucagon from slowly increasing liver glycogenolysis and Gluconeogenesis and raising the blood glucose level.

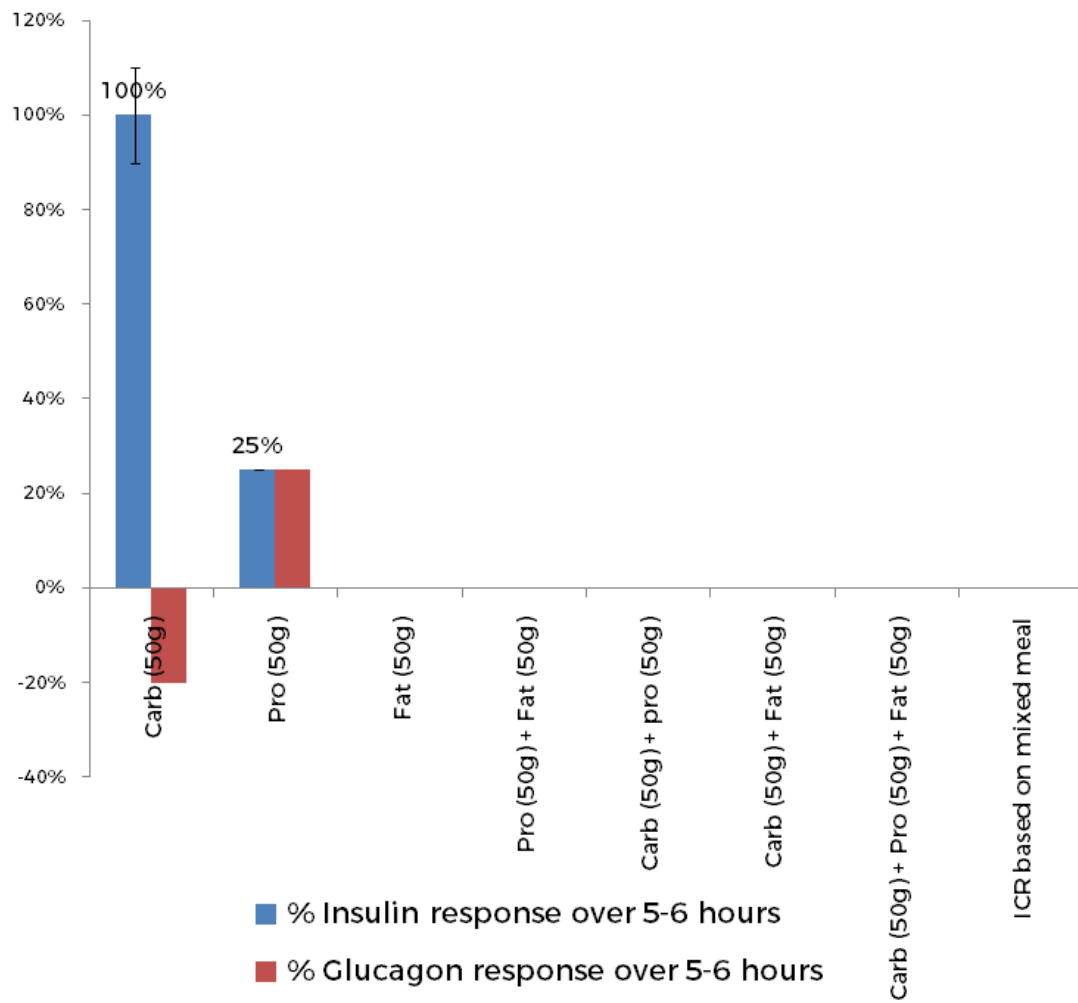
SUMMARY

Very simply, when protein alone is consumed at 50g, both Insulin and glucagon increase, therefore not altering the I:G. This allows AA to be used for anabolism whilst maintaining the glucose level. The increased GLP-1 stimulates insulin secretion, whilst the increased GIP and central nervous system activity stimulate glucagon secretion.

The job of the person with Type 1 Diabetes is to match the amount of insulin the B-Cells would release for protein only. Too little leading to catabolism and hyperglycaemia, too much leading to hypoglycaemia.

HOW MUCH INSULIN TO DELIVER FOR PROTEIN ON A GRAM BASIS?

Not an easy question to answer, but from the research above it would seem sensible to suggest that protein when ingested alone, elicits about 25% of the insulin response needed for carbohydrate alone. I have shown this graphically below in comparison to carbohydrate alone, and have included the equal increase in Glucagon response.



To go deeper into the physiology, biochemistry and research for protein only, access that section of the full report.

WHAT DOES THE PANCREAS DO WHEN FAT ONLY IS CONSUMED?

INSULIN

Put simply, the B-Cells secrete a little extra insulin into the blood to promote storage of FFA in the fat tissue.

GLUCAGON

Put simply, the A-Cells secrete a little extra glucagon into the blood to promote FFA oxidation and glycogenolysis.

People with Type 1 Diabetes do not have this automatic secretion of insulin from the B-Cells in response to GLP-1 when they consume fat. So consider this situation, it may explain a few things:

- If you have not provided enough, or any insulin for a fat only meal, the unchecked Glucagon will mildly elevate liver glycogenolysis and Gluconeogenesis. This could lead to a late rise in glucose after eating fat only meals.

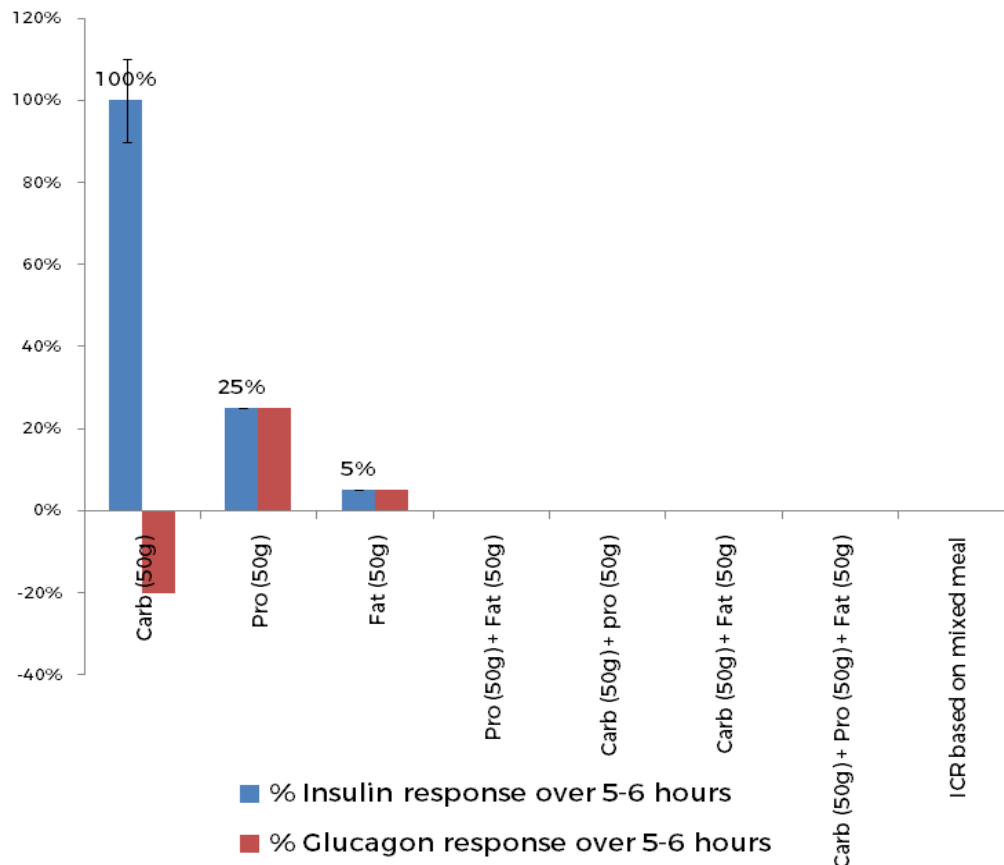
SUMMARY

Very simply, when fat alone is consumed at 50g, both Insulin and glucagon slightly increase, therefore not altering the I:G. This allows insulin to promote FFA to be stored as fat, which is positively regulated by an increase in GLP-1. This allows glucagon to increase FFA oxidation and liver glycogenolysis for energy production, which is positively regulated by GIP.

The job of the person with Type 1 Diabetes is to match the amount of insulin the pancreas would release for fat only. Too little leading to potential slight hyperglycaemia, too much leading to hypoglycaemia.

HOW MUCH INSULIN TO DELIVER FOR FAT ON A GRAM BASIS?

There is little research on the insulin requirement for fat alone over 5-6 hours for people with Type 1 Diabetes. Therefore we can use the studies comparing 560kcal of fat and protein in non-diabetic populations (6). The insulin and glucagon response to fat was only 20% of the response of protein. Considering protein alone has an estimated 25%insulin response compared to carbohydrate alone, this puts the insulin and Glucagon response for fat compared to carbohydrate at 5%.



To go deeper into the physiology, biochemistry and research for fat only, access that section of the full report.

SUMMARY OF PANCREATIC RESPONSE TO SINGLE MACRONUTRIENTS?

You now have a good understanding of what happens when the macronutrients are consumed individually. But rarely do we eat these macronutrients on their own, we usually eat mixed meals.

It would be nice if insulin requirement was just simply the addition of the individual macronutrient insulin requirements. For example if someone had a 1unit to 10g ICR:

- Carbohydrate at 100g, count 100% = 10units
- Protein at 10g, count 25% = 2.5 units
- Fat at 10g, count 5% = 0.5 units
- Then a meal of 100g carbohydrate, 10 grams protein and 10g fat would obviously need 13 units of insulin! Simple!

If only it was that simple! Unfortunately it is not. We need to look at what happens in real life, when the macronutrients are consumed together.

WHAT DOES THE PANCREAS DO WHEN PROTEIN AND FAT ARE CONSUMED?

INSULIN

Put simply, the B-Cells secrete extra insulin to move AA into the cells for anabolism, and promote FFA storage.

The big difference here is that the increased insulin requirement is not as immediate, it is more gradual over time. The stomach empties at a constant energy rate, therefore because fat is very energy dense, gastric emptying and consequent insulin signalling is delayed(7).

