Diabetes Management and the Transplant Patient

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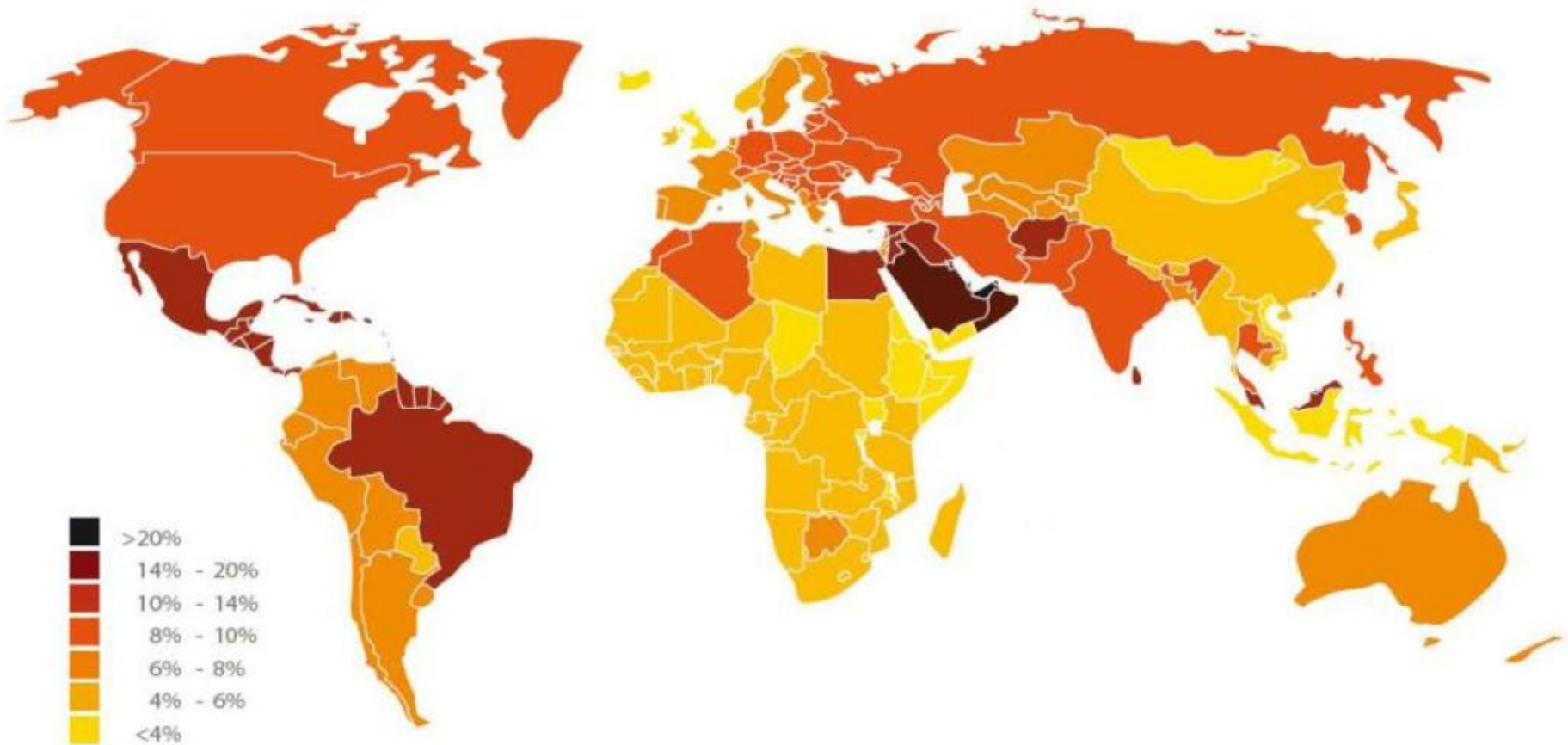
Estimated World Prevalence of Diabetes: 2025

