Glucose:
Let’s Get Real With the Facts

Think of a stress response, "fight or flight"...

describe the basics of what happens...

Glucose quickly fills muscle cells

BUT

Cortisol delays insulin secretion…?

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If there is less insulin, how does the glucose get into the cell to give you this quick energy?

Do cells need insulin present to uptake glucose?

There are 4 main types of glucose transporters

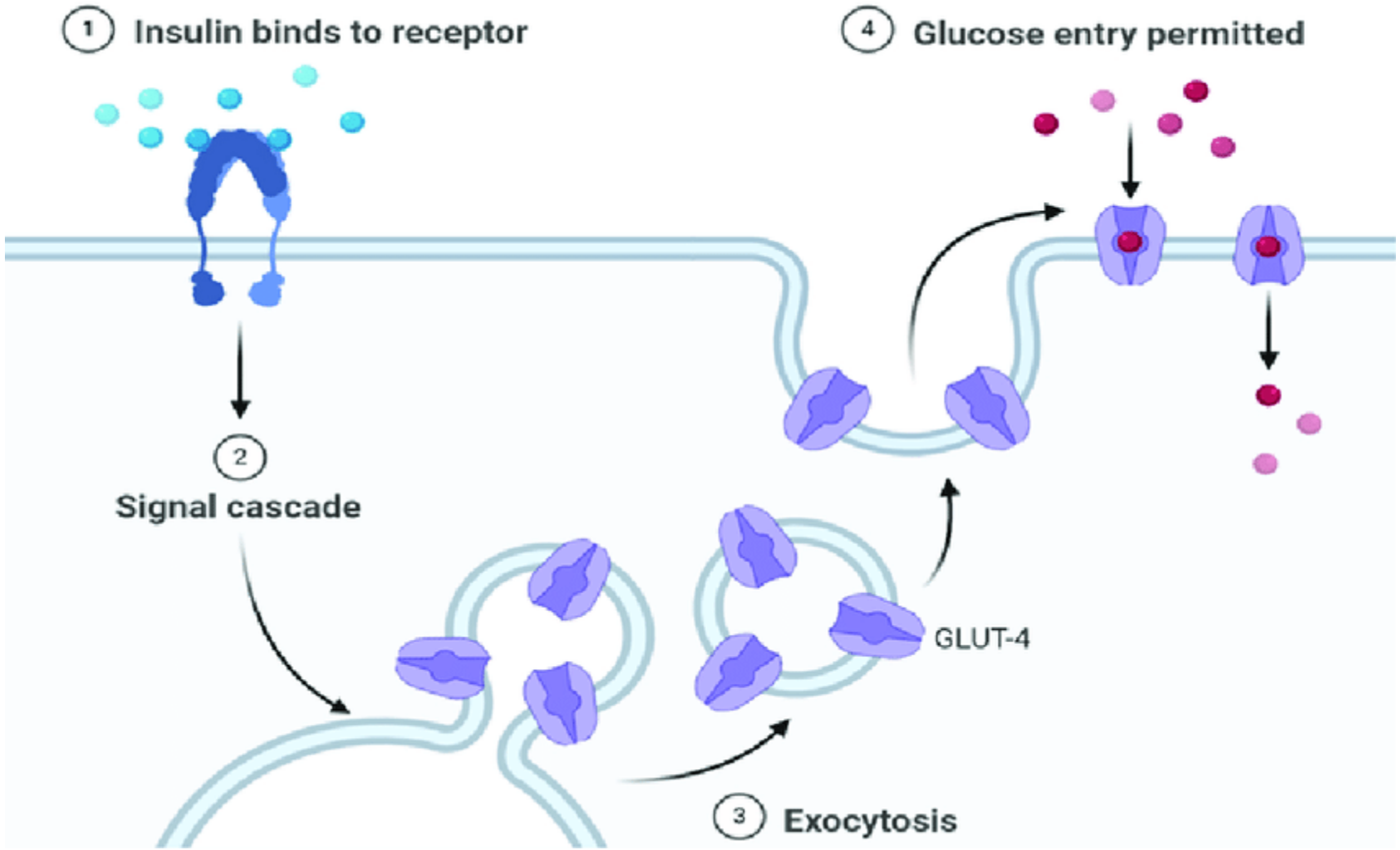
GLUT 1 – A general transporter, used when needed

GLUT 2 – delivers glucose to the digestive tract, liver and pancreas

GLUT 3 – delivers glucose to the CNS and the brain

GLUT 4 – delivers glucose to the heart, muscles and fat cells

GLUT 4 – uses insulin to deliver glucose to the heart, muscles and fat cells


1 Insulin binds to receptor
2 Signal cascade
3 Exocytosis
4 Glucose entry permitted
GLUT-4

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Insulin is not required for GLUT1, GLUT2 or GLUT3-mediated glucose transport and insulin is not needed for glucose transport into most brain cells.