GLUCAGON

Put simply, the A-Cells secrete extra glucagon to prevent hypoglycaemia induced by increased insulin, through liver glycogenolysis, and later to stimulate FFA oxidation and gluconeogenesis.

For the person with Type 1 Diabetes, if no insulin is administered for a fat and protein meal, the I:G will drop leading to liver glycogenolysis and Gluconeogenesis. Also the AA's will not be used for anabolism.

RESEARCH

In 2010 Utoff and colleagues assessed the effect of three carb free meals with no bolus insulin on ten males with Type 1 Diabetes. The three carb free meals all consisted of less than 3g carbohydrate, 32-34g protein and 48-52g fat, 580-612kcal (8). They first did a fasting test to ensure the background insulin was set appropriately. The glucose level stayed stable over the four hour fasting test (7.2 to 6.8mmol/l), suggesting adequate background insulin.

For each of the three carb free meals, there was a consistent glucose rise of more than 3mmol/l over four hours. The researchers showed the glucose would have very likely kept rising if the study continued to measure past four hours, possibly up to eight hours. This is supported by the consistent observation of the late post-prandial hyperglycaemia effect of both fat and protein in type 1 diabetes (9,10).

SUMMARY

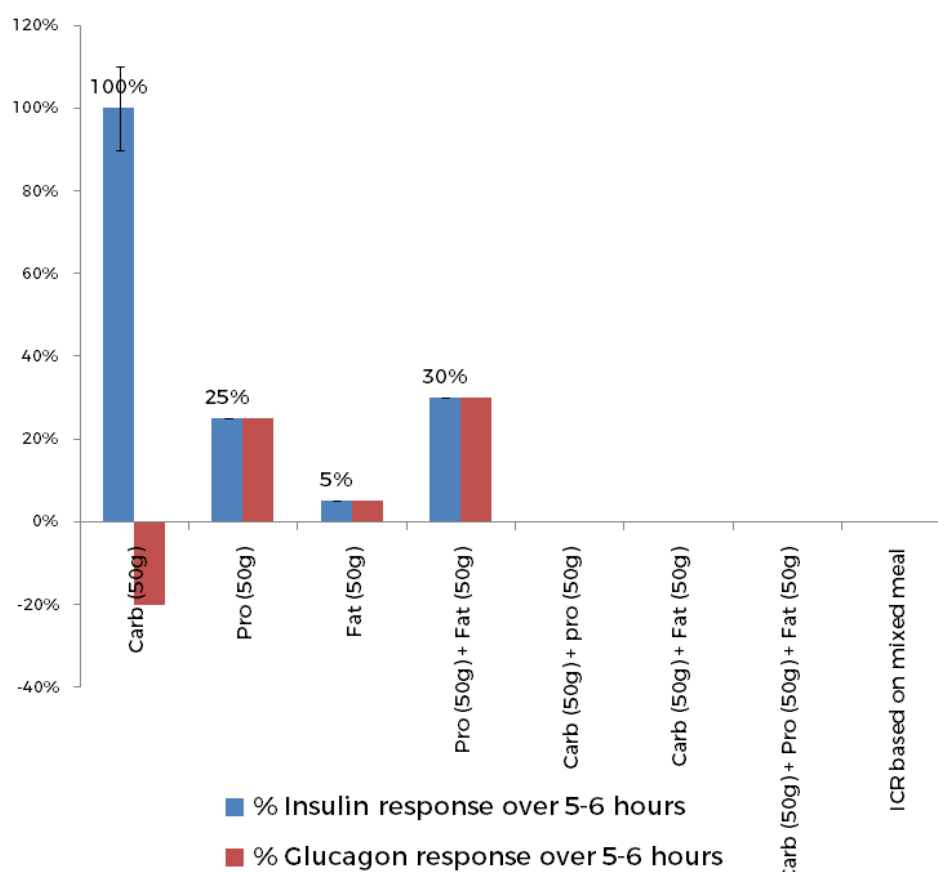
Very simply, when protein and fat are together both insulin and glucagon increase, therefore not altering the I:G. This allows AA to be used for anabolism, FFA to be stored

as fat or used for oxidation, all whilst maintaining the glucose levels. This is regulated by an increase in GLP-1 stimulating insulin secretion, and an increase in GIP stimulating Glucagon secretion.

The job of the person with Type 1 Diabetes is to match the amount of insulin the pancreas would release for protein and fat combinations. Too little leading to catabolism and hyperglycaemia, too much leading to hypoglycaemia.

HOW MUCH INSULIN TO DELIVER FOR PROTEIN AND FAT ON A GRAM BASIS?

Taken together, the two studies discussed, strongly suggest protein and fat together require insulin. It would seem protein exerts the majority of the insulin response initially, with fat becoming more prevalent later. The graphic includes an expected response of 30% for both insulin and Glucagon.



To go deeper into the physiology, biochemistry and research for protein and fat, to access that section of the full report.

WHAT DOES THE PANCREAS DO WHEN CARBOHYDRATE AND PROTEIN ARE CONSUMED?

INSULIN

Put simply, the B-Cells secrete extra insulin to move AA and glucose into the cells for anabolism and storage. More insulin is secreted than you would expect from their individual insulin secreting effects.

GLUCAGON

The A-Cells also increase Glucagon secretion, which is the opposite to the decrease caused by carbohydrate alone. The same signals that were discussed above are in play here for Glucagon. But one of the key factors to be mindful of here is:

- A. Insulin is the trump card! Although the GIP increases A-Cells secretion of Glucagon, the negative regulation from the high level of insulin dampens the glucagon rise compared to the protein only.

If this increase requirement is not met by the person with Type 1 Diabetes, what are the consequences?

- B. Lack of insulin to move the necessary glucose from the blood into the cells.
- C. The negative regulation of insulin on the A-Cells will not be as high as it should be. Therefore higher glucagon levels will increase liver glucose output, especially 3-4 hours after eating when the administered insulin is wearing off.

RESEARCH

In healthy subjects, ingestion of 50g of protein, such as lean beef, and 50g of pure glucose elicited a significant increase in insulin over four hours (4). The amount required was 127% of that required for 50g of pure glucose over four hours (4). Another study compared the insulin response when adding different protein sources (milk, cod, whey, and others) to white bread (100%). They found the increase in insulin response varied from 124-190% extra. Another study found the addition of protein to carbohydrate elicited 166% of the insulin response compared to carbohydrate alone (100%) (11).

This evidence is strongly suggestive that when protein is added to carbohydrate, the increase in insulin requirement is higher than you would expect for just protein alone, which is 20-30%. The evidence suggests for healthy people the requirement for carbohydrate and protein combined, compared to carbohydrate alone (100%) is in the region of 130-190% over 5-6 hours.

WHAT ABOUT RESEARCH ON PEOPLE WITH TYPE 1 DIABETES?

Research on children with Type 1 diabetes shows increasing protein from 5g to 40g protein for a 30g carbohydrate meal increased the glucose level at three hours by 2.4mmol/l. The glucose level stayed elevated at five hours by 2.6mmol/l, when compared to 30g of carbs alone (9). This suggests that extra insulin was needed in the first three hours.

SUMMARY

Very simply, when carbohydrate and protein are consumed, the insulin increases more than would be predicted from their individual effects. The main factor is the synergy of AA and glucose in the B-Cell leading to a very high energy state and insulin secretion. Glucagon only slightly increases, therefore the I:G is increased significantly. This allows anabolism and glucose storage as glycogen.

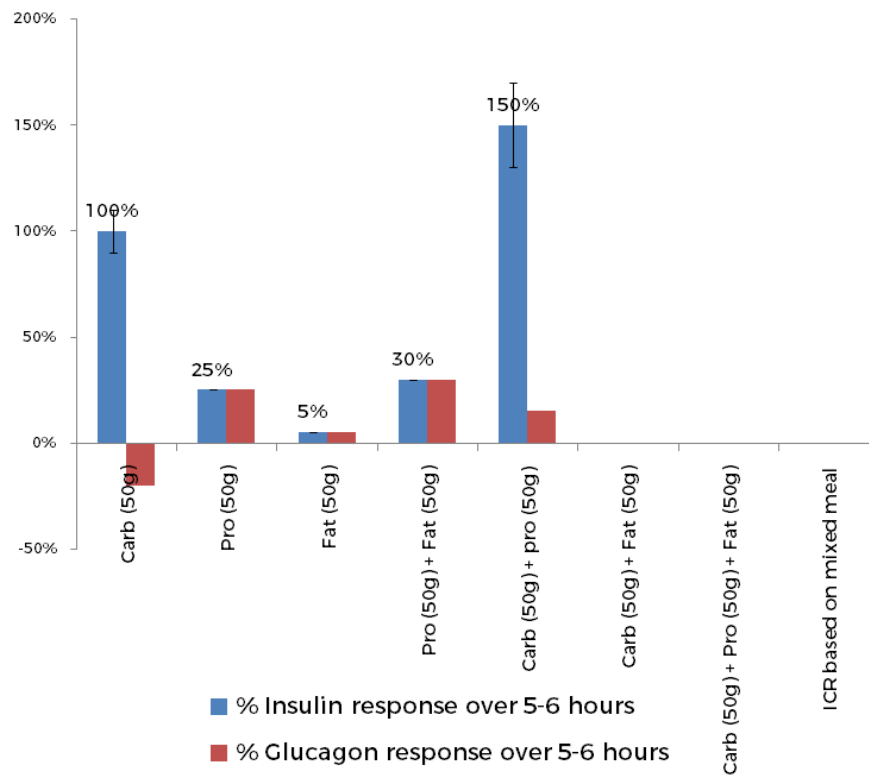
The job of the person with Type 1 Diabetes is to match the amount of insulin the pancreas would release for carbohydrate and protein. The main risk is too little leading to substantial hyperglycaemia, but too much would cause hypoglycaemia.

HOW MUCH INSULIN TO DELIVER FOR CARBOHYDRATE AND PROTEIN ON A GRAM BASIS?

The research suggests compared to carbohydrate alone (100%), the insulin requirement for carbohydrate and protein together is between 130-190%, dependent on the type of protein.

The data suggests that the average insulin requirement is about 150% for carbohydrate and protein, when compared to carbohydrate alone. For Glucagon, a slight increase from baseline of about 15%. Both are represented in the graph below. There is a 20% error bar

included to accommodate the type of protein.



