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

FAT ONLY

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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

WHAT DOES THE PANCREAS DO WHEN FAT ONLY IS CONSUMED?

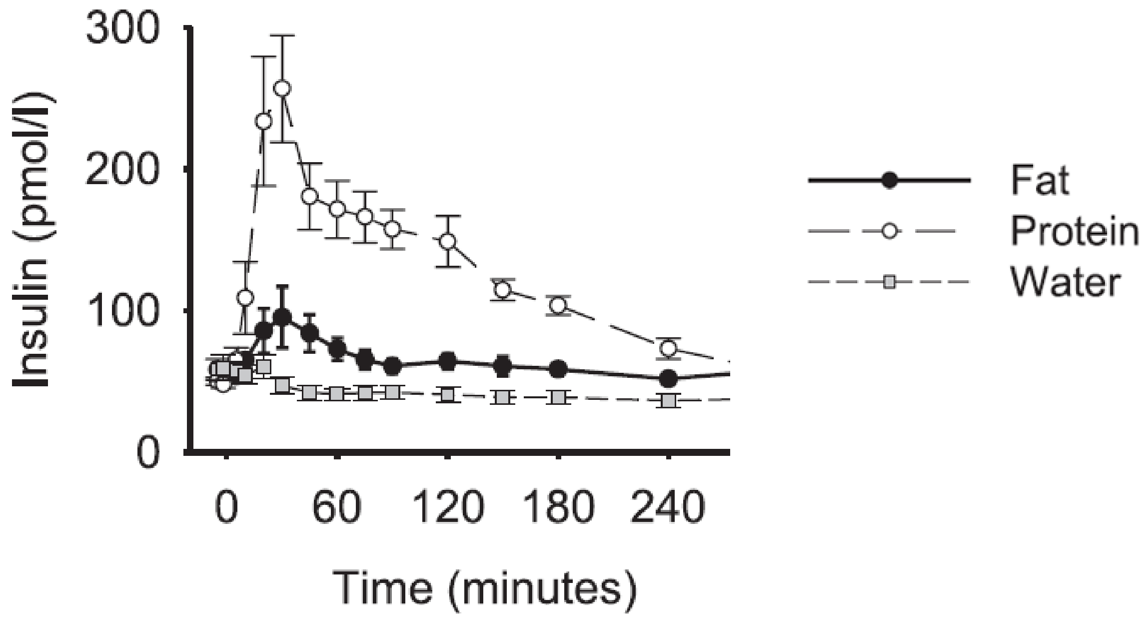
INSULIN

Put simply, the B-Cells secrete a little extra insulin into the blood to promotes storage of FFA in the fat tissue. There are a lot of signals that regulate insulin secretion, but the main and most important ones are:

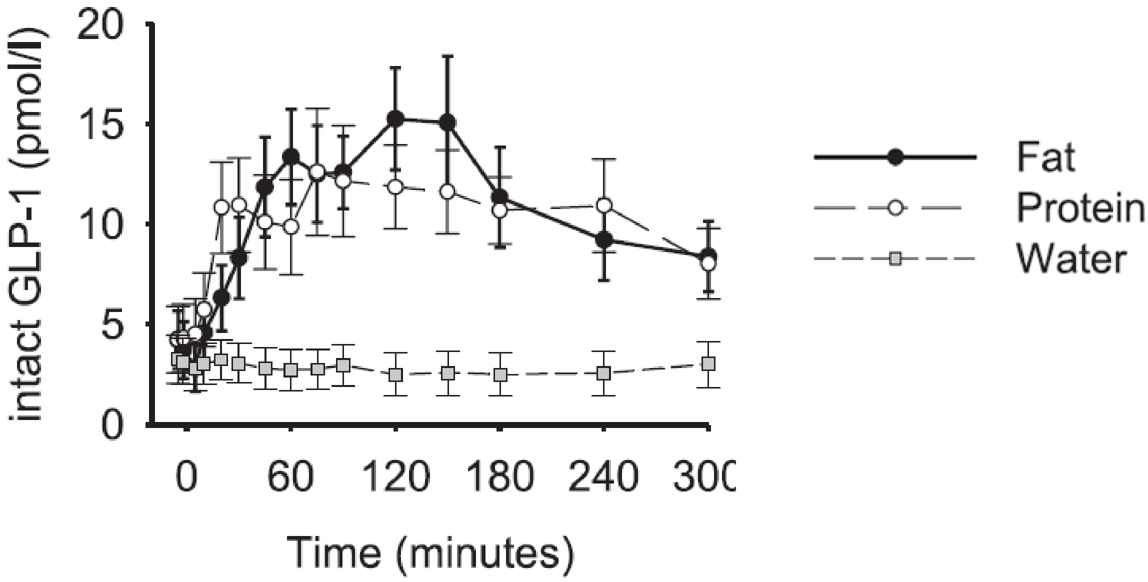
A. Intestinal Y cells release GLP-1 which is a strong positive regulator of B-Cell insulin secretion (10). The level of GLP-1 response to FFA is the similar to that of AA (65).