Remember, insulin's job is energy storage, not glucose lowering, although it can and does assist with that in heart, muscle and fat cells

GLUT 2 is also insulin independent since:

glucose enters beta cells via GLUT 2

to make the ATP required

to close the K+ channel

which is required to depolarize the membrane

for Ca+ channels to open and Ca+ to enter the cell

to bring about insulin release.

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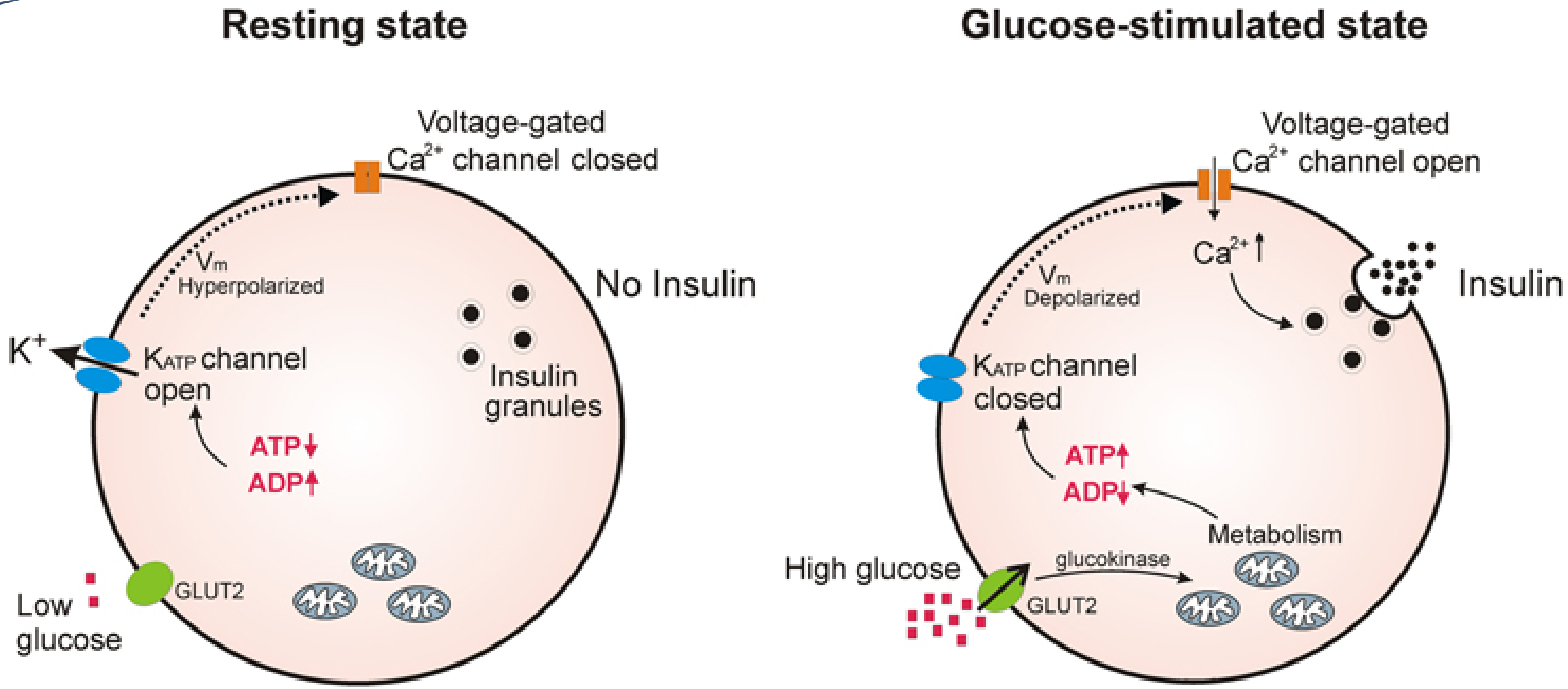
Beta Cells



GLUT 2 to make the ATP required to close the K+ channel which is required to depolarize the membrane for Ca+ channels to open and Ca+ to enter the cell to bring about insulin release

GLUT 4 – delivers glucose to the heart, muscles and fat cells but **it is still not fully insulin dependent**…

So how can we get glucose immediately into the cell without insulin and still using GLUT 4?