Prevalence estimates of diabetes, 2025



DIABETES IN THE U.S

A US REPORT CARD

DIABETES

38 Million

38 million people **have diabetes**

That's about **1 in every 10** people

1 in 5 people **don't know they have it**

PREDIABETES

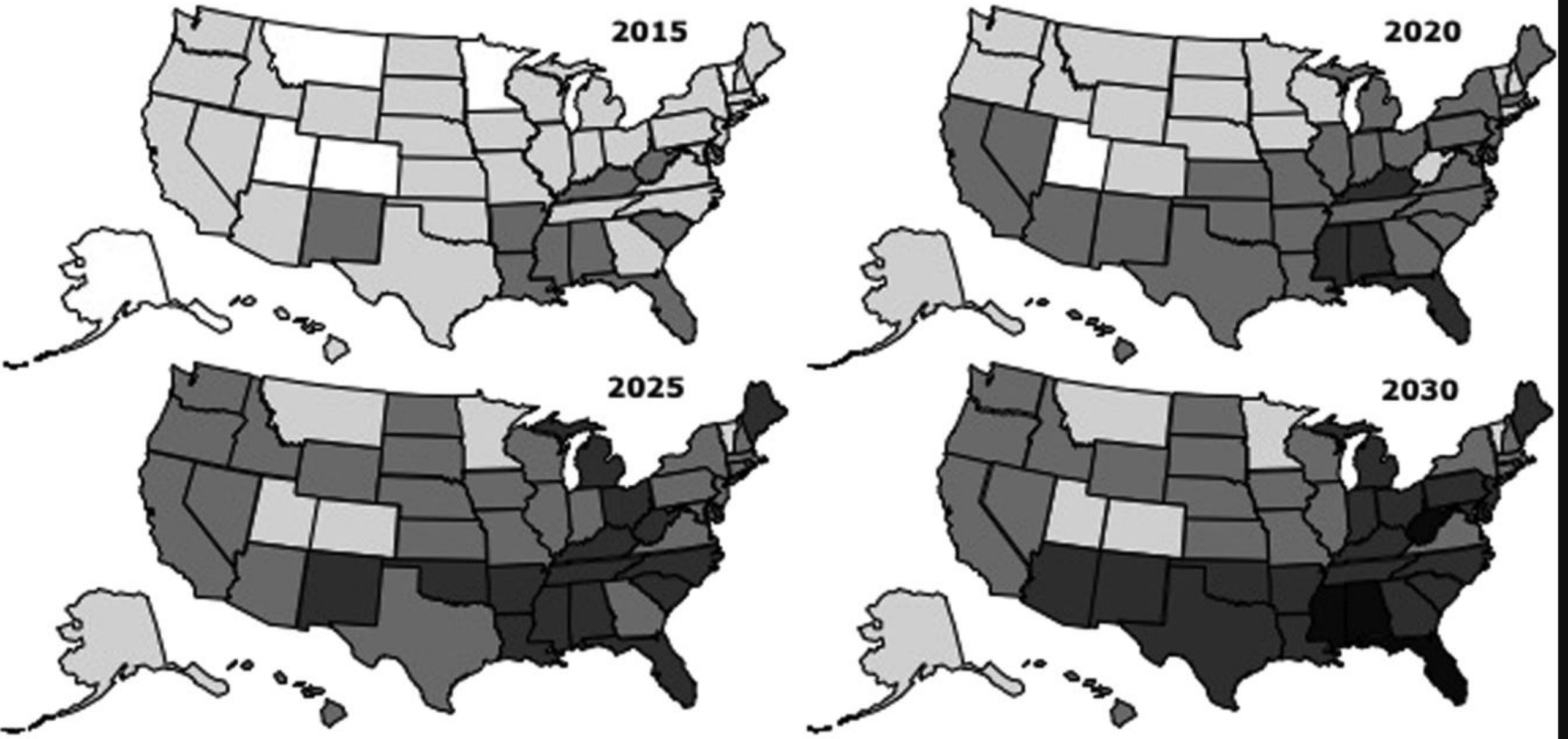
98 Million

98 million American adults—**more than 1 in 3** —have prediabetes

More than 8 in 10

adults with prediabetes **don't know they have it**

Increasing Prevalence of Diabetes



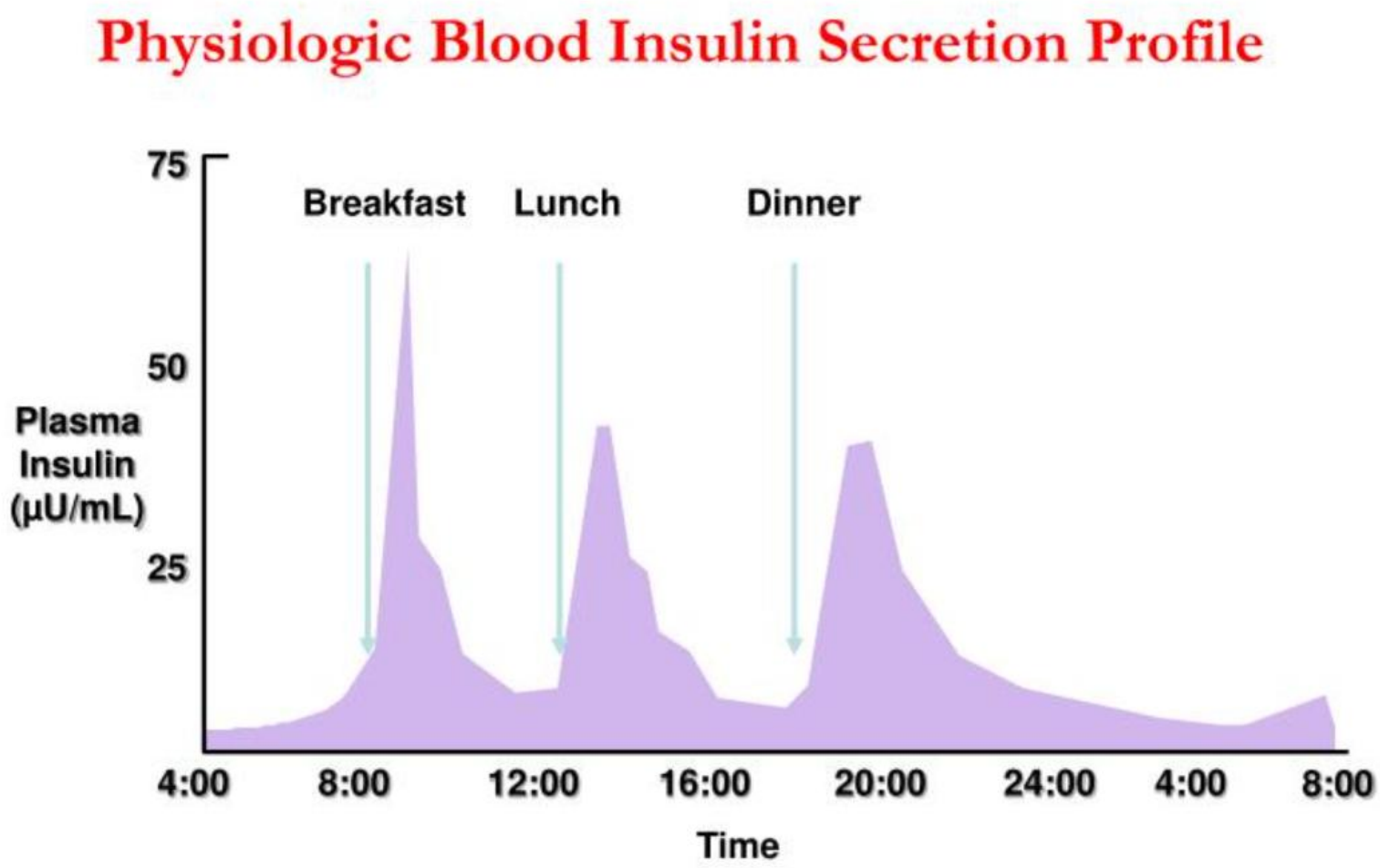
Endocrinology

16,181 licensed Endocrinologists in the US

Vanderbilt University Medical Center: Division of Endocrinology

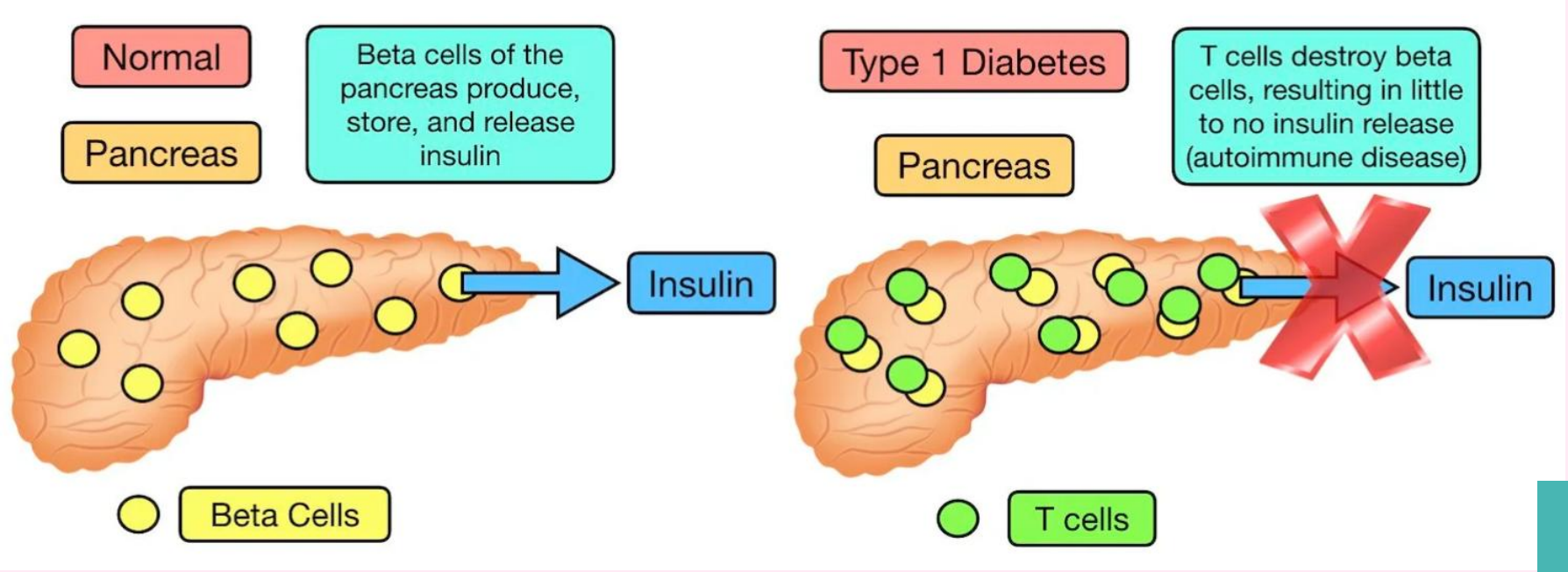
- 24 clinical Endocrinologists
- 24 APPs
  - 10 Inpatient
  - 9 Outpatient
  - 5 Hybrid

Normal Insulin Physiology



Adapted from White JR, Campbell RK, Hirsch I. Postgraduate Medicine. June 2003;113(6):30-36.

Type 1 Diabetes

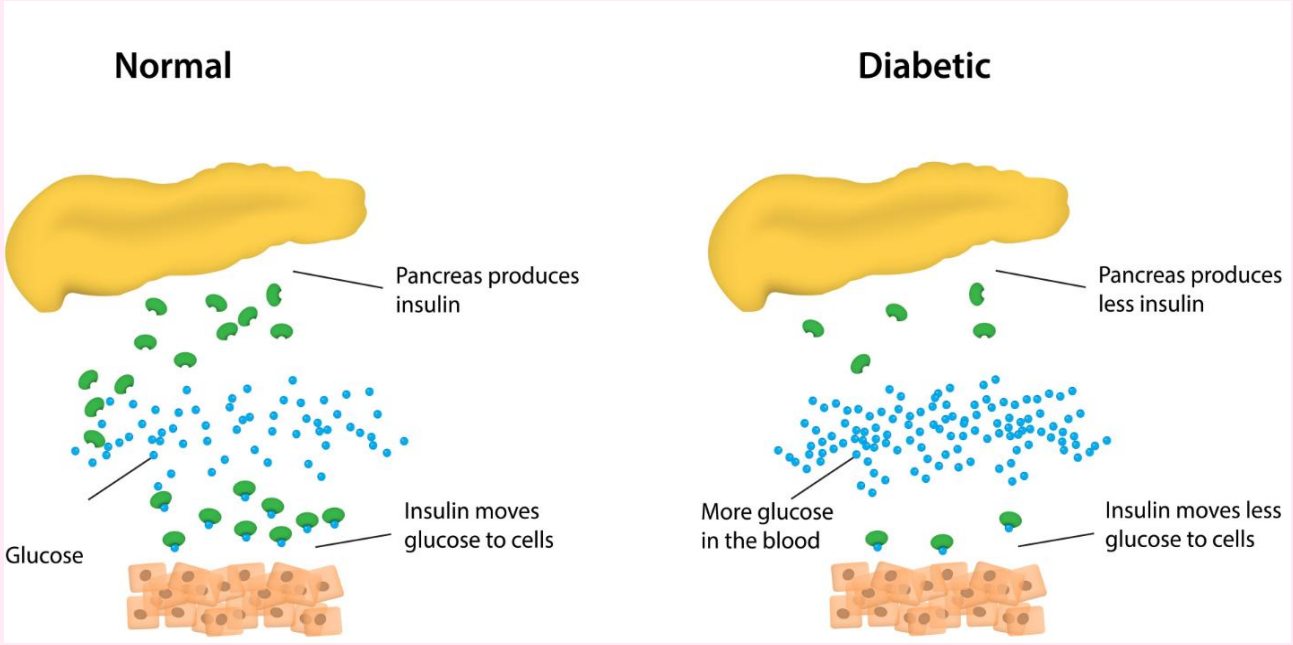


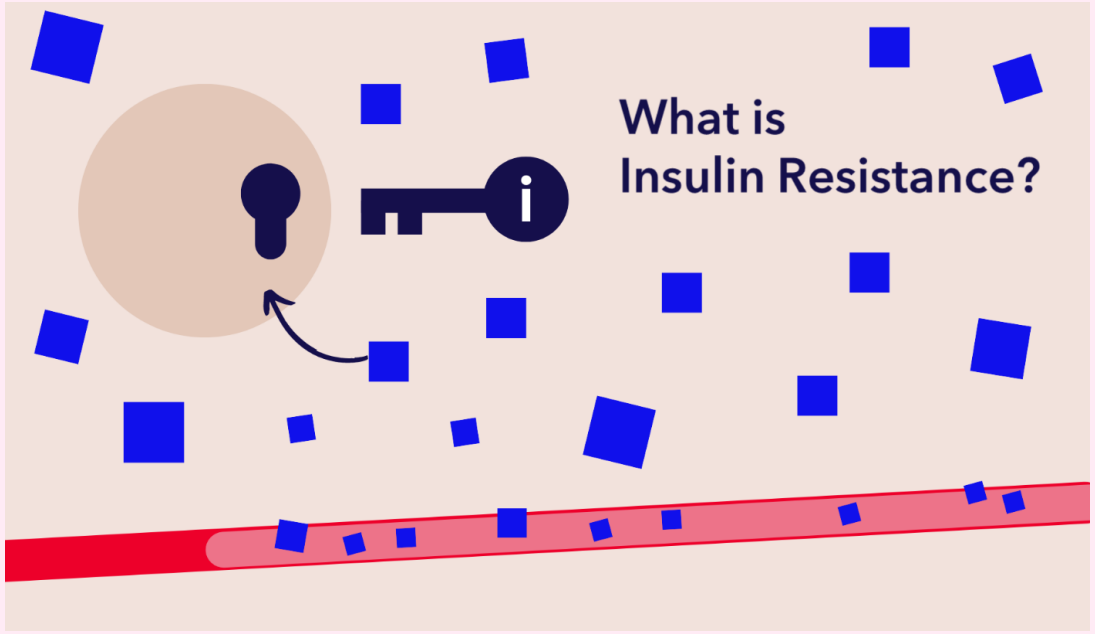
Autoimmune

Lifelong insulin treatment

Type 2 Diabetes

Insulin resistance + deficiency





Risk Factors:

