

Gestational diabetes (GDM) & vitamin D deficiency

GDM is a form of diabetes that occurs during pregnancy. It is diagnosed when higher than normal blood glucose levels first appear during pregnancy, and usually occurs around the 24th to 28th week of pregnancy. While maternal blood glucose levels usually return to normal after birth, there is an increased risk for the mother developing type 2 diabetes in the future. The baby may also be at risk of developing type 2 diabetes later in life.

up to **1 in 10 PREGNANT WOMEN** in Australia develop **GDM**¹

for most women **GDM disappears after birth**

up to **50%** GDM sufferers develop **TYPE 2 DIABETES** within 5 years⁴

LOW VITAMIN D associated with development of **GDM**²

over **3 in 5 PREGNANT WOMEN** in Australia have **INSUFFICIENT VITAMIN D**³

WHAT HAPPENS IN GDM⁴⁻⁶

- Dietary carbohydrates are broken down into glucose and absorbed into the bloodstream.
- The pancreas produces insulin.
- Various factors interfere with the action of insulin leading to increased levels of glucose in the bloodstream.
- Increased supply of glucose causes foetus to produce more insulin.
- High insulin levels in foetus promote increased growth and fat storage leading to macrosomia.
- Short and long-term consequences:

Maternal consequences:

 - delivery complications
 - increased risk of developing type 2 diabetes.

Foetal consequences:

 - ongoing increased insulin production
 - increased risk of childhood diabetes
 - increased risk of developing type 2 diabetes.

VITAMIN D AND INSULIN SENSITIVITY⁷⁻¹¹

THE ROLE OF VITAMIN D IN PANCREATIC BETA CELLS

ANTI-INFLAMMATORY

- down-regulates fas-induced inflammatory cytokine production (e.g. NFkB, IL-12, IL-2, TNF-α)
- protects against beta cell destruction

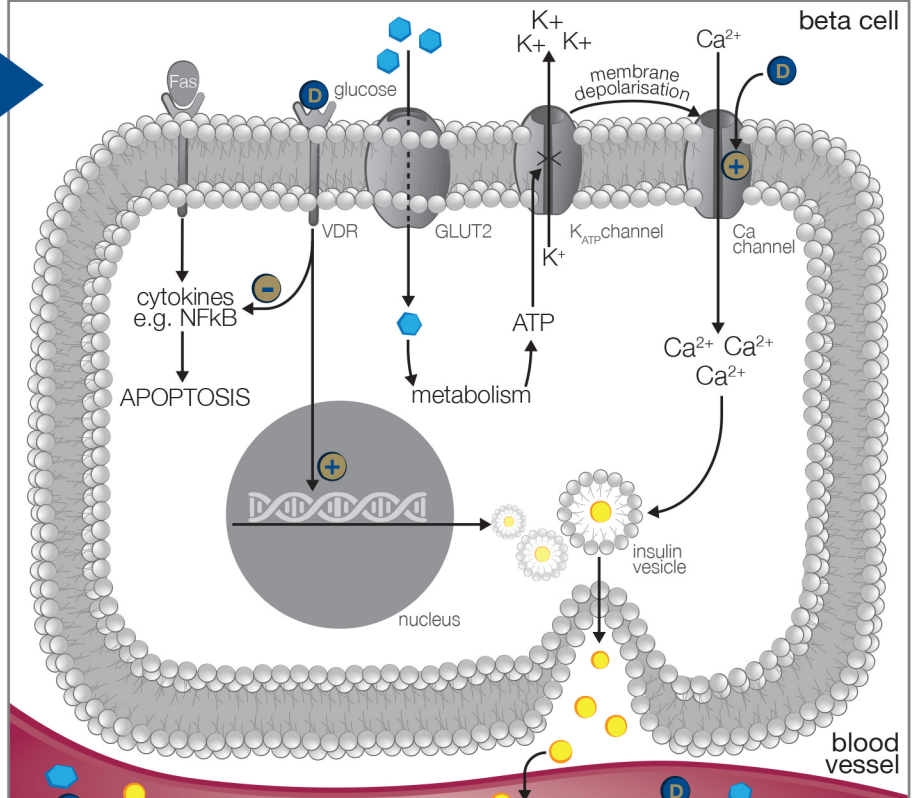
IMPROVES INSULIN PRODUCTION

- stimulates nuclear vitamin D receptors which up-regulate insulin transcription

IMPROVES INSULIN SECRETION

- upregulates calcium-dependent insulin secretion via regulation of:
 - extracellular calcium
 - calcium flux through beta cells
 - intracellular calcium pool
 (hypocalcaemia can lower glucose-stimulated insulin secretion)