To go deeper into the physiology, biochemistry and research for protein and carbohydrate, access that section of the full report.

WHAT DOES THE PANCREAS DO WHEN CARBOHYDRATE AND FAT ARE CONSUMED?

INSULIN

Put simply, the B-Cells secrete extra insulin to move glucose into the cells for storage, and promote FFA storage as fat tissue.

The big difference here is that the increased insulin requirement is not as immediate, it rises more gradually over time. The stomach empties at a constant energy rate, therefore because fat is very energy dense, gastric emptying will be delayed (7). In healthy individuals the addition of fat to a carbohydrate meal delayed both the glycaemic and insulin responses(12).

GLUCAGON

Put simply, the A-Cells secrete a little extra Glucagon to increase FFA oxidation.

RESEARCH

In one study it was shown that giving eleven type 1 adults an additional 50g of fat, to a high carbohydrate low fat (96g carb& 10g fat) meal, increased the insulin requirement by 42% on average. The 42% increase was determined by closed loop CGM insulin delivery. The 42% was still not really enough, as the glucose results after the meal with added 50g fat were on average higher than the 96g carb and 10g fat meal (10). Interestingly the extra requirement ranged in participants from -17% to 108%. The extra insulin was needed up to 5-10hours after ingestion, confirming the delayed gastric emptying effect.

A study on children with Type 1 Diabetes supports these findings (9). This particular study showed adding 30g fat to a 30g carbohydrate 5g fat meal actually lowered the post-meal glycaemic response in the first 90 minutes. However from 210-300minutes the additional fat increased the glucose.

SUMMARY

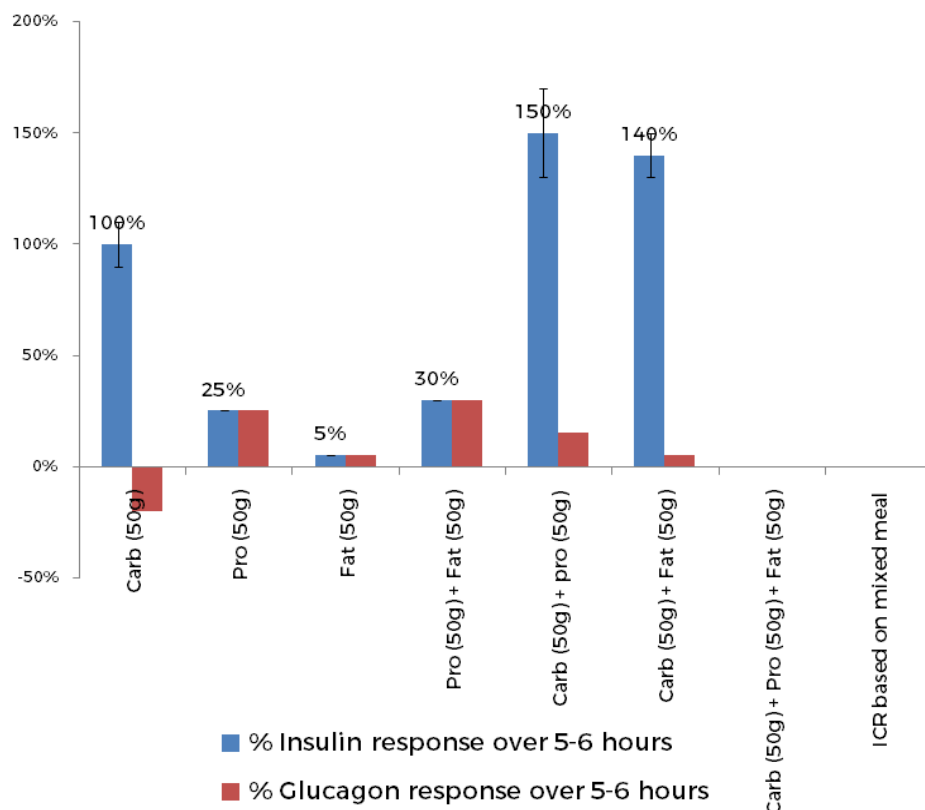
Very simply, when carbohydrate and fat are consumed the insulin increases more than would be predicted from their individual effects on insulin. The main factor is the synergy

of FFA and glucose in the B-Cell leading to a very high energy state and insulin secretion. Glucagon only slightly increases, therefore the I:G is increased significantly. The increase in insulin is not as immediate when compared to carbohydrate and protein, due to the delayed gastric emptying from the high fat intake. This change in I:G promotes glucose storage as glycogen, and FFA storage as fat over a prolonged period of time.

The job of the person with Type 1 Diabetes is to match the amount of insulin the pancreas would release for carbohydrate and fat. The first risk is too much insulin too early causing hypoglycemia. But later on, after about three hours has passed, the big risk is not having enough insulin leading to significant hyperglycaemia.

HOW MUCH INSULIN TO DELIVER FOR CARBOHYDRATE AND FAT ON A GRAM BASIS?

The research suggests compared to carbohydrate alone (100%), the insulin requirement when adding 50g fat is 142%. This is much greater than if you added the fat only response to the carbohydrate only response, that would have only been 105%. For Glucagon, a slight increase from baseline about 5%, as represented in the graph below. The error bar of 10% represents the variation in insulin response dependent on FFA type. Simply, MUFA require less insulin than SFA when combined with carbohydrate.



To go deeper into the physiology, biochemistry and research for carbohydrate and fat, access that section of the full report.

WHAT DOES THE PANCREAS DO WHEN CARBOHYDRATE, FAT AND PROTEIN ARE CONSUMED?

INSULIN

Put simply, the B-Cells secrete a lot more extra insulin for anabolism, glucose storage, and FFA storage.

GLUCAGON

Put simply, the A-Cells secrete slightly more Glucagon to promote FAA oxidation and gluconeogenesis later on.

If the person with Type 1 Diabetes does not deliver the extra insulin required there will be serious hyperglycaemia, that is prolonged.

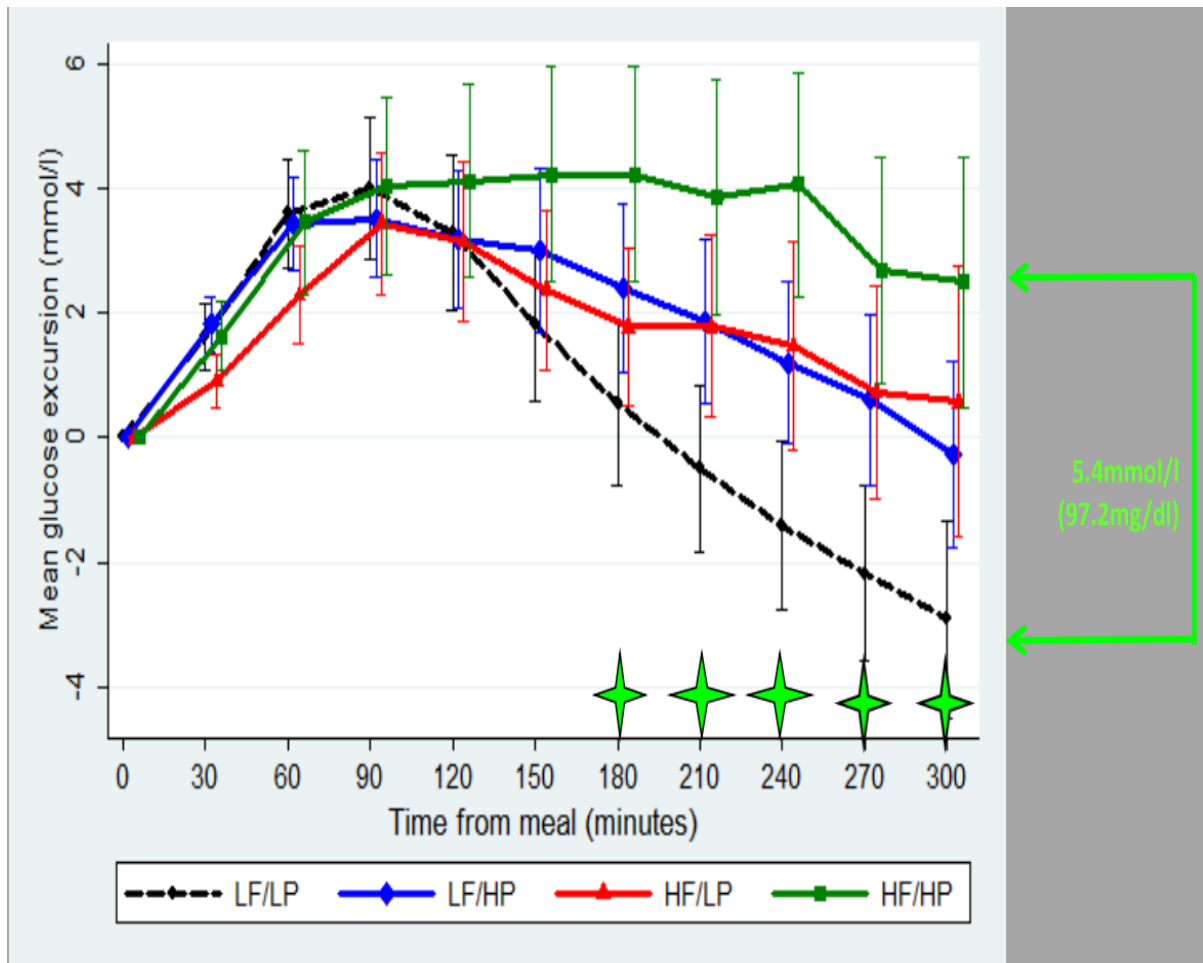
RESEARCH

We have already discussed the value of Smart and colleagues meal study, which looked at the effect of adding protein and fat to carbs (9). However, this study did the tri-factor, it also studied when both fat and protein are added to carbs. Let's take a detailed look at this study.