Muscle contractions (exercise) pull GLUT 4 vesicles to the membrane for instant access to glucose and this in turn supports insulin sensitivity.

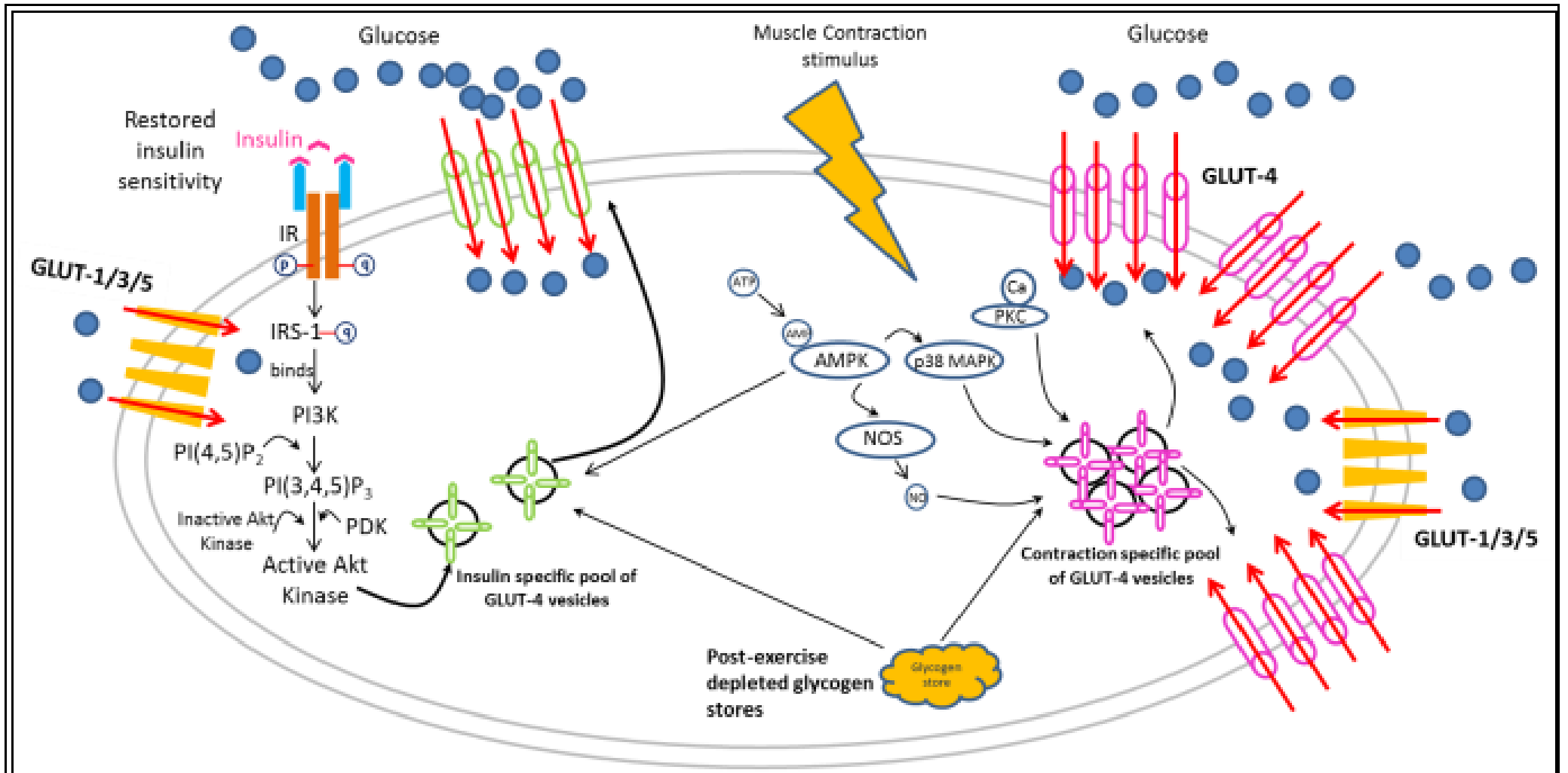


Figure 3: Exercise-induced muscle contraction stimulates GLUT4 translocation and increases glucose transport via an insulin-independent pathway in both normal and insulin resistant condition. Depletion of intracellular glycogen stores increases insulin sensitivity upto 48 hrs. post-exercise.

Now, how is this involved with an HTMA?