- Family history
- Overweight/ Obesity
- African American, Hispanic, Asian, Native American, Pacific Islanders

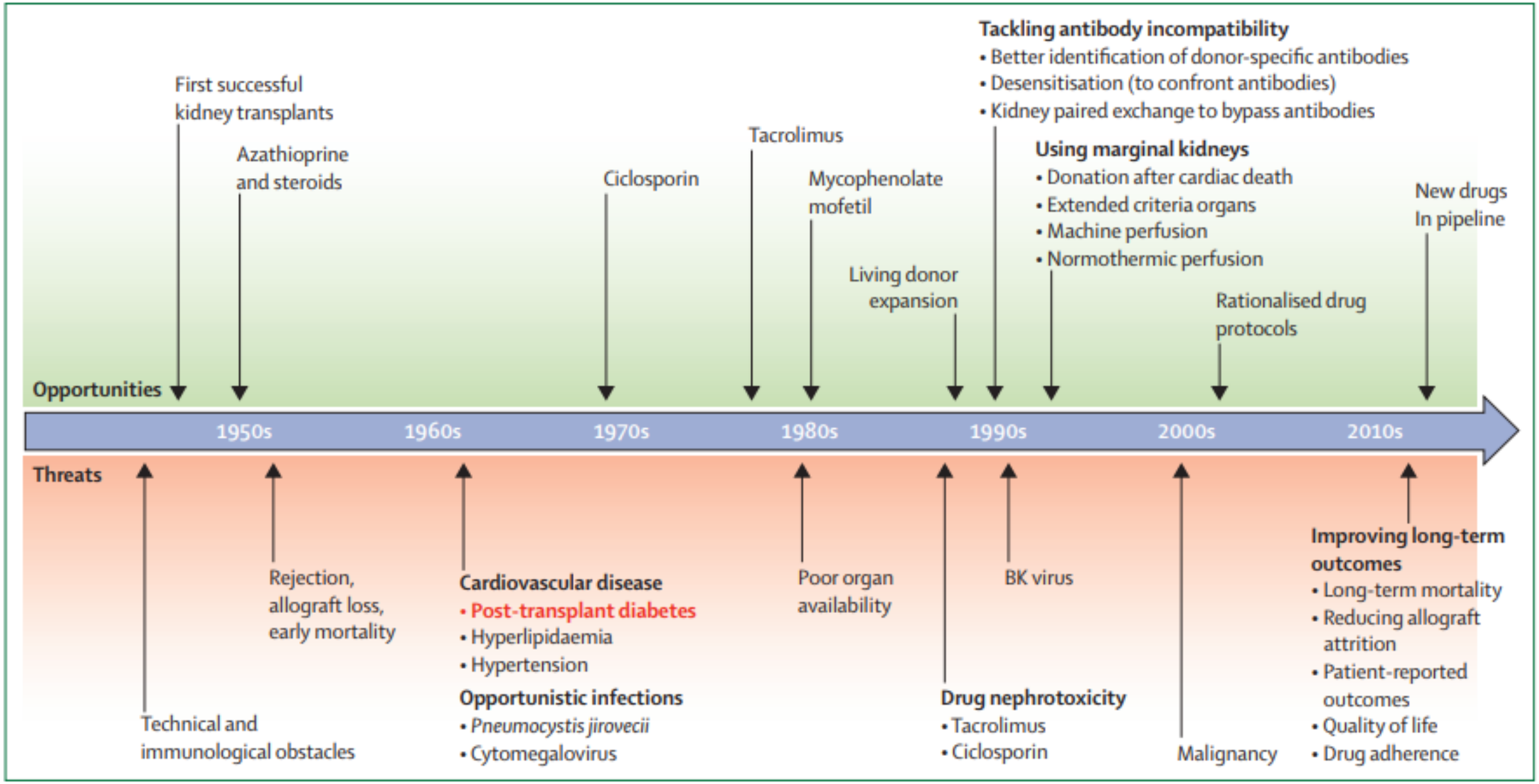


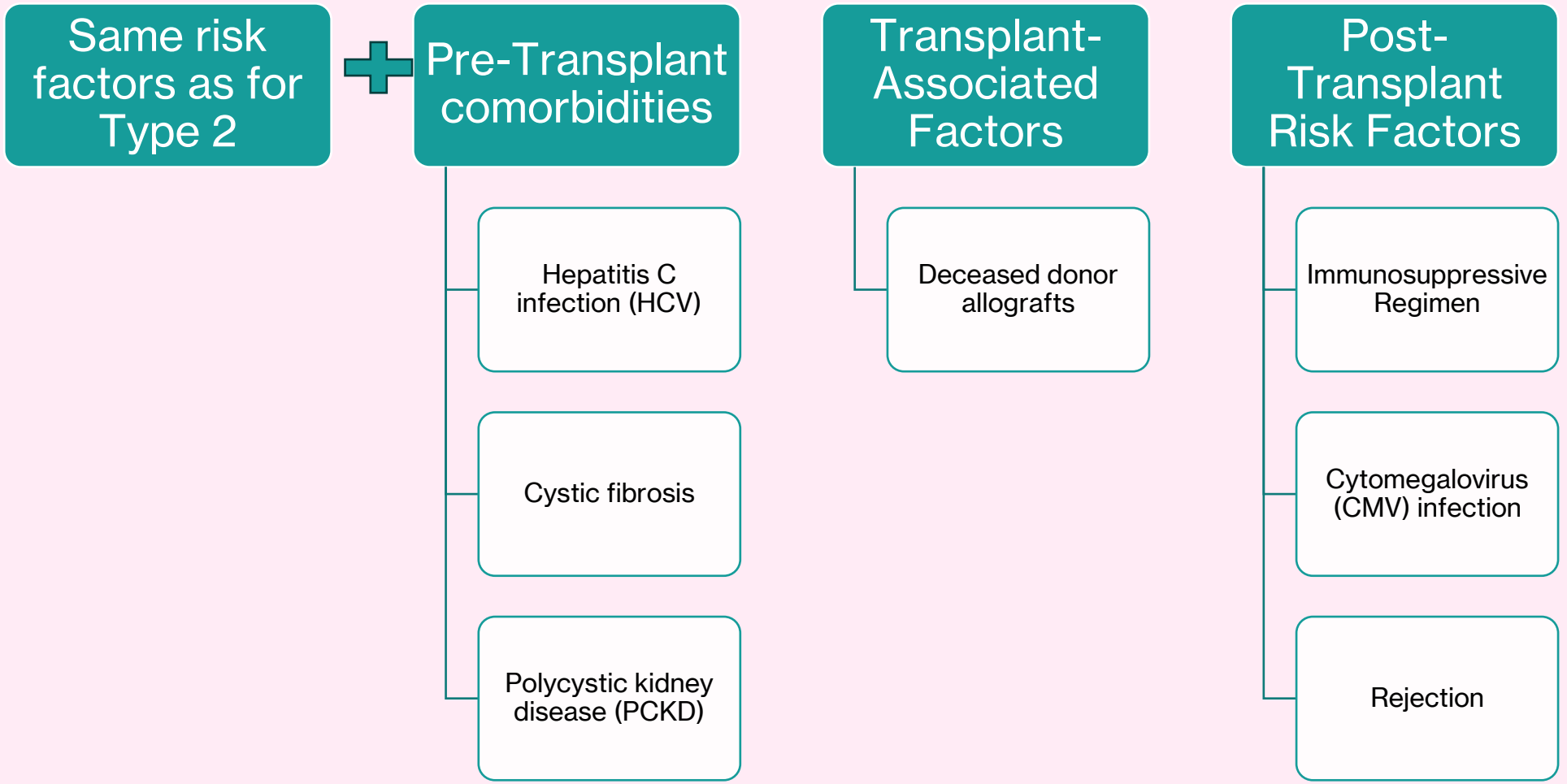
Figure 1: **Timeline of kidney transplantation, threats, and opportunities that have contributed to changes in practice**

The important take home message from this timeline is the need to understand the importance of post-transplant diabetes in view of competing risks and changes in clinical practice over time.

Organ	Incidence
Kidney	10-20%
Liver	30-40%
Heart	20-30%
Lung	20-40%

Incidence of PTDM

Post Transplant Diabetes Mellitus (PTDM)