This study first looked at what happens when you give the insulin dose suggested by the ICR for a moderate carbohydrate low fat low protein meal. Then using that same insulin dose, what happens to the glucose level when the other macronutrients are increased in a stepwise manner. I have put the macronutrient profile of all the different pancakes used in the study:

- Low Fat Low Protein (LF/LP) meal:
 - 30g carbohydrate, 4g fat, 5g protein, 176kcal
- Low Fat High Protein (LF/HP)
 - 30g carbohydrate, 4g fat, 40g protein, 316kcal
- High Fat Low Protein (HF/LP)
 - 30g carbohydrate, 35g fat, 5g protein, 446kcal
- High Fat/High Protein (HF/HP)
 - 30g carbohydrate, 35g fat, 40g protein, 595kcal

The graph below shows what happened to the thirty three adolescents' blood glucose profiles after consuming each of the four meals.



Copied from (9)

WHAT HAVE YOU NOTICED?

This study really helps bring together what we have been building towards. **What does the ICR actually cover?**

If you use your ICR and give insulin according to the carbohydrate in the meal, what happens when:

- That meal is LFLP? You will go hypo! In this study the glucose level was on average 2.9mmol/l (52mg/dl) lower after five hours compared to baseline. This speaks to the fact that when you work out your ICR it is based on your usual mixed meals, not just the carbohydrate content! You can see by 300 minutes both LFHP and HFLP are back to baseline level, -0.3mmol/l (-5mg/dl) and +0.6mmol/l (11mg/dl) respectively. This just goes to show your ICR accounts for the insulin response needed for a mixed meal, not just carbohydrate.

- After 180 minutes the HPLF meal glucose level is significantly higher than the LFLP meal, whereas the HFLP was not. This is just a reiteration of an earlier discussion, that the increase insulin for protein needs to be delivered before the meal. This will stop the rise at 180minutes.
- After 210 minutes the HFLP becomes significantly higher than the LFLP, and stays higher up to 300 minutes. This is just a reiteration of the earlier discussion, that the increase in insulin for fat needs to be delivered later on. Therefore give extra insulin as a split injection or extended wave on a pump
- When HPFH was consumed there was a cumulative effect of adding both protein and fat to LFLP. At 180minutes, HFHP was 3.7mmol/l higher than LFLP, and by 300 minutes that increased to 5.4mmol/l higher. The differences at 180minutes and 300minutes were almost a perfect addition of the individual effect of LFHP and HFLP. Basically showing that both fat and protein need accounting for when insulin dosing, and they are cumulative.

Let's bring this closer to home. In 2016, Bell and colleagues (13) used a closed loop insulin dosing method to find out how much insulin was needed when comparing a pizza base (50g carbs, 4g fat, 9g protein, 273kcal) with a cheese pizza (50g carbs, 44g fat, 36g protein, 764kcal) over six hours. Nine men and one woman with Type 1 Diabetes undertook the pizza challenge.

How much extra insulin do you think was needed?

A whopping 65% extra insulin on average. Not only that, due to the high fat content, the insulin needed to be spread out, 30% upfront and 70% over 2.5 hours via an extended wave on a pump.

Did all ten type 1 pizza addicts need 65% extra?

One person only needed 24% extra, whilst one person needed a massive 124% extra. This is not surprising when you consider the evidence I discuss in the full report, showing that people with insulin resistance have exaggerated insulin requirements for high protein and fat.

SUMMARY

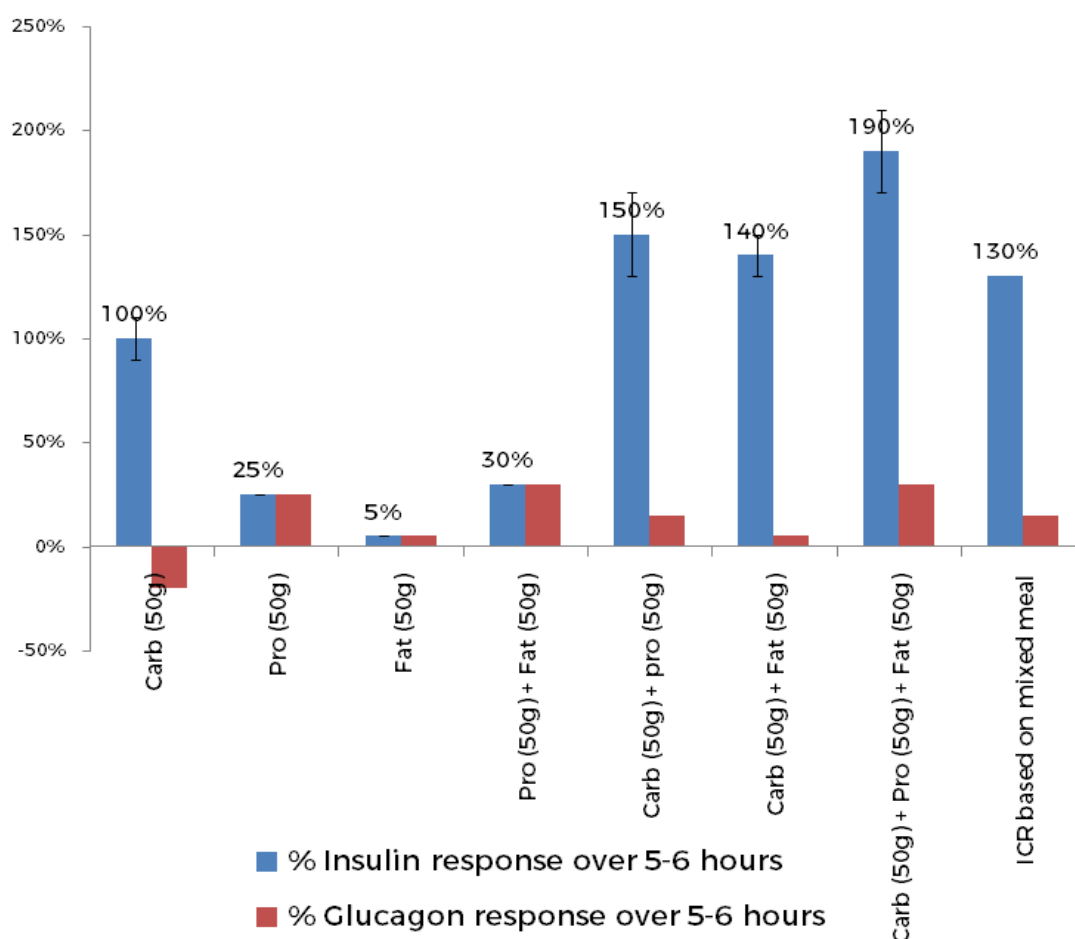
Very simply, when carbohydrate, fat and protein are consumed, the extra insulin required compared to carbohydrate alone is equal to adding the extra needed for both carbohydrate plus protein, and carbohydrate plus fat. The main factor is the synergy of glucose, AA, FFA in the B-Cell leading to a very high energy state and insulin secretion. Glucagon only slightly increases, therefore the I:G is increased significantly. The increase in insulin is both immediate due to AA, and delayed due to FFA. This allows AA to be used for anabolism, glucose to be stored as glycogen, and FFA to be stored fat.

The job of the person with Type 1 Diabetes is to match the amount of insulin the

pancreas would release for this tri-factor. The main risk is not giving enough insulin leading to early and persistent hyperglycaemia. The risk of hypoglycaemia is very low when a meal as large as this is consumed.

HOW MUCH INSULIN TO DELIVER FOR CARBOHYDRATE, PROTEIN AND FAT ON A GRAM BASIS?

This research suggests when adding protein and fat to carbohydrate, the increase in insulin requirement is cumulative. Therefore following our graphical model we need to add the extra 50% and 40% together, to get a 190% requirement in comparison to carbohydrate alone. We also need to add a 30% error bar to include the type of protein and fat. With a meal high in dairy protein and SFA (Butter, cheese, cream) having a higher requirement. This is discussed in the full report.



WHY DO MOST PEOPLE WITH TYPE 1 DIABETES NOT COUNT FAT AND PROTEIN?

Most people with Type 1 Diabetes who carb count do not see this effect regularly, Why:

- The ICR accounts for 130% of the effect of carbohydrate alone. Because of this there will be no blood glucose issue as long as the protein and fat content of the meals are moderate and consistent. This is shown in the above graph.
- Why 130% for the ICR?
- Smart and colleagues (9) showed the ICR worked perfectly for a 30g carbohydrate meal with high fat low protein (35g, 5g) and low fat high protein (5g, 40g). But the ICR gave too much insulin for the 30g carb low fat low protein (5g, 5g) meal. Finally the ICR did not give enough insulin for the 30g carb high fat high protein (35g, 40g) meal. It would therefore seem sensible that ICR accounts for about 130% of the effect on carbohydrate alone. This is obviously an approximation but helps put the insulin dosing strategies into context.

However, when they venture outside the normal fat and protein in meals, they have issues:

- Very high glucose levels after pizza, cheesy pasta, large meals with fancy desserts, BBQ, fish and chips.
- Hypo when have carb only meals such as fruit.

If the person with Type 1 Diabetes wanted to use this very labour intensive approach of counting all macronutrient grams, they would have to consider a few things.

- First: Count every gram of carb, fat and protein, and apply the below formula to get the insulin load. But they must remember to relax their ICR by about 60%. For example a 1u:10g would change to a 1u:16g. Also you would probably want to change the term to a meal ratio rather than carb ratio.
 - $\text{Insulin load} = ((\text{protein (g)} \times 0.5) + (\text{fat (g)} \times 0.4)) + \text{g carbohydrate}$
- Second, when the fat amount gets high (>40g), split the insulin 50/50 giving a second injection in an hour's time on MDI, or an extended wave for 2.5 hours if using a pump.

To go deeper into the physiology, biochemistry and research for carbohydrate, protein and fat, access that section of the full report.

Now we have a good understanding of what the pancreas would do, let's see if the currently available insulin dosing strategies are fit for purpose.

CURRENT AVAILABLE INSULIN DOSING STRATEGIES

We have developed a good understanding of the hormonal interplay required to maintain a stable blood glucose level after eating. We have also highlighted the unique challenges a person with Type 1 Diabetes faces. We are now in a fantastic position to evaluate the most popular insulin dosing strategies.

Let's have a look at the current insulin dosing strategies to;

- Appreciate their benefits and draw backs.
- Know when they will work and when they will not.
- See what diet types they fit best.
- See if we can swap and change strategies depending on meal composition, dietary strategy, or phase of training.