B. Increased FFA uptake by the B-Cells increases the energy state leading to insulin secretion. This effect is much weaker than that of AA (31)

Research on healthy adults consuming fat only (0.88g/kg, 560kcal) show the insulin level begins to increase at 30minutes post-ingestion, and remains elevated up to 240 minutes (9). At 30 minutes the insulin concentration doubles, but this is a much lower response of the fivefold increase in response to the same kcal volume of protein. Granted having a pure fat meal of 62g on its own is unlikely in one sitting!



As discussed earlier and shown in the graph below from the same study, the secretion on intact GLP-1 from the L-Cells of the intestine due to dietary FFA plays a major role in insulin secretion.



GLUCAGON

Put simply, the A-Cells secrete a little extra glucagon into the blood to promote FFA oxidation and glycogenolysis. There are a lot of signals that regulate Glucagon secretion, but the main and most important ones are:

C. G cells located in the small intestine sense the FFA and release GIP. GIP positively regulates A-Cell Glucagon secretion.

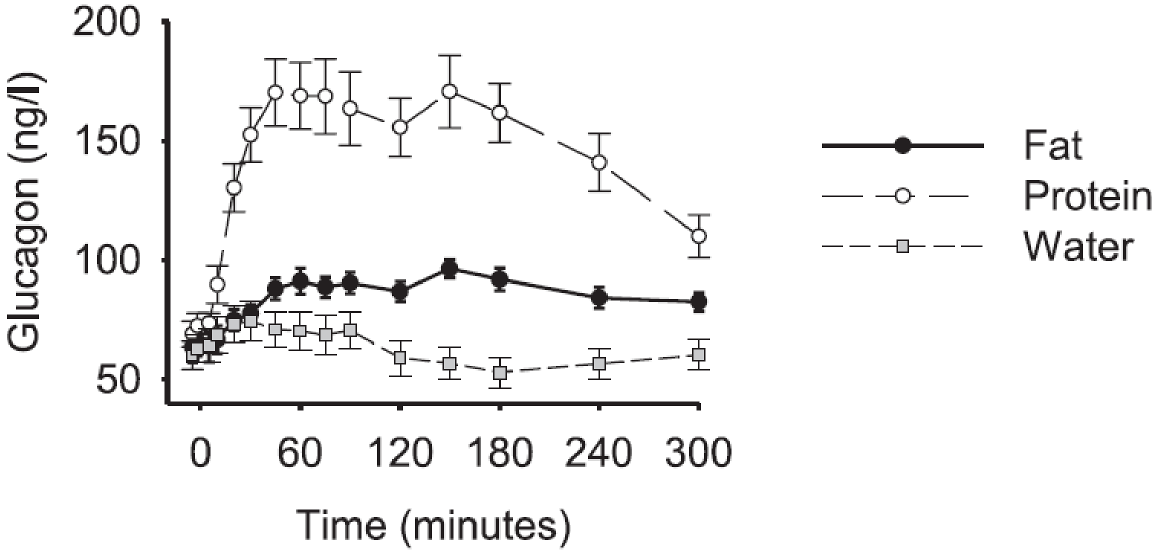
D. The FFA from fat digestion increase GLP-1 secretion from the L Cells of the intestine. This positively regulates B-Cell secretion of insulin, which in turn negatively regulates A-Cell Glucagon secretion. However, the GLP-1 secretion is moderate compared to when glucose is ingested, and the positive regulation by GIP leads to a net Glucagon increase after fat is consumed.