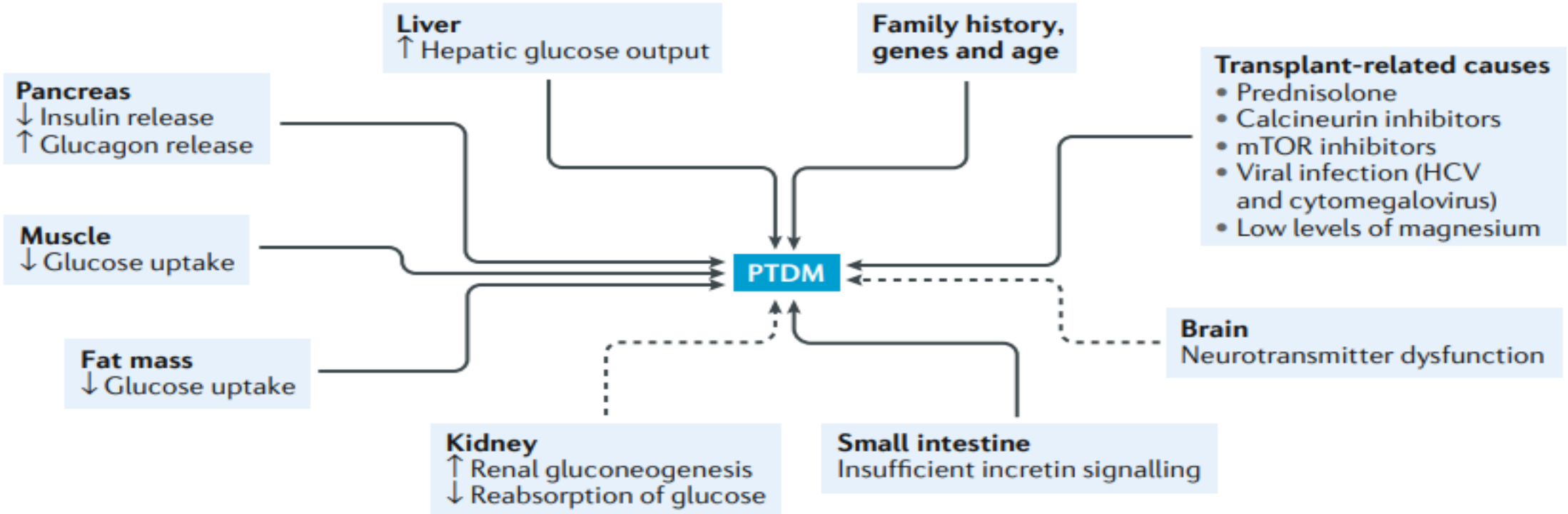


Fig. 1 | **Acknowledged contributors of hyperglycaemia in PTDM.** Common pathways in type 2 diabetes mellitus and post-transplant diabetes mellitus (PTDM) are impaired insulin release and impaired suppression of glucagon release. These effects are not fully compensated by glucagon-like peptide 1 (GLP1) release from the intestinal tract. Insulin-stimulated glucose uptake is reduced in muscle cells and adipocytes, and insulin-mediated suppression of hepatic glucose output is also reduced. In type 2 diabetes mellitus, it is documented that both renal gluconeogenesis and tubular reabsorption of glucose are increased, and crosstalk between insulin, the brain and systemic metabolism is also present. These mechanisms might also be operative in PTDM, but this has not yet been documented. Known contributors of hyperglycaemia in PTDM are shown with bold arrows. Broken arrows depict contributing organs in type 2 diabetes mellitus that have not yet been examined in PTDM. HCV, hepatitis C virus; mTOR, mechanistic target of rapamycin.

Smokey

- 56-year-old male
- Type 2 DM x 15 years
- HTN, HLD
- Diabetic nephropathy -> ESRD on HD x 3 years
- DDKT- received Methylprednisolone 500mg in OR
- Steroid taper:
  - POD 1: Methylpred 250mg
  - POD 2: Methylpred 125mg
  - POD 3: Prednisone 20mg daily

Mr. Commodore

- 65-year-old male
- Type 2 DM
- NICM, HErEF (on home Milrinone), HTN, CAD, CKD III, OSA, MDD, GERD, Iron Deficiency Anemia, Glaucoma, Prostate Cancer (now in remission)
- OHT
- Steroid Course
  - Methylprednisolone 125mg q8hours x 2 doses
  - Methylpred 100mg x 1
  - Methylpred 80mg x 1
  - Methylpred 60mg x 1
  - Methylpred 40mg x 4
  - Prednisone 30mg daily until biopsy

Insulin drip for the first 5 days after transplant

Pre-Transplant

Diagnosing PTDM

- Day 0-45: do not diagnose PTDM
- Day 46-365:
  - Oral Glucose Tolerance Test (OGTT)
    - Fasting glucose 126mg/dl or > **and/or**
    - 2-hour plasma glucose 200mg/dl or >
  - Fasting glucose 126mg/dl or >
  - Random glucose 200 or > WITH SYMPTOMS
  - Hemoglobin A1C 6.5% or > (may underestimate PTDM if used <1 year post-transplant)
- Greater than 1-year post-transplant
  - OGTT
  - Hemoglobin A1C
  - Fasting glucose/ Random glucose

What Alters A1C

- Hematologic conditions
  - Anemia
  - Accelerated erythrocyte turnover
  - Thalassemia
  - Sickle cell disease
  - Reticulocytosis
  - Hemolysis
- Physiologic States
  - Aging
  - Pregnancy
- Drugs/Medications
  - Alcohol
  - Opioids
  - Vitamin C
  - Vitamin E
  - Aspirin
  - Erythropoetin
  - Dapsone
  - Ribavirin
- Disease States
  - HIV infection
  - Uremia
  - Hyperbilirubinemia
  - Dyslipidemia
  - Cirrhosis
  - Hypothyroidism
- Medical Therapies
  - Blood transfusion
  - Hemodialysis
- Miscellaneous
  - Glycation rate
  - Protein turnover
  - Race and ethnicity*
  - Laboratory assay
  - Glycemic Variability
  - Smoking
  - Mechanical heart valves
  - Exogenous testosterone?

Fructosamine

- Glycated albumin; reflects average glucose levels over the preceding 2-3 weeks

- A1C= 0.017 x fructosamine + 1.61

Table 1.

Correlations Between Estimated Average Glucose, A1c, and Fructosamine[1,9,4]

Glucose (mg/dl)	A1c (%)	A1c (mmol/mol)	Fructosamine (µmol)
97	5	31	131
126	6	42	203
154	7	53	273
183	8	64	345
212	9	75	417
240	10	86	487
269	11	97	559
298	12	108	631

A1c hemoglobin A1c

Steroids

1. Decrease insulin sensitivity
2. Increase hepatic gluconeogenesis
3. Increase appetite -> weight gain
4. Effect on glucose is dose dependent

Calcineurin Inhibitors (CNIs)

Tacrolimus > Cyclosporin

Increased islet cell apoptosis

Decreased beta cell mass

Mammalian Target of Rapamycin Inhibitors (mTORI)

- Sirolimus
  - Impaired insulin secretion
  - Reduction in insulin signaling

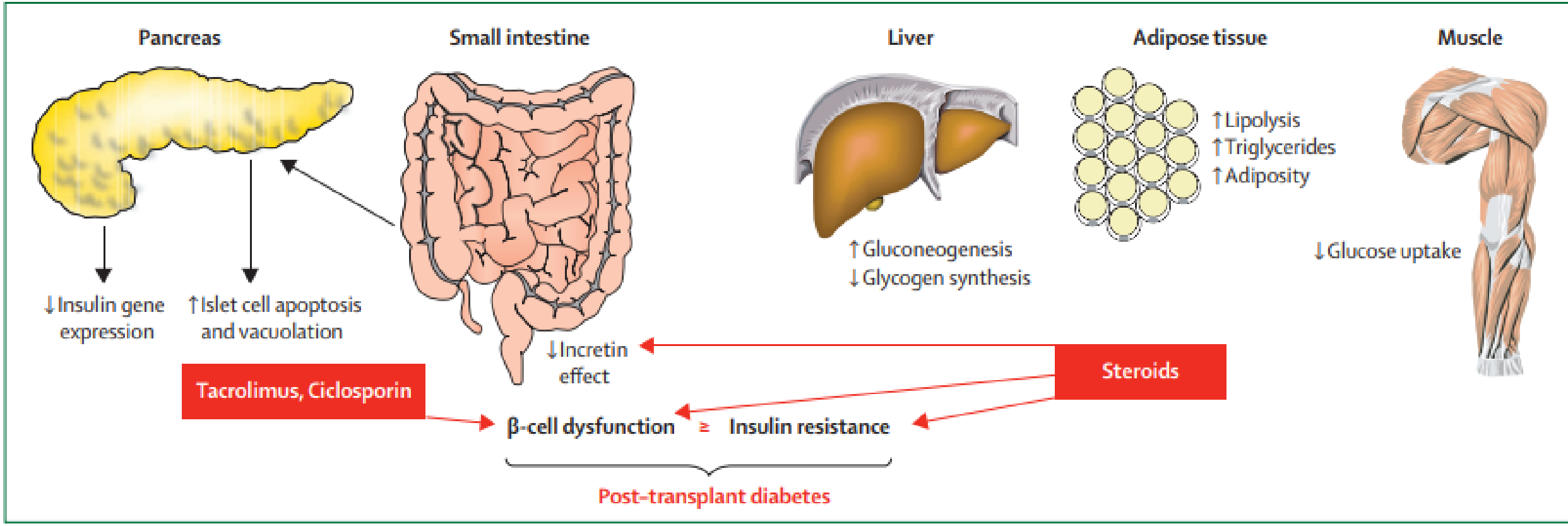


Figure 2: **Pathophysiology of post-transplant diabetes**

Consensus is that post-transplant diabetes is a combination of pancreatic β-cell dysfunction in the presence of insulin resistance, but the relative contributions of these pathophysiological components is debated. Additionally, the relative contributions from other components involved with glucose metabolism including incretin effect and adipocytokines remains unclear. This uncertainty has important implications in the design of targeted interventions to prevent or manage development of post-transplant diabetes. Commonly used immunosuppressants, such as the calcineurin inhibitors (tacrolimus and ciclosporin) and steroids, are implicated in the pathophysiology of post-transplant diabetes as shown.