CARBOHYDRATE COUNTING

This method is based on counting the carbohydrate content of a meal, then applying an ICR to determine the insulin dose. For example if you have a ratio of 1unit for 10g carbs and eat 30g, that's 3 units, simple!

Carbohydrate counting is the gold standard method for determining meal time insulin dose recommended by the main regulatory bodies (ADA 14, ISPAD 15). Therefore you would assume there is a solid evidence base on carbohydrate counting's effectiveness.

WHAT DOES THE RESEARCH SAY ABOUT CARBOHYDRATE COUNTING?

The only high quality meta-analysis performed on the effectiveness of carbohydrate counting was performed by Bell in 2014 (16). Only seven (5 adult and 2 paediatric studies) out of 311 studies passed the inclusion criteria, highlighting the poor quality of research studies on carbohydrate counting.

The seven studies combined showed an insignificant HbA1c reduction of only 0.35% over three months or longer. Carbohydrate counting was only found to be significantly beneficial for adults (reduction of 0.4%), but not for children.

Why is that?

Accuracy is one issue. Carbohydrate counting accuracy within 10-15g can be achieved by 50-80% of motivated parents, under experimental conditions in the lab. But for adolescents, only 23% achieve the necessary level of accuracy (17). For adults, the research consistently shows 50% achieve accuracy within 10-15g (18).

So if you are within 10-15g accuracy, all is good, right?

Not quite. We have discussed carbohydrate counting only works when protein and fat content are within usual amounts for the individual (9).

Taken together this research shows:

If you keep fat and protein consistent then carbohydrate counting accuracy only needs to be within 10-15g.

As discussed earlier, ICR's are tried and tested eating usual mixed meals, therefore they already take into account the glucose raising effect of your usual protein and fat intake. So really it's not an ICR, it's a usual mixed meal ratio using carbohydrate as the main determinant.

FAT AND PROTEIN UNITS (FPU) - WARSAW METHOD.

The Warsaw FPU method acknowledges fat and protein have an effect on post-prandial blood glucose level and require insulin(19). The FPU method assumes that 100kcal of either fat (11g) or protein (25g) has the same insulin requirement as 10g carbohydrate. So if the person has a 1u:10g ICR, then 100kcal of fat and protein requires 1 unit of insulin, called 1 FPU. If ICR is 1u:20g, then 100kcal of fat and protein needs 0.5 units insulin, 0.5FPU.

How that FPU insulin is delivered is unique. The insulin for the carbohydrate is always given before eating. Then the insulin required for the FPU is then spread out using an extended bolus over a number of hours via a pump. The length of extension for the FPU is determined by the total FPUs:

- 1 FPU = 3 hours
- 2FPU = 4 hours
- 3FPU = 5 hours
- >3FPU = 8 hours

WHAT DOES THE RESEARCH SAY ABOUT THE WARSAW FPU METHOD?

Research has shown following the FPU system caused a lot of hypoglycaemia when compared to carbohydrate counting(19).

We must also consider the maths involved. We already discussed the most motivated group's only count carbohydrate with 50-80% accuracy, adults 50%, and adolescents at 23%. What would happen if they also needed to count fat and protein grams and apply formulas?

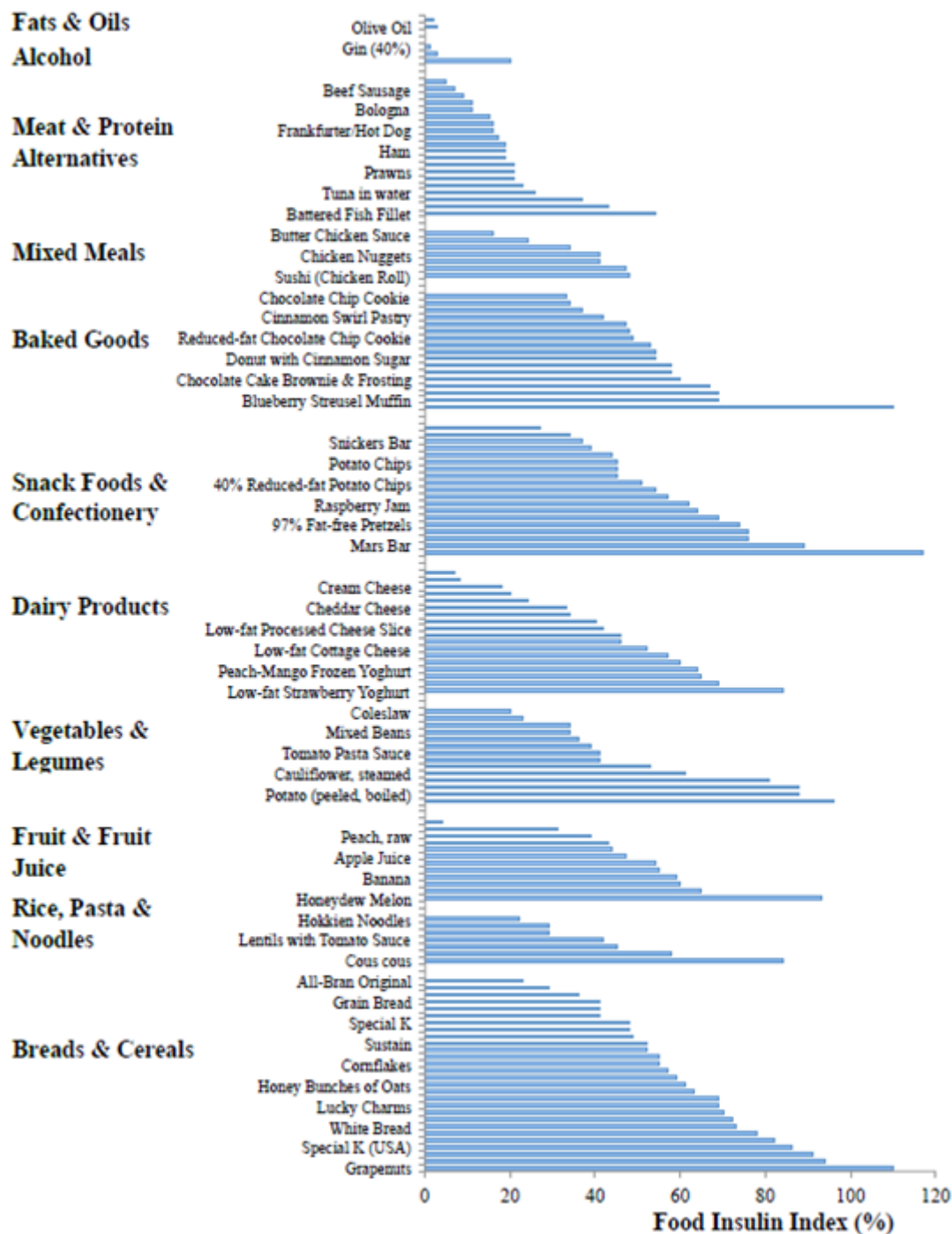
Food Insulin Index (FII) and Food Insulin Demand(FID):

The FII was developed by measuring the incremental area under the plasma insulin curve observed by a 1000 kJ portion of a test food (20), expressed as a percentage of the

response to a 1000 kJ portion of the reference food (glucose) within ten lean, healthy subjects.

Therefore instead of measuring the glucose response, like the GI, it measures the insulin requirement. This should be much more useful for people with Type 1 Diabetes.

The FII database has been added to over 20 years and now has 147 foods, some single foods and some meals. The FII is a percentage insulin response over two hours compared to 100% of Glucose, a summary is presented below in the table.



Copied from Bell (20)

WHAT DOES THE RESEARCH SAY ABOUT THE FOOD INSULIN INDEX (FII) METHOD?

The FOODII(21) study tested the FII vs. carbohydrate counting in 26 patients with Type 1 Diabetes over three months. The team developed resources, a SMART phone APP, and other educational materials equivalent to those available for the carb counting.

After three months they found no difference in HbA1c between the FII and carb counting groups. They did find a trend towards a reduction in hypoglycaemia in favour of the FII. The FII counters reported after the first couple of weeks learning it was easy to follow, certainly not more difficult than carb counting.

The FII in principle sounds like it should have improved HbA1c, why did it not show clear benefits for glycaemic control?

- The FII only accounts for two hours insulin demand. To measure true insulin requirement, a period of 5-8 hours is needed. Again this likely means the insulin demand for mixed meals is massively underrepresented.

SUMMARY

If you are thinking that none of the current insulin dosing systems are fool proof, you are correct. But at least you now understand the pro's and con's of each approach. You also know how different macronutrients require a different insulin response depending on whether they are eaten singularly or combined.

Rather than choosing an insulin dosing strategy and trying to make your diet fit around it, flip it round. Why not choose the type of nutritional intake that suits your lifestyle, food preferences, and goals in life...then choose an insulin dosing strategy that best suits that nutritional intake?

To go deeper into the research for the currently available insulin dosing strategies, access that section of the full report.

CHANGING THE GAME: CHOOSE YOUR DIET FIRST, THEN CHOOSE YOUR INSULIN DOSING STRATEGY?

We will evaluate the most common dietary approaches, and see which insulin dosing strategy fits best.

GOVERNMENT AND DIABETES ASSOCIATIONS RECOMMENDATIONS

The ADA (14) and ISPAD (15) recommend a nutritional intake of:

- 50% carbohydrate
- 30% fat
- 20% protein
- Low GI
- Starchy wholegrain foods
- At least five fruit & vegetables a day
- Choose MUFA and keep SFA low
- Keep sugar less than 5-10% of total energy

The reason why all the governments and agencies promote this type of intake is because:

The wealth of research evidence resoundingly supports that this type of intake gives the best chance for a healthy disease free life.

If you were to believe the "guru bloggers", you would think all the governments and diabetes associations are conspiring against people with type 1 diabetes. Reality check! This type of intake is based on very sound evidence. It is not a conspiracy, it's sound science.

A diet that is 50% carbohydrate will provide the necessary fast burning fuel to optimally power:

- Fast paced sports activities such as football, basketball, rugby, netball, tennis, rowing.
- Resistance exercise to promote strength and muscle growth.
- Endurance activity where optimal performance is the main goal. Events such as competitive running, triathlon and swimming.
- Recovery from exercise.

