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

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CARBOHYDRATE AND FAT

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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 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 same regulators of B-Cell insulin secretion that have already been detailed are at play here. Most notably the increase GLP-1, and the increased substrates for glycolysis in the B-Cell.

The big difference here is that the increase insulin requirement is not as immediate; it rises more gradually over time. The stomach empties at a constant energy rate (8.4 kJ/min), therefore because fat is very energy dense, gastric emptying will be delayed (33). In healthy individuals the addition of fat to a carbohydrate meal delayed both the glycaemic and insulin responses (34).

GLUCAGON

Put simply, the A-Cells secret a little extra Glucagon to increase FFA oxidation. The same regulators of A-Cell Glucagon secretion that have already been detailed are at play here. Most notably the increased GIP to positively regulate, and increased insulin to negatively regulate.

One study confirmed that when adolescents with type 1 diabetes consumed a high fat high carbohydrate meal, GLP-1 and GIP both increased significantly (32). This suggested the expected physiological changes occur in people with Type 1 Diabetes.

RESEARCH

In one study it was shown that giving eleven type 1 adults an additional 50g of fat to 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 (29). 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 also showed the glucose raising properties of fat when combined with carbohydrate (15). This study compared a 30g carbohydrate, 5g fat, 5g protein meal, with the exact same meal but will an additional 30g of fat added.

30g fat to a 30g carbohydrate 5g fat meal. After 5 hours the glucose level was 3.5mmol/l higher with the additional fat meal. Interestingly the glucose was lower at 90 minutes with the additonal fat meal, presumably due to slower gastric emptying.