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 Gluconeogenisis. This could lead to a late rise in glucose after eating fat only meals.

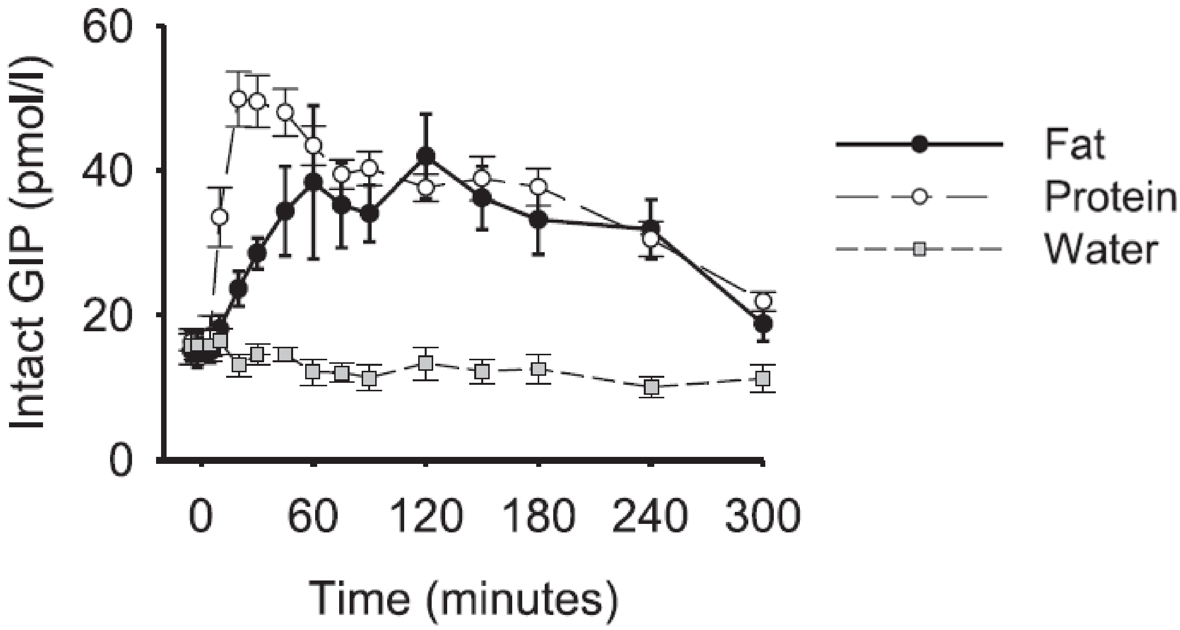
Research on healthy adults consuming fat only (0.88g/kg, 560kcal), shows Glucagon levels increase two fold at 120 minutes post-ingestion, and remain at that level until 300 minutes (9). This point is very important, because as shown in the above insulin graph, the insulin does peak at 30 minutes, but then drops close to baseline by 300 minutes.

Therefore, glucagon will take the predominant role in FFA oxidation, liver glycogenolysis, and potentially Gluconeogenisis after 120 minutes. This research stopped and 300 minutes, therefore it's difficult to know how long this elevation would have lasted.



As discussed earlier and shown in the graph below from the same study, the secretion on intact GIP from the K-Cells of the intestine, due to dietary FFA plays a role in Glucagon secretion. Interestingly you can also see the late peak of intact GIP is at 120 minutes, the time when Glucagon rises after fat ingestion. However, statistical analysis did not find the relationship between GIP and Glucagon to be resoundingly strong.

A recent review paper has highlighted there are different forms of GIP, some that stimulate insulin secretion and other forms that stimulate Glucagon secretion (15). This may be measurement error, or it speaks to the multitude of complex neural and hormonal signals that regulate Glucagon secretion discussed in the Glucagon section (1,13,15).



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.