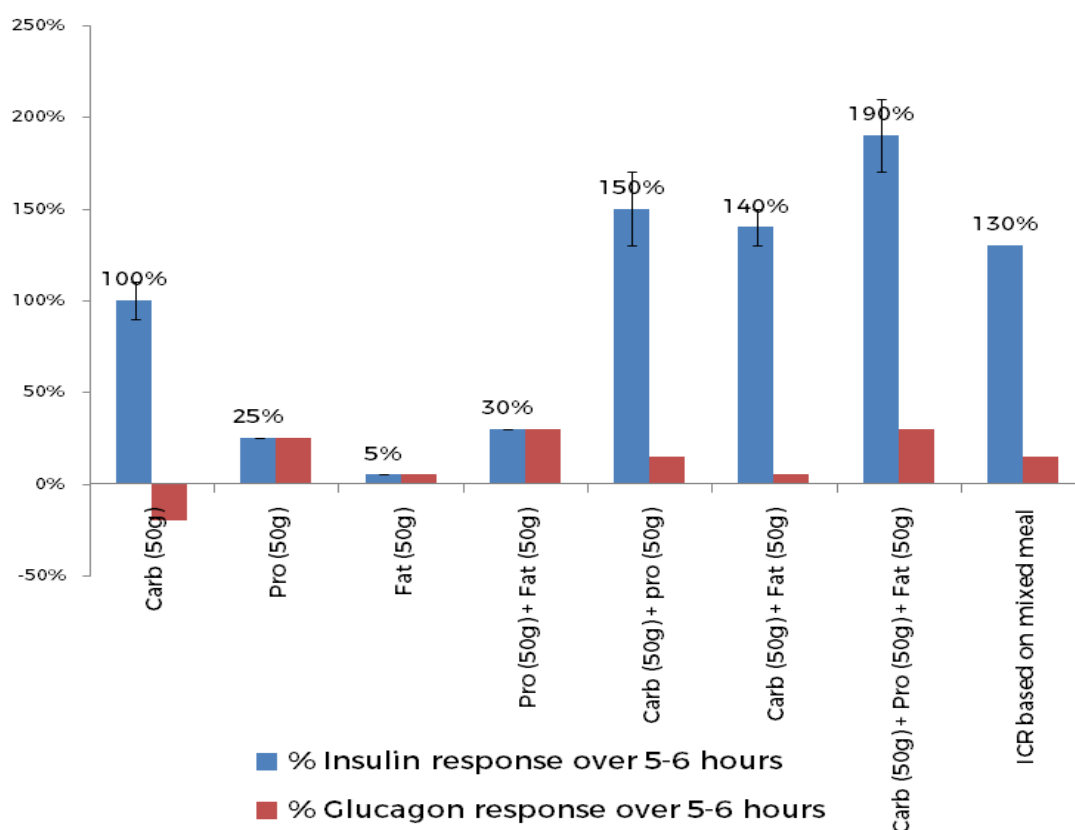
That being said, it is challenging to attain a HbA1c of <6.5% (48mmol/l) eating 50% of energy intake from carbohydrate. It can be done if you;

- Choose mainly low GI options.
- Have a good daily level of physical activity.
- Test blood glucose regularly and correct frequently.
- Deliver meal time insulin 15-30 minutes before meals.

- Plan exercise effectively and replace carbohydrate when you are most sensitive to insulin.
- Regularly monitor and change your diabetes regime according to your results.

Here are some tips to get your insulin dose as accurate as possible.

Consistency is the key to getting good diabetes control when carbohydrate counting. Keep your macronutrient split and amounts consistent, then just count the carbohydrate and give the insulin based on your ICR. Remember from the graph below that usual mixed meal ICR accounts for about 130% of the effect of carbohydrate.



- Working out your ICR for your usual mixed meals.
 - **Breakfast:** If you calculate your ICR for cereal with milk, which is usually about 50g carb, 15g protein, 10g fat, the ICR will work for breakfasts of that composition. If you change to fry up that is 20g carbohydrate 30g protein, 30g fat, you will certainly have a high glucose reading if you only bolus for the carb amount using your ICR!
 - **Lunch:** If you calculate your ICR for sandwich, yoghurt, crisp, which is usually 50g carb, 20g protein, 20g fat. The lunch ICR will work for meals very similar to that. When you change to salad with a lot of protein and fat dressing (Caesar Salad), which is 10g carbohydrate, 30g protein, 20g fat, you have the same problem as breakfast.
 - **Evening meal:** If you calculate your ICR for meat, potatoes and vegetables, which is usually 50g carb, 25g protein, 20g fat. When you change to heavily

cheesed pizza, which is 50g carb, 35g protein, 40g fat, you can expect very high reading for a prolonged time. The other way round, what happens if you establish your ICR on consuming high protein and fat foods for evening meal such as Pizza, Lasagna, Cheesy pasta. When you decide to go for the meat, two vegetables and potatoes you go hypo!

- **Snacks:** If you calculate your ICR for snacks on a breakfast bar or crisps, which is usually 15g carb, 1-5g protein, 10g fat. When you change to fruit only, which is 10-15g carb, 0g protein, 0g fat, you can expect a hypo if you use that ICR.

If you decide to change the fat and protein amounts significantly, you must expect high and low blood glucose levels, unless you make allowances. Below are some simple suggestions on how you can make allowances.

- What do you do when you go outside the norm with high fat and protein, such as a pizza? You can use the latest research review(22) suggestion:
 - If protein is 40g or more and carbs are at least 30g, add an extra 15-20% insulin and give before meal.
 - So a 10 unit bolus would become a 12 unit bolus.
 - Easy equation, insulin dose x 1.20
 - When a meal is more than 35g fat and is at least 30g carbohydrate, add 30-35% extra insulin, and spread the insulin out, 50% upfront, 50% later.
 - So a 10 unit bolus would become a 13.0 unit bolus. If on a pump use a dual/split/combo/multiwave wave bolus. If injections give one injection of 7 units before eating, followed by another of 6.5 unit injection in one hours time.
 - Easy equation, insulin dose x 1.3 then divide by 2
 - When you have a meal than is at least 30g carb, at least 40g protein, and at least 35g fat, you need to add 50% extra insulin and spread the insulin out, 50% upfront, 50% later.
 - So a 10 unit bolus would become a 15 unit bolus. If on a pump use a dual/split/combo/multiwave wave bolus. If injections give one injection of 7.5 units before eating, followed by another of 7.5 unit injection in one hours time.
 - Easy equation, insulin dose x 1.5 then divide 2

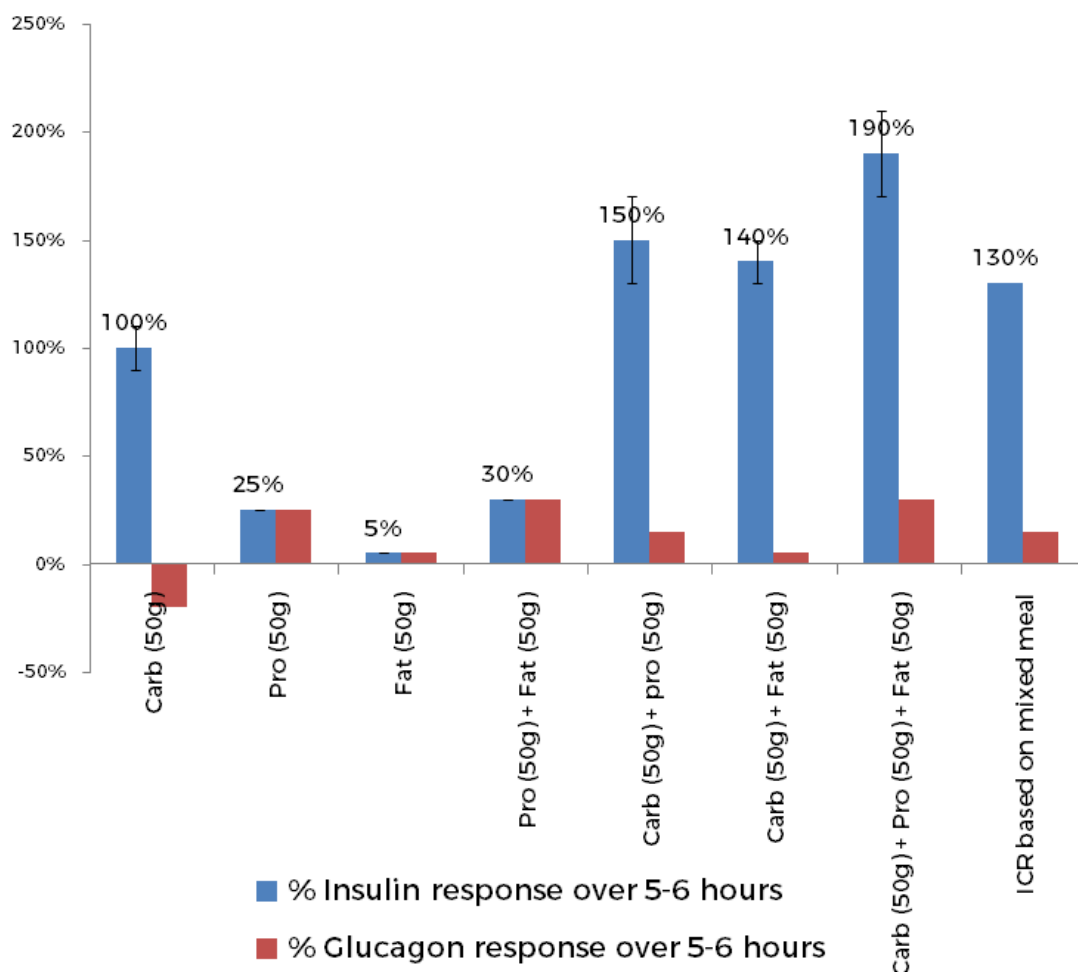
HIGH PROTEIN MODERATE TO LOW CARBOHYDRATE DIETS

The usual make up of this type of diet is protein at 1.5 - 3.0g/kg/day, moderate to low carbohydrate of 50-250g per day, and moderate fat diet. For example, Zone Diet or a physique athlete cutting diet.

There is going to be a significant increase in the insulin requirement from protein with this type of intake. More than can be accounted for by counting carbohydrate alone. Therefore the best approach would be to look at the work of the FII and count the insulin demand as:

- Insulin demand = carbohydrate (g) + (protein (g) x 0.5)

This also mirrors the effect of high protein and moderate carbohydrate meals for the graphical model developed.