DEFICIENCY ASSOCIATED WITH INHERITED GENE POLYMORPHISMS



THE ROLE OF VITAMIN D IN PERIPHERAL TISSUE CELLS

IMPROVES GLUCOSE UTILISATION IN CELL

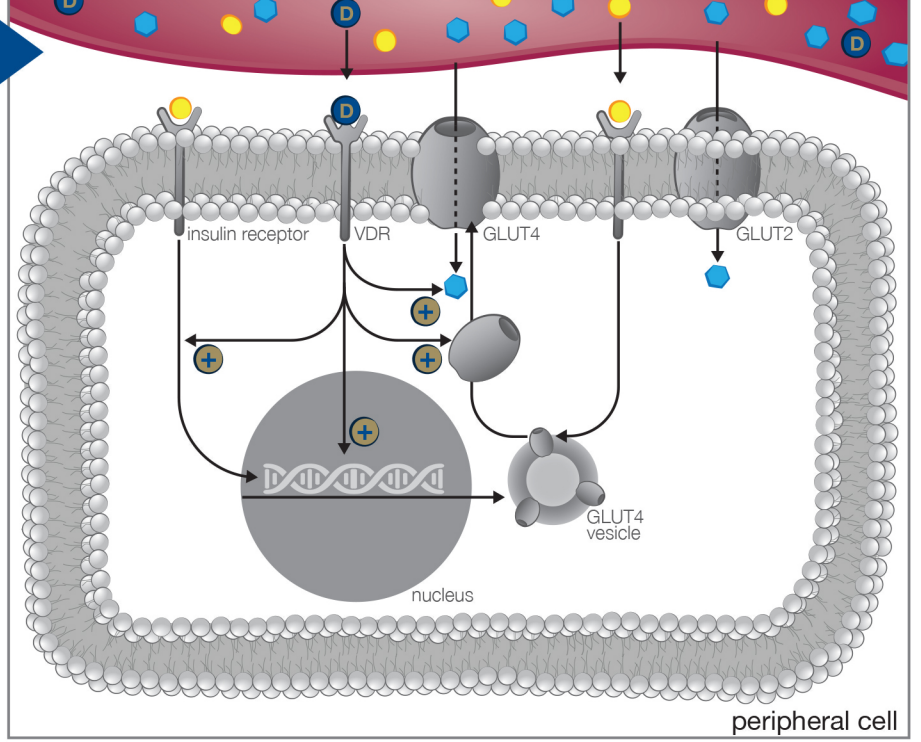
IMPROVES GLUCOSE UPTAKE

- stimulates nuclear vitamin D receptors which up-regulates GLUT4 production
- up-regulates translocation of GLUT4 from intracellular vesicles to plasma membrane

IMPROVES INSULIN FUNCTION

- improves insulin receptor gene expression
- improves insulin-responsive GLUT4 translocation

K ⁺ : potassium	● insulin
Ca ²⁺ : calcium	● glucose
VDR: vitamin D receptor	● vitamin D
ATP: adenosine triphosphate	⊕ up-regulation
NFkB: nuclear factor-kappaB	⊖ down-regulation
TNF-α: tumour necrosis factor-alpha	IL: interleukin
Fas: a cell surface receptor of the TNF family	GLUT: glucose transporter



VITAMIN D AND INFLAMMATION⁷

The activation of inflammatory pathways in cells interferes with proper insulin signalling. Vitamin D down-regulates inflammatory cytokine production from macrophages and inhibits expression of NFkB.