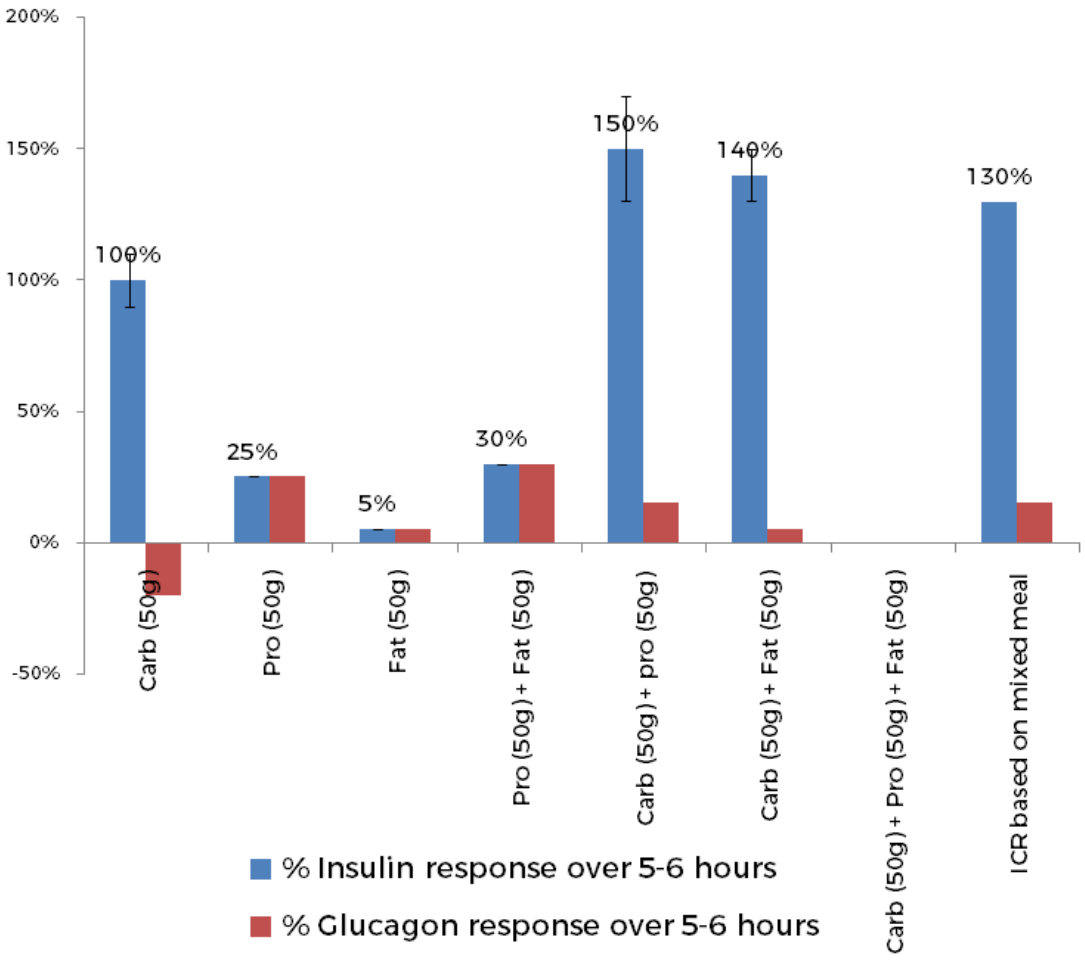
SUMMARY

Very simply, when carbohydrate and fat are consumed both at 50g, 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 sate 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 hypoglycaemia. 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 just 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 dependant on FFA type. This is discussed in the next section, but simply, MUFA require less insulin than SFA when combined with carbohydrate.



The equation for working out the insulin load from fat and carbohydrate:

Insulin load = (fat (g) x 0.4) + (g) carbohydrate

The Type 1 Diabetes research discussed suggests the extra insulin requirement for fat in this combination mostly happens after 210 minutes (15). Therefore, the extra insulin delivered to account for the fat must be given by splitting the injection, or using the extended bolus options on a pump. This would prevent going hypoglycaemic initially, and prevent hyperglycaemia later.

The current suggestion from the latest research review is (73):

- If on a pump, split the total dose 50% upfront and 50% spread over two and a half hours, via an extended wave.
- If on MDI, give 50% upfront and the other 50% in 60-90minutes time.

WHY DO MOST PEOPLE WITH TYPE 1 DIABETES NOT TAKE THIS INTO ACCOUNT?

Most people with Type 1 diabetes who carb count do not see the effect of usual fat intake on blood glucose level for a few reasons:

- As discussed in the carbohydrate and protein section, the ICR is tested on a mixed meal that contains their usual meal amounts of carbs, protein and fat. Therefore, it already accounts for some of the fat effect by covering 130% of the effect of carbohydrate alone.

- They very rarely go more than 3-4hours without eating again, so the effect of rising glucagon 4-8hours after fat is covered by the next meals insulin dose.

Times when they will really notice it, and probably avoid these situations as a consequence:

- Have a very heavy chocolate cake that is very high in carbohydrate and fat. Way above the usual that the ICR caters for.

- A bag of chips (large fried potato chunks!) for the chippy, that is loaded with fat in amounts much higher than the ICR is tested on.

- Going on a donut and biscuit raid. The amount of fat is much more than the usual ICR is ready to deal with.

Bonus: Do all free fatty acids behave the same?

Animal research has shown the more saturated the fatty acid is, the greater the release of Somatostatin, a pancreatic hormone that positively regulates A-Cell release of Glucagon. Therefore, Glucagon is secreted at a higher rate, leading to increase liver glucose output (23).This has been replicated in healthy human subjects where SFA compared to MUFA speeds up the glucose output from the liver two hours after ingestion (27).

The FII (70) analysed the effect of different fatty acids on the insulin requirement over two hours. They showed of the FFAs, MUFA was most strongly correlated with to the lowest insulin response, and SFA the highest insulin response.

It would seem total fat is the most important parameter, but if you were looking to reduce insulin dose, you may want to choose MUFA options, such as olive oil, avocado etc.

The graphical model discussed above has a 10% error bar to account for the FFA type consumed with carbohydrate.

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