You would then need to relax your current ICR to prevent hypos. Relaxing the ratio by 20% would seem like a sensible place to start, considering the ICR is already based on 130% of the effect of glucose.

1. For example, if your ICR is 1unit for 10g carbs, make it 1units for 12g insulin demand.
2. Easy equation; ICR x 1.2

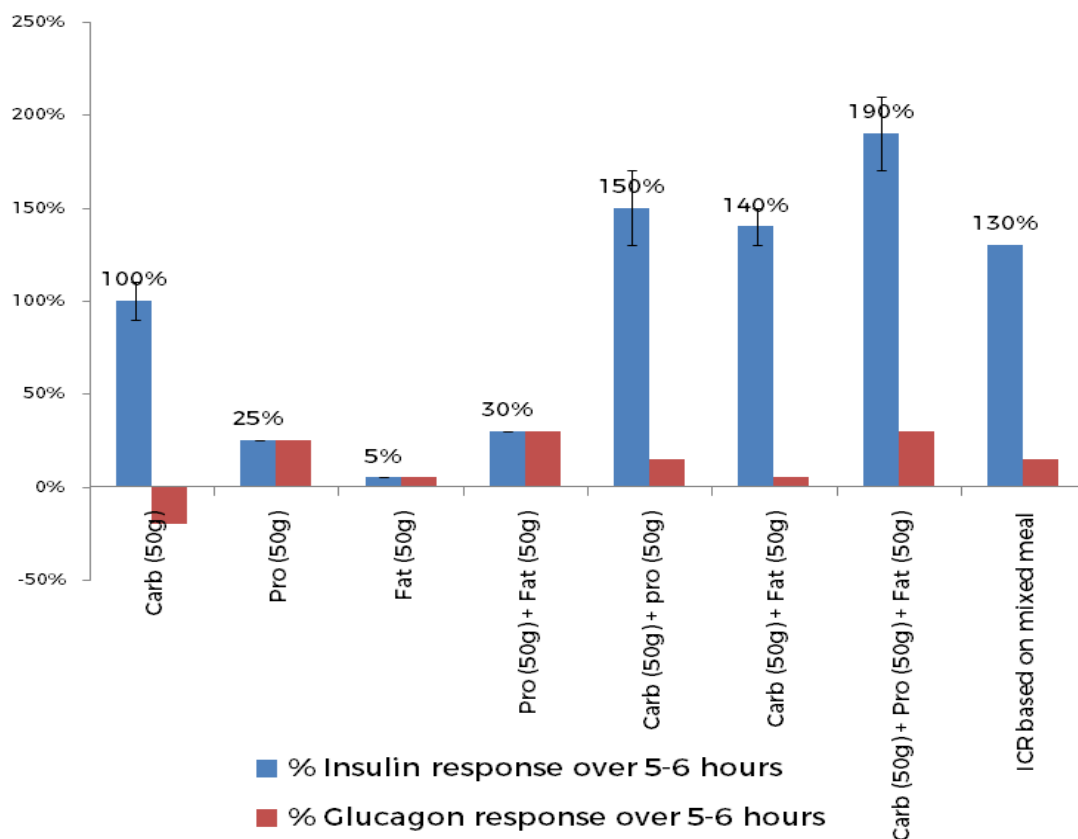
This will prevent too many hypos at the offset. You should adjust your ICR from

monitoring.

Consistency rules! Try and keep your protein portions distributed equally from meal to meal. For example, 25-40g portions at each meal.

VERY LOW CARBOHYDRATE DIETS - LESS THAN 50 GRAMS A DAY

For people choosing to go very low carbohydrate, the insulin requirement for protein and fat is much lower than when they are combined with carbohydrate. Following the research summarised in the graphical model below, only 25% of the protein will need to be considered when insulin dosing, with very little effect from fat, which will most likely be covered by the background/basal insulin.



A formula that may be worth starting with is:

Insulin load = carbohydrate (g) + (protein (g) x 0.25)

You could then apply your ICR to get an insulin dose.

People following this type of approach are generally in two camps:

- A Dr Bernstein style approach, which is typically high in protein >2.0g/kg/day, carbs aiming for maximum 24g per day, and the rest made up with fat. You are **UNLIKELY** to get into nutritional ketosis with this approach, because the protein intake, and therefore insulin delivery, is too high.

- A nutritional ketosis approach which is much lower in protein, usually 0.6-1.2g/kg/day, and 70-80% of the total energy coming from fat. Protein is lower to keep the insulin low. This allows the body to regulate its metabolic machinery to oxidise FFA and ketones as the major fuel source.

The difference in protein levels between the two approaches is very important. The Dr Bernstein style does not aim to achieve nutritional ketosis. It has a much higher protein intake aiming to stimulate anabolism. Whereas nutritional ketosis has a much lower protein and insulin intake, with the aim of increasing production of ketone bodies by the liver.

Why is this important?

Nutritional ketosis is not recommended for people aged under 18 years. A recent research review on children in prolonged nutritional ketosis to treat refractory epilepsy reported (23):

- Increase risk of faltering growth, that catches up when nutritional ketosis is stopped.
- Abnormal lipid profiles linked with increased risk of cardiovascular issues.
- Kidney stones.

DOES THIS MEAN LOW CARBOHYDRATE DIETS SHOULD BE AVOIDED FOR CHILDREN WITH TYPE 1 DIABETES?

It is clear that if children are constantly in nutritional ketosis, it's not good for health (23). However, the vast majority of children following the Dr Bernstein approach have protein intakes way too high to achieve nutritional ketosis. A big mistake is believing that all low carbohydrate approaches are the same. Therefore also believing all low carbohydrate approaches must be detrimental to children with type 1 diabetes is a mistake.

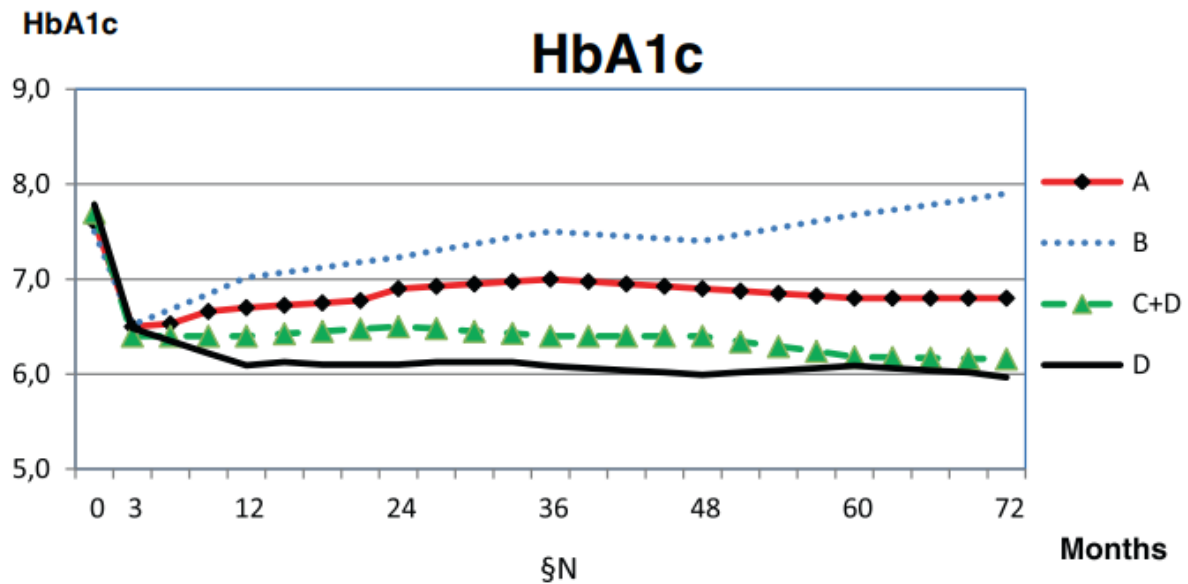
Is the low carbohydrate approach better for managing diabetes, and is it safe in the long term?

The evidence is sparse to say the least.

Only one study (24) has any meaningful long term data on this. A group of 48 adults with Type 1 Diabetes were educated on how to safely consume a diet of less than 75g of carbohydrate per day. They followed them up at 3 months, 3 years and 4 years. The below graph shows what happened to their HbA1c, depending on their adherence to the diet:

- A = All 48 people
- B = Non-adherent to <75g carbohydrate

- C = Partially adherent to <75g carbohydrate
- D = Adherent to <75g carbohydrate



A	48.....	23.....	7
B	25.....	10.....	3
C+D	23.....	13.....	4
D	13.....	8.....	3

Copied from (24)

The main points to understand from this study are:

- If you fully adhere (D) or partially adhere (C) to a low carbohydrate approach, you will get a sustained drop over four years in HbA1c of 1.8% and 0.7% respectively.
- Only 27% of people can fully adhere to low carb, whilst an additional 21% can partially adhere.
- The 27% who fully adhered actually improved their lipid profile. A significantly better TC:HDL, with no increase in other complications. The authors used microvascular and cardiovascular algorithms to predict that the low carb adherers reduced their risk of both sets of complications by 20-40%.
- The low carbohydrate adherers reduced their total bolus insulin by 10units, but background insulin remained the same.

The very limited data suggests a few things. If you are an ADULT and you adhere to a low carb diet, you will very likely improve your HbA1c and complications risk. However, sticking to the diet is going to be a challenge, as only one in four can.

WHAT ABOUT CHILDREN?

The honest answer is nobody knows, because it has not been studied properly! An observational study on the TYPE ONE GRIT (Dr Bernstein approach) group has just been completed by Boston Children's Hospital. It will be very interesting to read the findings when it is published!

There are a few big questions I hope Boston Children's Study will answer:

- Is faltering growth an issue with an adequate energy, high protein low carb diets that DO NOT aim for nutritional ketosis?
- Are there detrimental lipid profile changes?
- If the lipid profile is deranged, is that off-set by keeping HbA1c below 6%?
- What is the social impact?
 - Does restricting food choices have a negative impact on social development?
 - Does having excellent control with less hypos and more predictability improve confidence in living with diabetes?
 - Should it be a personal choice that is NOT dictated by the health care team?