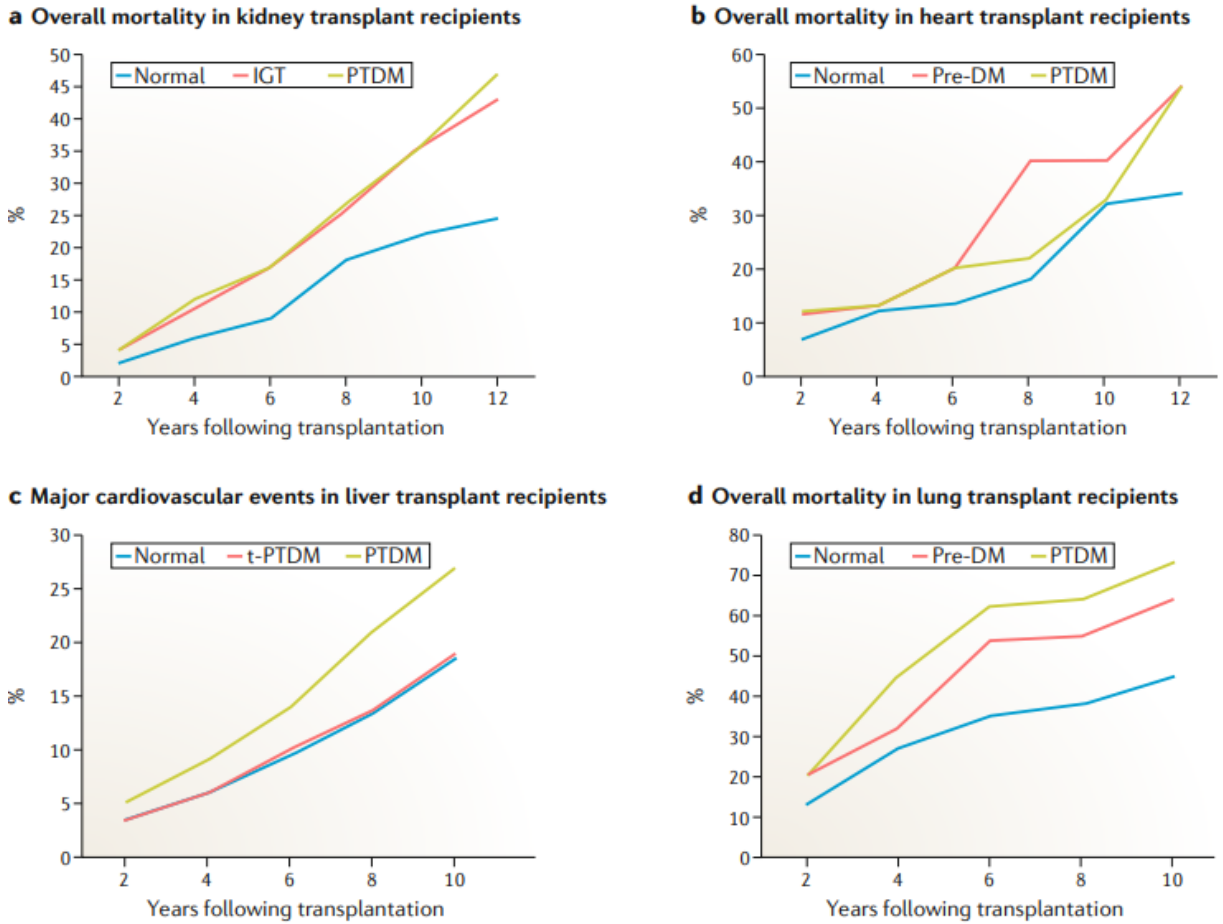


Fig. 5 | **Long-term outcomes of recipients of different organ transplants according to their PTDM status. a** | Overall mortality in recipients of kidney transplants. Mortality is highest in patients with post-transplant diabetes mellitus (PTDM) and in those with impaired glucose tolerance (IGT) compared with patients with normal glucose tolerance. **b** | Overall mortality in recipients of heart transplants. Mortality is highest in patients with pre-existing diabetes mellitus (pre-DM) and in those with PTDM compared with patients with normal glucose tolerance. Data from REF.[12]. **c** | Major cardiovascular events in recipients of liver transplants. Patients with PTDM have a higher incidence of cardiovascular events than patients with normal glucose tolerance or those with transient PTDM (t-PTDM) that reverts within 6 months. **d** | Overall mortality in recipients of lung transplants. The incidence is highest in patients with PTDM and in patients with pre-DM. Part **a** adapted from REF.[22], Springer Nature Limited. Part **c** adapted with permission from REF.[15], Wiley-VCH. Part **d** adapted with permission from REF.[122], Wiley-VCH.

Goals

- Optimize long-term outcomes
- Prevent microvascular and macrovascular complications
- Lessen premature mortality
- Decrease risk of graft loss
- Lower glucose by targeting cause of hyperglycemia
- Avoid interaction with immunosuppression meds

Glucose targets

- During hospitalization: 100-180mg/dl
- At home:
  - Fasting: 80-130mg/dl
  - Post-prandial: <180mg/dl
- Continuous Glucose monitor (CGM):
  - > 70% time in range (TIR)
    - Target range is 70-180mg/dl
- Hemoglobin A1C <7%

Treatment of PTDM

Continue education

Nutritional counseling

Exercise

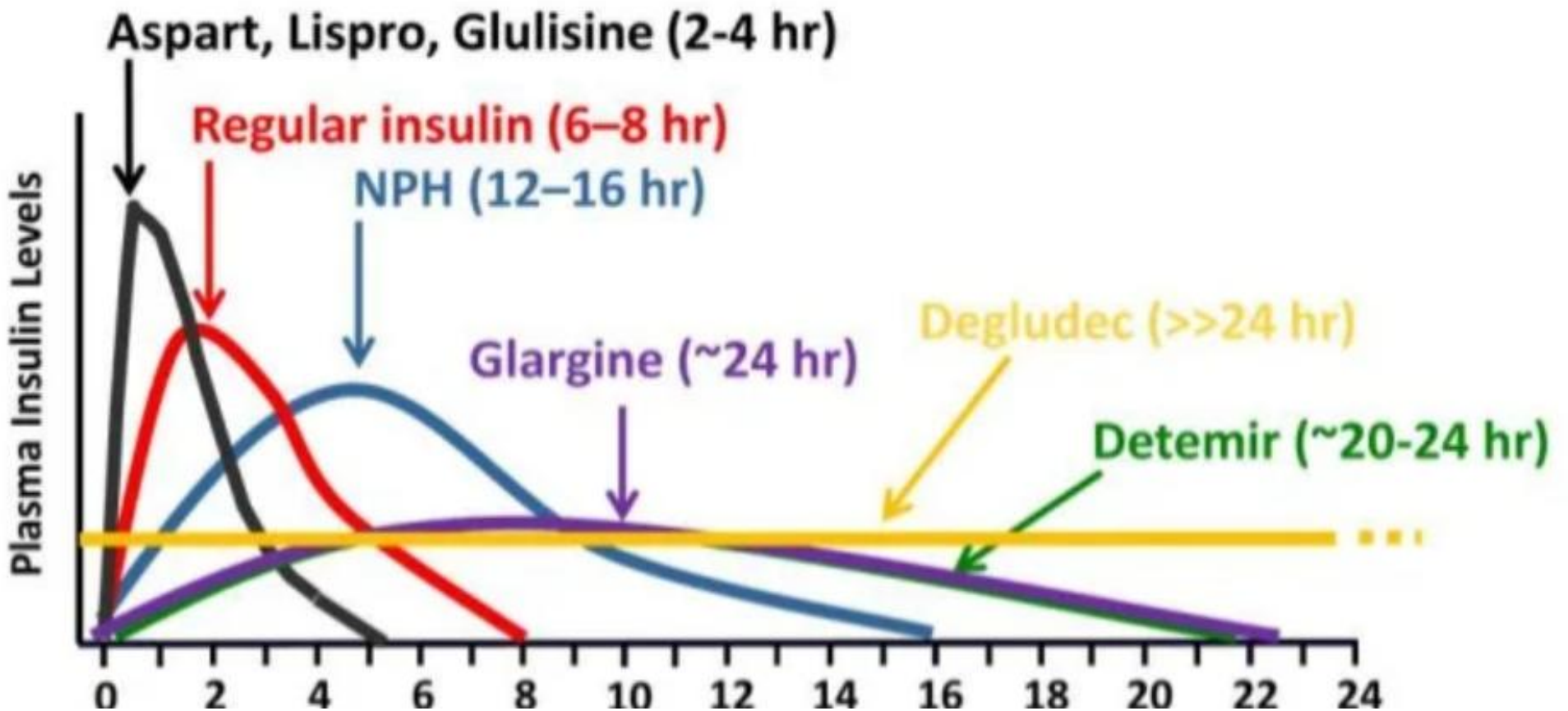
Insulin

Consider other medications

Doses of immunosuppression medications are stable

Less than 20 units of insulin/day

Rapid- and Long-Acting Insulin Profiles



Insulin	Onset	Peak	Duration
Ultra-Rapid Acting			
Insulin lispro (Lyumjev™)	10-15 mins	1-2 hrs	4-6 hrs
Insulin aspart (Fiasp®)	10-15 mins	1-2 hrs	4-6 hrs
Rapid-Acting			
Insulin lispro (Admelog®, Humalog®)	15 mins	1-2 hrs	4-6 hrs
Insulin aspart (Novolog®)	15 mins	1-2 hrs	4-6 hrs
Insulin glulisine (Apidra®)	15 mins	1-2 hrs	4-6 hrs
Short-Acting			
Regular (Humulin R, Novolin R)	30 mins	3 hrs	8 hrs
Intermediate-Acting			
NPH (Humulin N, Novolin N)	1-2 hrs	4-8 hrs	12 hrs
Long-Acting			
Insulin degludec (Tresiba®)	1 hr	9 hrs	42 hrs
Insulin detemir (Levemir®)	2 hrs	3-9 hrs	20-22 hrs
Insulin glargine (Basaglar®, Lantus®, Toujeo®, Semglee®)	2 hrs	--	24 hrs

Initiating Insulin

0.3 units/kg: BMI <18.5 or high risk for hypoglycemia

0.4 units/kg: Normal (BMI 18.5-24.9)

0.5 units/kg: Overweight (BMI 25-29.9)

0.6 units/kg: Obesity (BMI >30) or highly insulin resistant

Basal insulin: 50% TDD

Bolus insulin: divide remaining 50% into 3 equal doses before meals

Correction scale:

- Low-dose: Insulin naïve, AKI, ESRD, Type 1 DM
- Moderate-dose: steroids, tube feeds, obesity, or requiring high insulin doses

Smokey- POD 1

- Methlypred 250mg
- Weight: 107.4kg/ BMI 33.69
- Insulin regimen: start 0.6 units/kg TDD
  - 107.4 x 0.6 = 64 units
  - 64/ 2 = 32 units
  - Glargine: 32 units qhs
  - Lispro 32/3 meals= 10 units
  - **Double prandial insulin for steroids
  - Lispro 20 units with meals
  - Correction scale 4 units:50mg/dl for BG >150mg/dl ACHS

Smokey- POD 2-3

Glucose	Lispro
118	20
218	28
186	24
145	0

POD 2
Methylpred 125mg

No change in insulin

POD 3
Prednisone 20mg daily

Decrease Lispro to 16 units with meals
Decrease correction scale to 3 units:50mg/dl for BG >150mg/dl

Glucose	Lispro
124	20
165	24
82	20
110	0

Mr. Commodore

- Transitioned off insulin drip
  - **give basal insulin at least 2 hours before stopping insulin drip
- Methylpred 40mg daily
- Weight: 84.2kg
- BMI 27.81
- Start weight-based insulin dosing
  - 0.3 units/kg TDD
  - Basal: 12 units
  - Prandial: 8 units with each meal
  - Correction scale: 2 units:50mg/dl for BG >160mg/dl ACHS

Mr. Commodore

	10/07 0700 - 10/08 0659						10/08 0700 - 10/09 0659								
Time:	1804	2115	2118	2119	0607	0617	0735	0736	1141	1211	1212	1634	1800	2111	
Glucose (mg/dl)	Graphs cannot display in the current view														
Glucose															
POC glucose		233				197			273			225		249	POC glucose
Blood glucose					172										Blood glucose
Medication Dosing															
insulin glargine,hum.r...			12												insulin glargi...
insulin lispro SUBQ (...	14			4				10		9	12		18		insulin lispro ...
methylprednisolone s...							40								methylpredni...