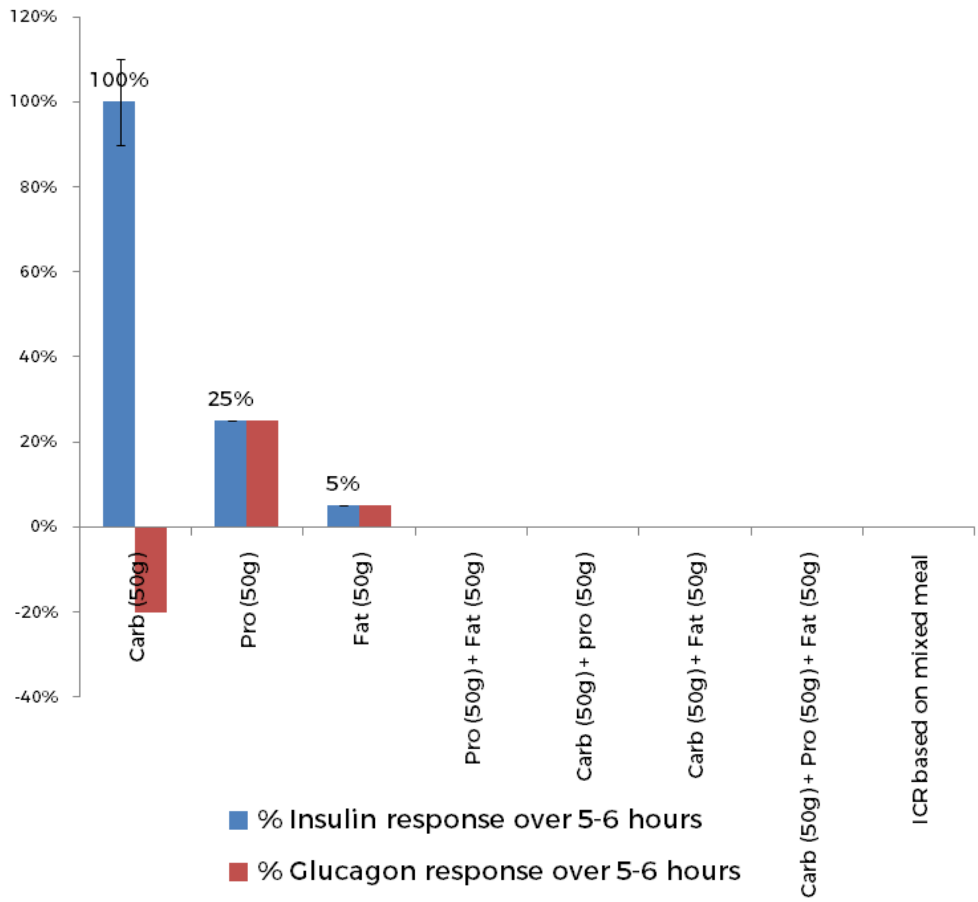
The job of the person with Type 1 Diabetes is to match the amount of insulin the pancreas would release for fat only. Too little leading to potential slight hyperglycaemia, too much leading to hypoglycaemia.

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 above study which compared 560kcal of fat and protein (9). The insulin and glucagon response to fat was only 20% of that 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%.

There is some evidence that corroborates this 5% effect. The Food Insulin Index (FII) group assessed the insulin requirement of 27g of fat from butter and olive oil, and 25g fat from Avocado. They found a 2%, 3%, and 4% insulin response respectively, when compared to glucose over two hours (67).Granted this was only over two hours, but it does paint a consistent picture.

The graph following now shows 50g fat alone has a 5% insulin requirement.



If you counted 0.05g of every gram of fat, you would not be too far off. So simply, for fat only meals this equation would work out the insulin load:

Insulin load = protein (g) x 0.05

The average person with type 1 Diabetes will rarely encounter this issue for a number of reasons:

- They rarely drink 50mls of olive oil just for kicks.
- Even if they do, they are generally eating in the next 3-4 hours so will not encounter the later raise in glucagon, and covered in next meal time insulin dose.

The only people this may apply to are those on a ketogenic diet who consume 70-80% of their energy intake as fat. Generally, they have a slightly higher background rate of insulin to cover this and often do not even bolus at meal times.

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 13.5units 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. The next sections piece the puzzle together.

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