A low carbohydrate diet will very likely improve diabetes control for the average person with type 1 diabetes. However, it is important to consider other lifestyle factors when choosing a nutritional strategy. Some very likely situations that a low carb diet will not be optimal for:

- Anyone who plays a sport that requires fast bursts of energy, such as football, basketball, HITT training etc. Notice it says OPTIMAL. You can do these activities on a low carb diet, you just will not be as explosive, and therefore as good.
- Anyone who wants to compete at their best in endurance events such as half marathon, triathlon and cycling races. Again, these activities can be done on a low carbohydrate diet, there is no doubt. A low carbohydrate diet will certainly achieve better blood glucose control during the activity. However, without adequate muscle glycogen stores, the muscles will not be able to create energy at a high enough rate to perform optimally.
- Anyone wanting to gain significant amounts of muscle in a bulking phase. You need the insulin stimulus and glycogen stores for anabolism, and carbohydrate to fuel hard training, and support recovery (25).
- Anyone who likes to eat carbohydrate foods. They would find it very challenging in social situations. The low carb Dr Bernstein group call themselves GRIT for a reason, and that has to be understood and respected.

As an adult if you decide to follow a nutritional ketosis approach, you may want to cycle in and out of nutritional ketosis to prevent hormonal disturbances, such as thyroid issues. Above all, do plenty of reading and homework before starting. Don't just do it because the in vogue guru thinks it's the best thing since sliced bread. Some key things to read would be:

- [The Art and science of Low Carbohydrate Living](#)
- [The Art and Science of Low Carbohydrate Performance](#)

SUMMARY

You can choose any diet type and achieve first class diabetes control, but only if you think like a B-Cell. This document has provided you with that level of thinking.

The contentious issue for most people with type 1 diabetes is;

Should I go low or high carb?

To answer the question you need to ask:

- What are my diabetes management goals?
- How important is OPTIMAL exercise performance?
- How much time am I willing to invest in managing my diabetes?
- How will I cope with being restricted in social situations?

A person who would suit a lower carbohydrate approach would say:

- I want the lowest HbA1c possible, less than 6.0% (42mmol/mol).
- I am happy with average exercise performance. It's a worthwhile trade-off.
- I do not want to spend too much time checking my glucose level after eating.
- I am happy with restricted food options in social situations, because then I do not have to worry about my diabetes control.

A person who would suit a higher carbohydrate approach would say:

- I am satisfied with a HbA1c less than 7.0% (52mmol/mol).
- My focus is on optimal exercise performance. I am happy to trade this for a slightly higher HbA1c.
- I am happy to take more after meal glucose readings, correct with insulin, and spend more time in reviewing and making changes.
- I want the freedom at social situations to eat and drink what I desire, even though I know there will be blood glucose consequences.

Whichever nutritional approach you choose, this article has provided you with necessary skills. It's now time to experiment.

To make your experimentation easy, I have developed an excel calculator that incorporates all of the knowledge and formulas we have worked through. You simply enter your carbs, fat protein, and ICR, and then voila, the calculator presents you with all of the insulin dosing options.

To experiment with the calculator access the members site

To watch the video module of how the calculator works go to the Bolus Wizard section of the Members site

To go deeper into the research for choosing your nutritional intake then selecting an appropriate insulin dosing strategy , that section of the full report

CONCLUSION

It would be nice if all you had to do was count your carbs, use an ICR, and everything will be ok. But the reality is you cannot! Well you can, but just don't expect good diabetes control.

Respect the fact that your current ICR's are set for the insulin requirement for your usual mixed meals. If you keep to consistent amounts of carbs, fat and protein at meal times, your ICR's will work. When venturing outside your usual meals, you will need to increase or decrease the insulin accordingly, and spread the delivery for very high fat meals.

You must change your insulin dosing strategy to include protein counting if you follow a high protein diet that is moderate to low in carbohydrate. If you decide to try the Dr Bernstein or Ketogenic diet, you will need to account for a small amount of protein when insulin dosing.

This document and the calculator offer a great starting point. It's now time to take some action and start experimenting. One tool that will prove invaluable in your experimentation is CGM. CGM will give you immediate feedback on what does and does not work.

If you often say; diabetes is really hard to manage, I never get my meal time doses right, it's like having a crap part time job, my friends don't have to deal with the grind. Consider this:

There has never been a better time to have Type 1 Diabetes. There is quick acting insulin, pumps, CGM, downloads, and an abundance of quality information and. Make the most of what's available, and meet the challenge head on with persistence!

REFERENCES

1. Lafrance L, Rabasa-Lhoret R, Poisson D, Ducros F, Chiasson JL. Effects of different glycaemic index foods and dietary fibre intake on glycaemic control in type 1 diabetic patients on intensive insulin therapy. *Diabet Med* 1998;15: 972–978
2. Nansel TR, Gellar L, McGill A. Effect of varying glycemic index meals on blood glucose control assessed with continuous glucose monitoring in youth with type 1 diabetes on basalbolus insulin regimens. *Diabetes Care* 2008;31: 695–697
3. Charlton MR, Adey DB, Nair KS. Evidence for a catabolic role of glucagon during an amino acidload. *J Clin Invest* 1996;98(1):90–9. [PubMed: 8690809]
4. Krezowski P, Nuttall F, Gannon M, Bartosh N. The effect of protein ingestion on the metabolic response to oral glucose in normal individuals. *Am J Clin Nutr*.1986; 44: 847 - 56.
- 5 . Berger S, Vongaraya M. Insulin response to ingested protein in diabetes. *Diabetes*.1966; 15: 303 - 6.
6. Carr RD, Larsen MO, Winzell MS, Jelic K, Lindgren O, Deacon CF, Ahren B. Incretin and islet hormonal responses to fat and protein ingestion in healthy men. *Am J Physiol Endocrinol Metab* 295: E779 –E784, 2008. First published July 2, 2008;doi:10.1152/ajpendo.90233.2008
7. Carbonnel F, Lemann M, Rambaud JC, et al. Effect of the energy density of a solid-liquid meal on gastric emptying and satiety. *Am J Clin Nutr*. 1994;60(3):307–11.
- 8 .Uthoff, H. et al (2010) Skipping meals or carbohydrate-free meals in order to determine Basal insulin requirements in subjects with type 1 diabetes mellitus? [Exp Clin Endocrinol Diabetes](#). 2010 May;118(5):325–7. doi: 10.1055/s-0029-1241199. Epub 2010 Jan 12.
9. Smart CEM, Evans M, O'Connell SM, et al. Both dietary protein and fat increase postprandial glucose excursions in children with type 1 diabetes, and the effect is additive. *Diabetes Care* 2013;36:3897–3902
10. Wolpert HA, Atakov-Castillo A, Smith SA, Steil GM. Dietary fat acutely increases glucose concentrations and insulin requirements in patients with type 1 diabetes: implications for carbohydrate-based bolus dose calculation and intensive diabetes management. *Diabetes Care* 2013;36:810–816
11. Esteves de Oliveira FC, Pinheiro Volp AC, Alfenas RC. Impact of different protein sources in the glycemic and insulinemic responses. *Nutr Hosp*. 2011; 26: 669–76.
12. Welch IM, Bruce C, Hill SE, et al. Duodenal and ileal lipid suppresses postprandial blood glucose and insulin responses in man: possible implications for the dietary management of diabetes mellitus. *Clin Sci (Lond)*. 1987;72(2):209–16.

13. Bell et al (2016) Optimized Mealtime Insulin Dosing for Fat and Protein in Type 1 Diabetes: Application of a Model-Based Approach to Derive Insulin Doses for Open-Loop Diabetes Management *Diabetes Care* 2016;39:1631–1634 | DOI: 10.2337/dc15-2855
14. ADA (2016) Standards of Medical Care - 2016. Available @ http://care.diabetesjournals.org/content/suppl/2015/12/21/39.Supplement_1.DC2/2016-Standards-of-Care.pdf
15. ISPAD (2014) Clinical Practice Consensus Guidance. Available @ <https://www.ispad.org/?page=ispadclinicalpract>
16. Bell K, Barclay AW, Petocz P, Colagiuri S, Brand-Miller JC. The efficacy of carbohydrate counting in type 1 diabetes: a systematic review and meta-analysis. *Lancet Diabetes & Endocrinology* 2014; 2(2): 133–140
17. Bishop F, Maahs D, Spiegel G, et al. The carbohydrate counting in adolescents with type 1 diabetes (CCAT) study. *Diabetes Spectrum*. 2009; 22: 56–62.
18. Nebel I-T, Bluher M, Starcke U, Muller UA, Haak T, Paschke R. Evaluation of a computer based interactive diabetes education program designed to train the estimation of the energy or carbohydrate contents of foods. *Patient Educ Couns*. 2002; 46: 55–9.
19. 168. Pankowska E, Szypowska A, Lipka M, Szpotanska M, Blazik M, Groele L. Application of novel dual wave meal bolus and its impact of glycated hemoglobin A1C levels in children with type 1 diabetes. *Pediatr Diabetes*. 2009; 10: 298–303.
20. Bell, K. et al (2016) Algorithms to Improve the Prediction of Postprandial Insulinemia in Response to Common Foods. *Nutrients* 2016, 8, 210; doi:10.3390/nu8040210
21. Bell (2014) Clinical Application of the Food Insulin Index to Diabetes Mellitus. Available at: <https://ses.library.usyd.edu.au/handle/2123/11945>
22. Bell, K. et al (2015) Impact of Fat, Protein, and Glycemic Index on Postprandial Glucose Control in Type 1 Diabetes: Implications for Intensive Diabetes Management in the Continuous Glucose Monitoring Era. *Diabetes Care* 2015;38:1008–1015 | DOI: 10.2337/dc15-0100
23. Sharma S., Punnet, J. (2014) The ketogenic diet and other dietary treatments for refractory epilepsy in children [Ann Indian Acad Neurol. 17\(3\); Jul-Sep 2014](#)
24. Neilsen et al (2012) Low carbohydrate diet in type 1 diabetes, long-term improvement and adherence: a clinical audit. *Diabetology and Metabolic Syndrome*, 4:23.
25. Hawley JA, Burke LM, Phillips SM, Spriet LL: Nutritional modulation of training-induced skeletal muscle adaptations. *J Appl Physiol* 2011, 110:834–845.