- Increase Lantus to 15 units
- Increase Lispro to 12 units with meals
- Continue correction scale 3 units: 50mg/dl for BG >150mg/dl
- Diabetes education- including insulin teaching

Mr. Commodore

	10/08 0700 - 10/09 0659				10/09 0700 - 10/10 0659										
Time:	2220	2221	0607	0611	0841	1015	1136	1156	1636	1820	2121	2232	2257	0506	
Glucose (mg/dl)	Graphs cannot display in the current view														
Glucose															
POC glucose				192			139		190		195				POC glucose
Blood glucose			173											122	Blood glucose
Medication Dosing															
insulin glargine,hum.r...	15												16		insulin glargi...
insulin lispro SUBQ (...		6			21			18		21		3			insulin lispro ...
PredniSONE ORAL (...						30		10							PredniSONE...

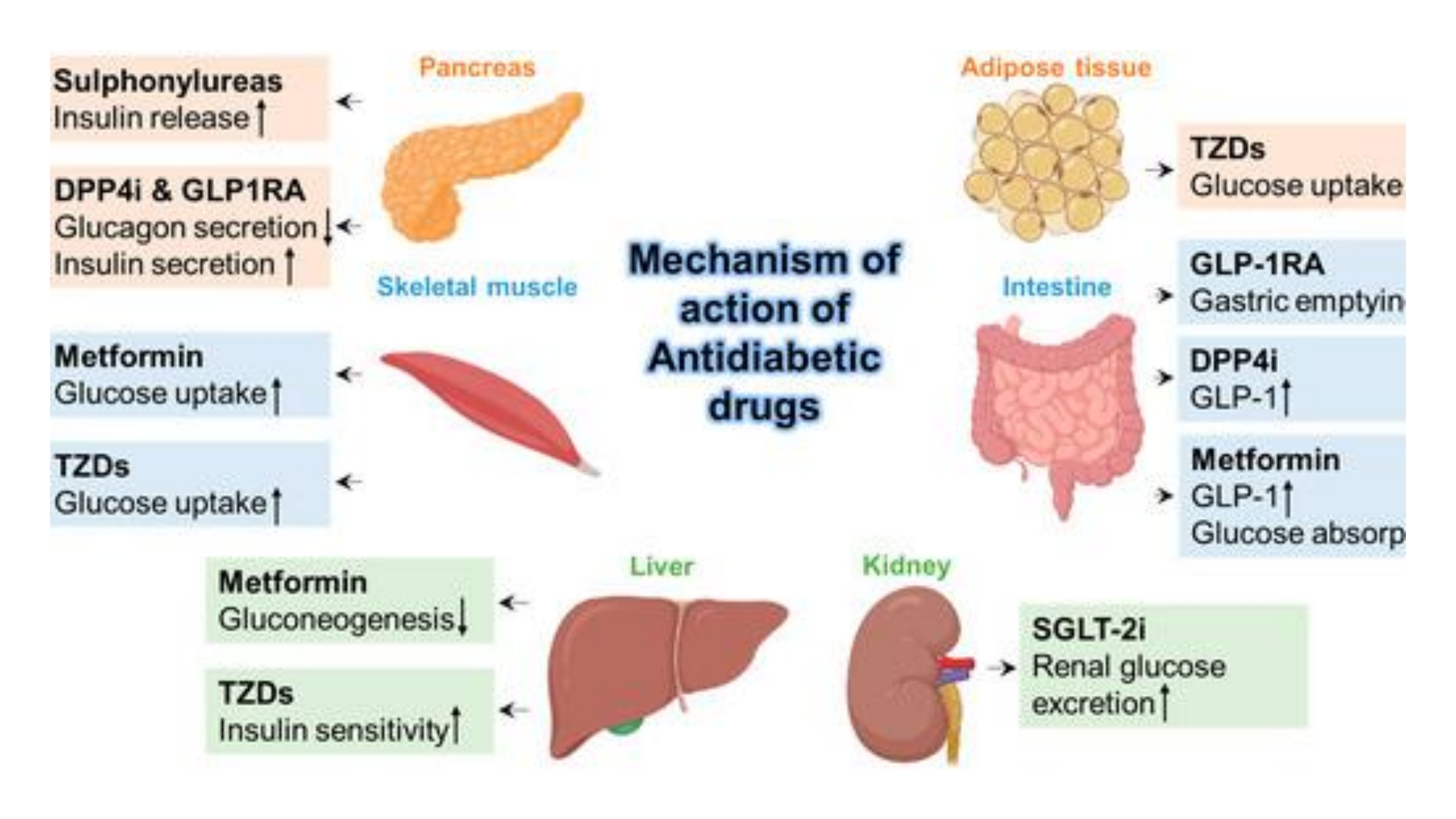
- Prednisone 40mg daily
- Increase Lantus to 16 units
- Increase Lispro to 18 units with meals
- Continue correction scale 3 units: 50mg/dl for BG >150mg/dl
- Diabetes education- including insulin teaching

Mr. Commodore

	10/09 0700 - 10/10 0659											10/10 0700 - 10/11 0659			
Time:	0841	1015	1136	1156	1636	1820	2121	2232	2257	0506	0620	0834	1025	1120	
Glucose (mg/dl)	Graphs cannot display in the current view														
Glucose															
POC glucose			139		190		195				134			284	POC glucose
Blood glucose										122					Blood glucose
Medication Dosing															
insulin glargine,hum.r...									16						insulin glargi...
insulin lispro SUBQ (...	21			18		21		3				18			insulin lispro ...
PredniSONE ORAL (...		30		10									40		PredniSONE...

- Continue Lantus 16 units
- Increase Lispro to 24 units with meals
- Increase correction scale to 5 units: 50mg/dl for BG >150mg/dl

Mechanism of action of Antidiabetic drugs



Agents	Advantages	Disadvantages/Comments
Insulin Sensitizers		
Biguanides (e.g. metformin[1], buformin)	Weight neutral or weight loss, no hypoglycemia, cardioprotection (metformin), low cost	Gastrointestinal intolerance, lactic acidosis with renal impairment[1] Although metformin has been shown to have cardioprotective effect, GLP-1 RA is currently the anti-diabetic agent of choice in high CVD risk patients or in those with known ASCVD
Thiazolidinedione derivatives (e.g. pioglitazone)	Low hypoglycemic risk, does not depend on renal excretion	Fluid retention: worsen CHF, edema (especially with insulin), weight gain, accelerated bone loss, increase fracture risk Contraindicated in NYHA functional classes III-IV CHF or hepatic impairment
Insulin Secretagogues		
Sulfonylureas (e.g. glipizide, glyburide)		Weight gain, edema, hypoglycemia (particularly in elderly and patients with renal impairment) Glyburide may accumulate in renal insufficiency SMZ-TMP coadministration increases hypoglycemia risk (95)
Glinides (e.g. repaglinide, nateglinide)	Glinides: rapid onset and offset. Best suited for patients whose food intake is erratic (best taken before meals, and the dose may be omitted if a meal is skipped)	Weight gain, hypoglycemia (lower risk than sulfonylureas)
Glucagon-like polypeptide-1 receptor agonists (GLP-1RAs)		
Examples: dulaglutide, liraglutide, exenatide (renal dosing)	Weight loss (delayed gastric emptying, early satiety), low risk of hypoglycemia, cardiorenal protection	GI intolerance Risk of pancreatitis Thyroid cancer[2] (FDA black box warning)
Dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonists		
Tirzepatide	Cardioprotection Renoprotection (ongoing clinical research)	Greater weight loss compared with semaglutide
Dipeptidyl Peptidase-4 inhibitors (DPP-4i)		
Examples: vildagliptin, sitagliptin,[3] linagliptin	Weight neutral, low risk for hypoglycemia Possible β-cell preservation	Renal dosing except linagliptin (clinicians should refer to package inserts) Risk of pancreatitis, ketoacidosis
Sodium-glucose cotransporter type 2 inhibitors (SGLT2i)		
Examples: empagliflozin, canagliflozin, dapagliflozin, ertugliflozin	Cardiorenal protection	Glycemic benefit is reduced or even absent when GFR < 45-60 mL/min Avoid or contraindicated[4] (96)

Figure 4.

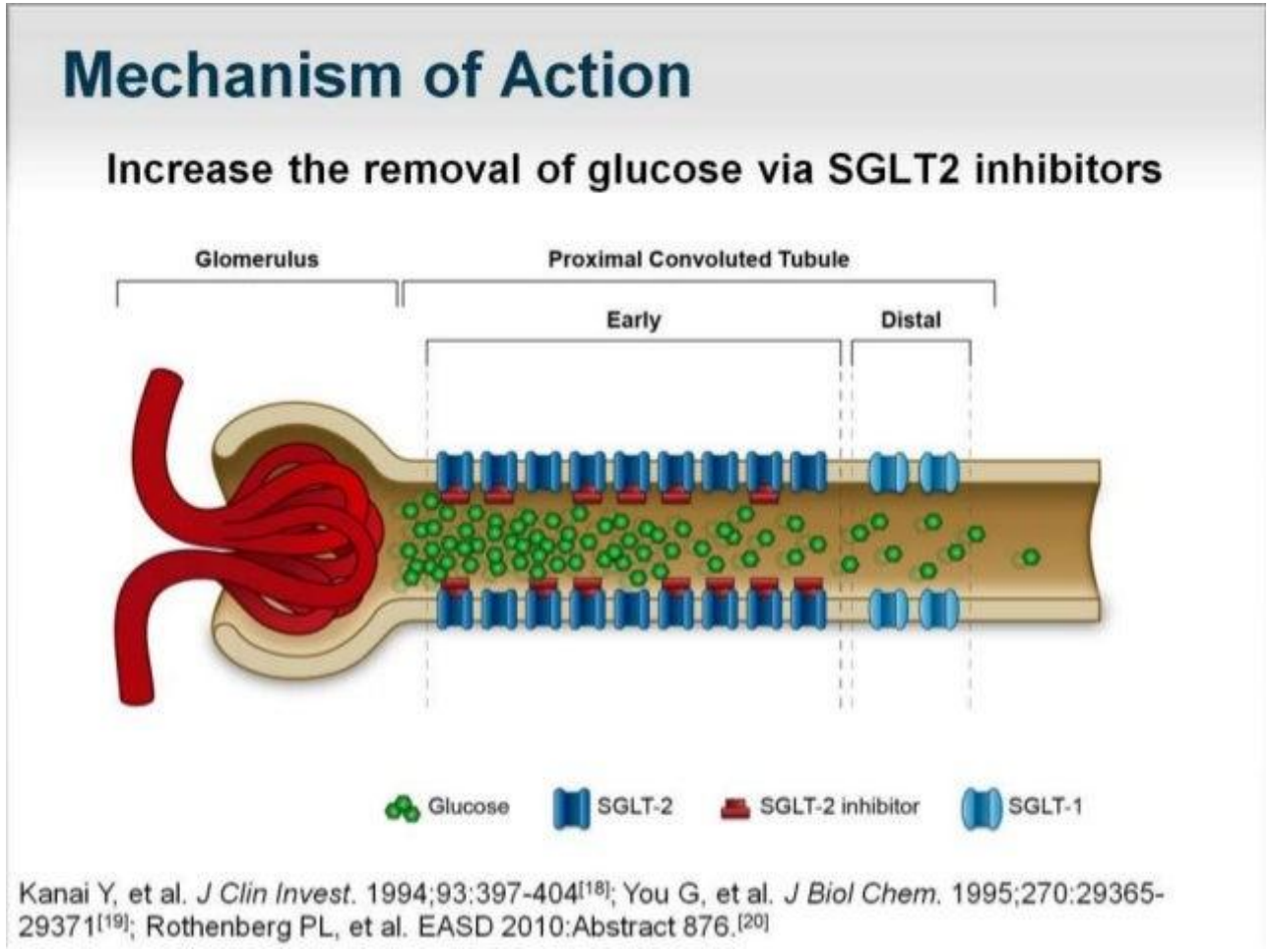
The potential advantages and disadvantages of various classes of antihyperglycemic agents. [1] KDIGO guidelines: Reduce dose if estimated glomerular filtration rate (eGFR) < 45 cc/min/1.73 m^2. Discontinue if eGFR < 30 cc/min/1.73m^2. . [2] Contraindicated in patients with personal history or family history of medullary thyroid cancer or multiple endocrine neoplasia (MEN) type 2. [3] Sitagliptin may prolong QT interval particularly when used with cyclosporine. [4]Recurrent genital mycotic infections, UTIs, pyelonephritis or urosepsis, neurogenic bladder, vesicoureteral reflux, urinary incontinence, indwelling Foley catheter, clean intermittent catheterization, presence of ureteral stent, hypotension or recurrent volume depletion. Others: patients at risk for or with history of lower limb amputation, risk factors for Fournier gangrene, peripheral vascular disease, and recurrent pancreatitis.

Table 3 | Use of hypoglycaemic drugs in patients with PTDM and impaired renal function

Drug class	eGFR 60–90 ml/min/1.73 m²	eGFR 30–59 ml/min/1.73 m²	eGFR 15–30 ml/min/1.73 m²	eGFR <15 ml/min/1.73 m²
Sulphonylureas	Used without dose adjustment	Used with or without dose adjustment; caution for hypoglycaemia	Not generally recommended due to risk of hypoglycaemia	Not generally recommended due to risk of hypoglycaemia
Glinides	Used without dose adjustment	Used without dose adjustment	Should not be used	Should not be used
Biguanides	Used without dose adjustment	Used with or without dose adjustment*	Should not be used	Should not be used
Glitazones (PPAR-γ activators)	Used without dose adjustment	Used without dose adjustment	Used with or without dose adjustment	Should not be used
DPP-4 inhibitors	Used without dose adjustment	Used with or without dose adjustment	Used with or without dose adjustment	Used with or without dose adjustment
GLP-1 analogues‡	Not studied in PTDM	Not studied in PTDM	Not studied in PTDM	Not studied in PTDM
SGLT2 inhibitors§	Not studied in PTDM	Not studied in PTDM	Not studied in PTDM	Not studied in PTDM
Insulin	Used without dose adjustment	Used with or without dose adjustment; caution for hypoglycaemia	Used with or without dose adjustment; caution for hypoglycaemia	Used with or without dose adjustment; caution for hypoglycaemia

*Not approved in the USA for patients with GFR <60 ml/min/1.73 m². ‡Have been used safely in patients with type 2 diabetes and GFR >30 ml/min/1.73 m²; no documentation in PTDM. §Reduced glucose-lowering effect at GFRs <60 ml/min/1.73 m²; no documentation in PTDM. Abbreviations: DPP-4, dipeptidyl peptidase-4; eGFR, estimated glomerular filtration rate; GLP-1, glucagon-like peptide-1; PPAR, peroxisome proliferator-activated receptors; PTDM, post-transplant diabetes mellitus; SGLT2, sodium-glucose linked transporter 2.

Sodium Glucose Transport 2 Inhibitors (SGLT2)



Kanai Y, et al. *J Clin Invest*. 1994;93:397-404[18]; You G, et al. *J Biol Chem*. 1995;270:29365-29371[19]; Rothenberg PL, et al. EASD 2010:Abstract 876.[20]

Glucagon-Like Peptide

- **Tablet**: Rybelsus
- **Daily**: Victoza
- **Weekly**:
- Ozempic
- Trulicity
- Mounjaro (+ GIP)

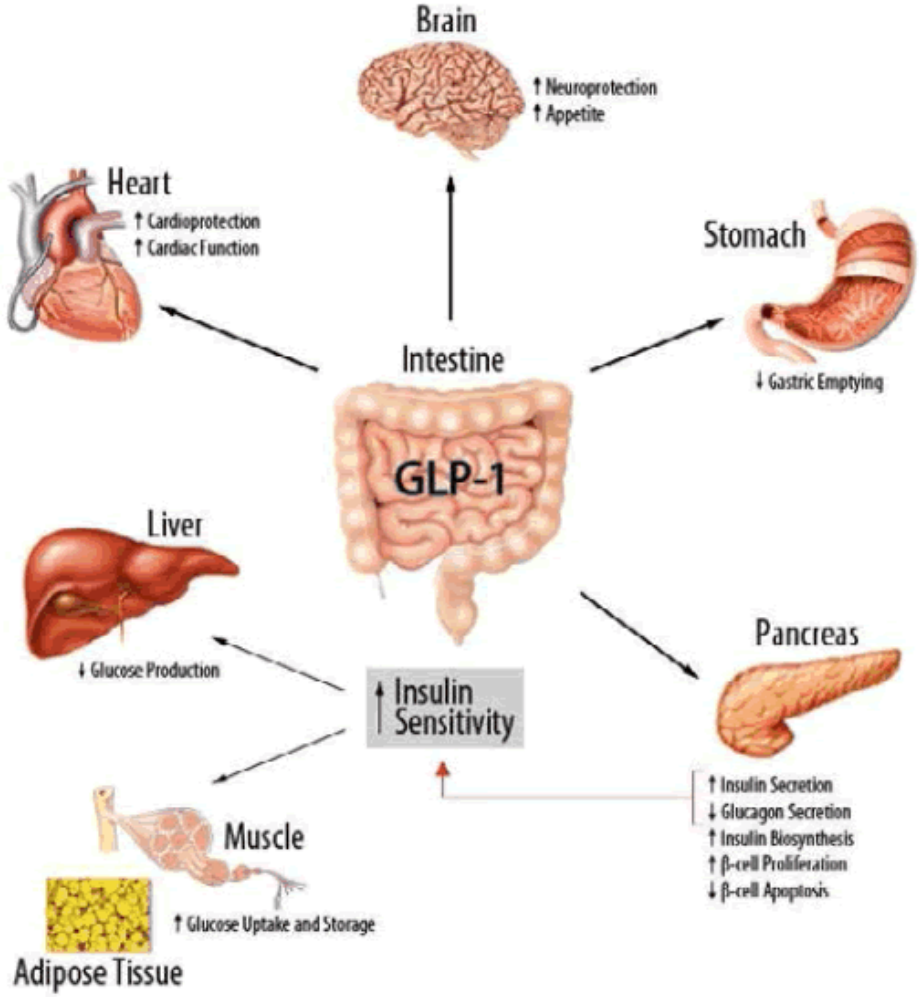


Figure 4: Biological Activities of GLP-1. The main action of GLP-1 occurs at the pancreas where GLP-1 stimulates insulin secretion and inhibits glucagon secretion in a glucose dependent manner. In addition, GLP-1 also decreases appetite, slows gastric emptying, increases β-cell proliferation, increases cardiac function as well as other physiological actions as indicated in the diagram. Redrawn from [91].

Smokey

- 8 months post-transplant
- Immunosuppression regimen stable
- Prednisone 5mg daily
- Weight up 28 pounds since transplant
- Hemoglobin A1C 7.2%
- Insulin regimen
  - Lantus 28 units qhs
  - Lispro 10 units with meals
  - Lispro correction scale 2 units:50mg/dl for BG >150mg/dl

Smokey

- Next steps
  - Continue Lantus 28 units qhs
  - Decrease Lispro to 8 units with meals
  - Decrease Lispro correction scale to 1 unit:50mg/dl for BG >150mg/dl
  - Start Mounjaro 2.5mg weekly
  - Nutrition counseling
  - Encourage physical activity/ exercise

Mr. Commodore

- 6 months post-transplant
- Immunosuppression regimen is stable
- Prednisone stopped
- Hemoglobin A1C 5.8%
- Fructosamine 250umol/L
- Insulin regimen:
  - Lantus 12 units qhs
  - Lispro correction scale: 1 unit:50mg/dl for BG >150mg/dl ACHS
- Adjustments?
  - Jardiance 25mg daily
  - Stop Lantus
  - Continue Lispro correction scale

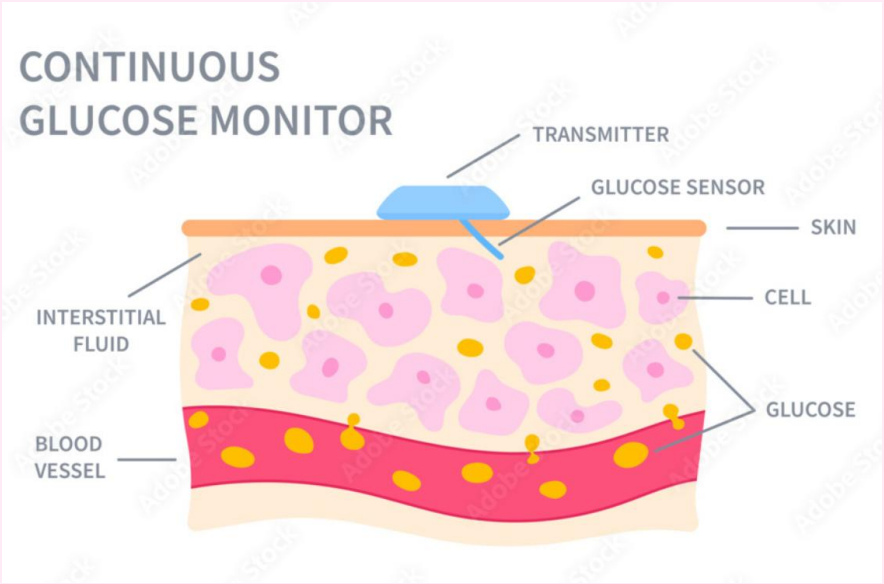
Hypoglycemia

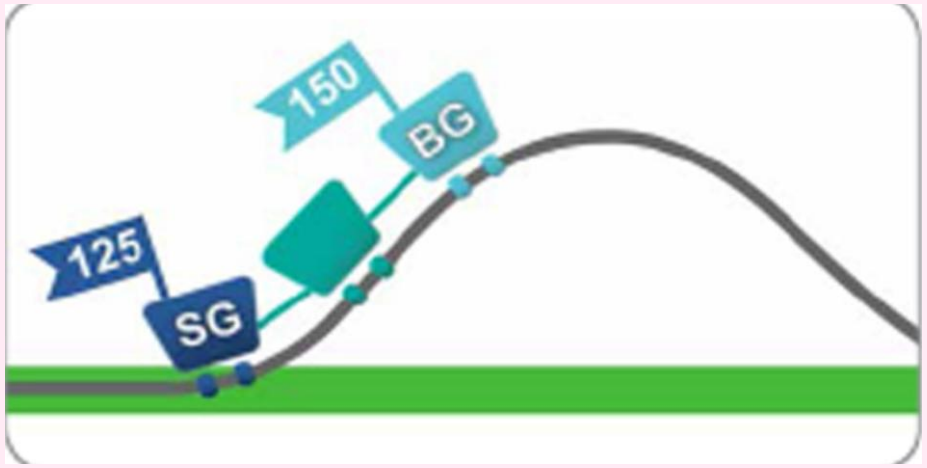
Glucose <70mg/dl

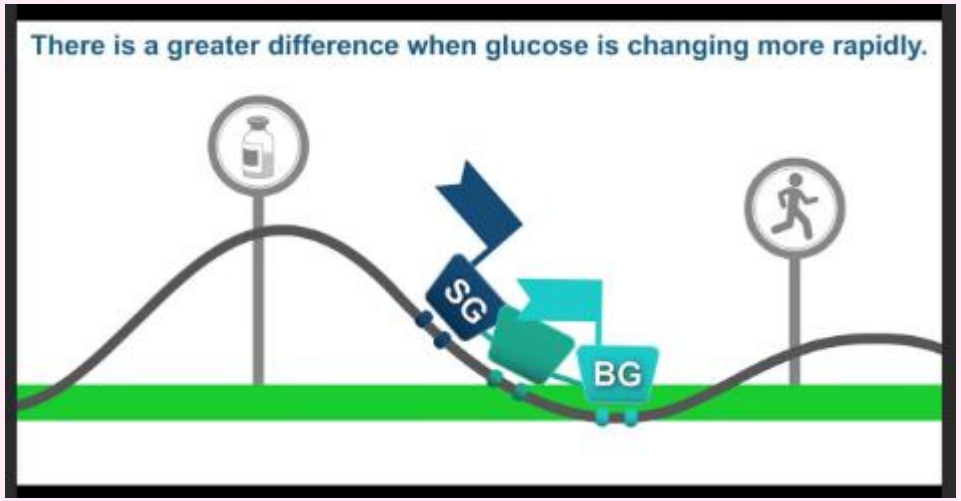
Treatment:

- **Alert**: 15 grams of fast-acting carbohydrates
  - 4 ounces of fruit juice or REGULAR soda (NOT diet or zero)
  - 4 glucose tablets
- **Altered/ Unable to take PO**
  - Glucagon: glucagon, Gvoke, Zegalogue, Baqsimi nasal spray

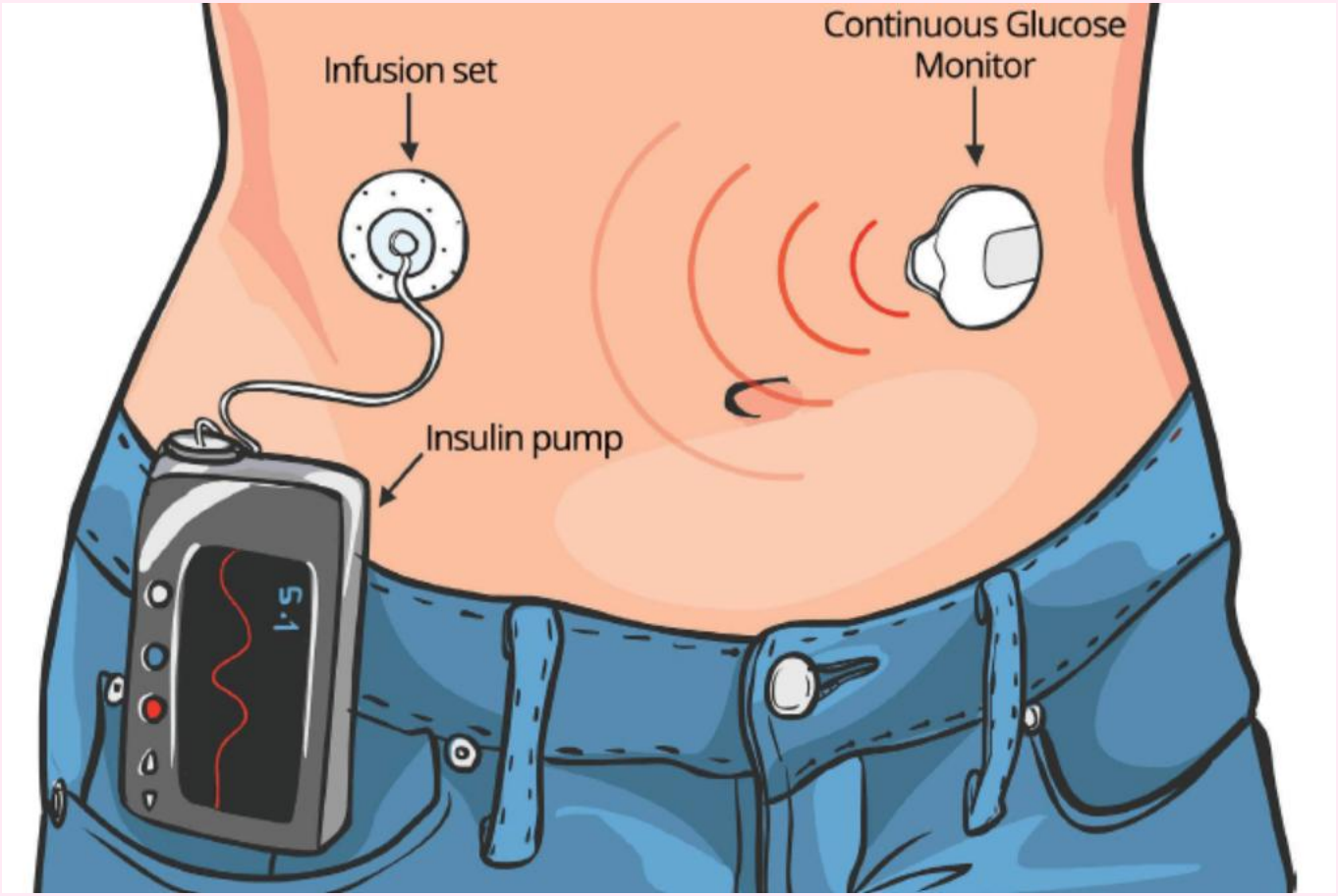
Continuous Glucose Monitors (CGMs)







Automated Insulin Pumps



Long Term Follow-Up

Monitor for microvascular complications

- Annual eye exam
- Annual foot exam
- Annual microalbumin/ BMP

Cardiovascular risk reduction

- Lipids
- Blood pressure

Summary

- Patients with diabetes > diabetes providers
- Treat hyperglycemia with insulin in the hospital
- When immunosuppression regimen is stable, can consider changing to oral meds/ GLP-1 to treat hyperglycemia
- Persistent PTDM should be treated as Type 2 DM, including monitoring for micro and macrovascular complications

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