# COMPLEXITIES AND NUTRITIONAL MANAGEMENT OF PANCREATIC CANCER RELATED DIABETES (TYPE 3C)

Anna Burton

Senior Specialist Pancreatic Dietitian

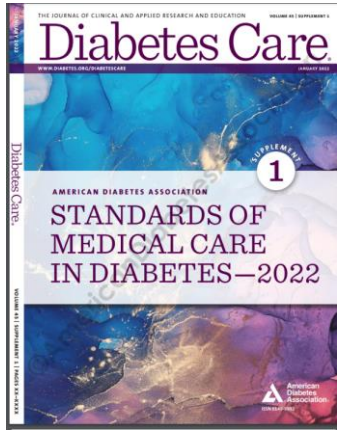
Leeds Teaching Hospital NHS Trust

# Poll – Terminology used within your service to describe diabetes of the exocrine pancreas?

- Type 3c diabetes
- Secondary diabetes
- Pancreatogenic diabetes
- Diabetes of the exocrine pancreas (DEP)
- Pancreatic diabetes
- Post pancreatitis diabetes mellitus (PPDM)
- Pancreatic cancer related diabetes (PCRD)
- Pancreatic cancer – Diabetes Mellitus (PC-DM)
- Other


Brittle diabetes
Pancreatogenic
Type 3c diabetes
PPRD
DEP
CFRD
PCRD
PDAC-DM
Pancreatic diabetes
Diabetes of the exocrine pancreas

# American Diabetes Association - 2022




- 'Pancreatic Diabetes is the preferred umbrella term'.
- Diverse set of aetiologies within the classification of diabetes in the context of the exocrine pancreas.
- Pancreatitis can lead to Post Pancreatitis Diabetes Mellites (PPDM).
- Distinguishing feature is concurrent pancreatic exocrine insufficiency.
- Diagnostic criteria:
  - Monoclonal faecal elastase test or direct exocrine function tests.
  - Pathological pancreatic imaging (endoscopic, US, MRI, CT).
  - Absence of type 1 associated autoimmunity.

*Diagnostic criteria by Ewald (2013)

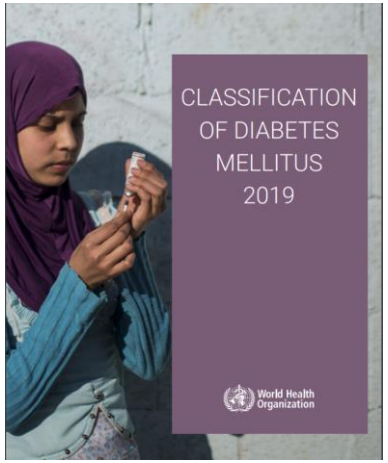
## Etiologic classification of diabetes mellitus.

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic
II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)
III. Other specific types
   A. Genetic defects of β-cell function
      1. MODY 3 (Chromosome 12, HNF-1α)
      2. MODY 1 (Chromosome 20, HNF-4α)
      3. MODY 2 (Chromosome 7, glucokinase)
      4. Other very rare forms of MODY (e.g., MODY 6: Chromosome 18, insulin promoter factor-1; MODY 6: Chromosome 2, NeuroD1; MODY 7: Chromosome 9, carboxyl ester lipase)
      5. Transient neonatal diabetes (most commonly 2AC/HYAMI imprinting defect on 6q24)
      6. Permanent neonatal diabetes (most commonly KCNJ11 gene encoding Kir6.2 subunit of β-cell $K_{ATP}$ channel)
      7. Mitochondrial DNA
      8. Others
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson-Mendenhall syndrome
      4. Lipoatrophic diabetes
      5. Others
   C. Diseases of the exocrine pancreas
      1. Pancreatitis
      2. Trauma/pancreatectomy
      3. Neoplasia
      4. Cystic fibrosis
      5. Hemochromatosis
      6. Fibrocalculous pancreatopathy
      7. Others
   D. Endocrinopathies
      1. Acromegaly
      2. Cushing's syndrome
      3. Glucagonoma
      4. Pheochromocytoma
      5. Hyperthyroidism
      6. Somatostatinoma
      7. Aldosteronoma
      8. Others
   E. Drug or chemical induced
      1. Vacor
      2. Pentamidine
      3. Nicotinic acid
      4. Glucocorticoids
      5. Thyroid hormone
      6. Diazoxide
      7. β-Adrenergic agonists
      8. Thiazides
      9. Dilantin
      10. γ-Interferon
      11. Others
   F. Infections
      1. Congenital rubella
      2. Cytomegalovirus
      3. Others
   G. Uncommon forms of immune-mediated diabetes
      1. Stiff-man syndrome
      2. Anti-insulin receptor antibodies
      3. Others
   H. Other genetic syndromes sometimes associated with diabetes
      1. Down syndrome
      2. Klinefelter syndrome
      3. Turner syndrome
      4. Wolfram syndrome
      5. Friedreich ataxia
      6. Huntington chorea
      7. Laurence-Moon-Biedl syndrome
      8. Myotonic dystrophy
      9. Porphyria
      10. Prader-Willi syndrome
      11. Others
IV. Gestational diabetes mellitus

Patients with any form of diabetes may require insulin treatment at some stage of their disease. Such use of insulin does not, of itself, classify the patient.

American Diabetes Association Dia Care 2014;37:S81-S90

# WHO classification of diabetes - 2019




**Diseases of the exocrine pancreas**

- Any process that diffusely damages the pancreas: pancreatitis, trauma, infection, pancreatectomy.
- Diabetes related to pancreatic adenocarcinoma is caused by other mechanisms then reduction of beta cell mass.

**Table 2: Types of diabetes**

| |
|---|
| **Type 1 diabetes** |
| **Type 2 diabetes** |
| **Hybrid forms of diabetes** |
| Slowly evolving immune-mediated diabetes of adults |
| Ketosis prone type 2 diabetes |
| **Other specific types** (see Tables) |
| Monogenic diabetes |
| - Monogenic defects of β-cell function |
| - Monogenic defects in insulin action |
| Diseases of the exocrine pancreas |
| Endocrine disorders |
| Drug- or chemical-induced |
| Infections |
| Uncommon specific forms of immune-mediated diabetes |
| Other genetic syndromes sometimes associated with diabetes |
| **Unclassified diabetes** |
| This category should be used temporarily when there is not a clear diagnostic category especially close to the time of diagnosis of diabetes |
| **Hyperglyacemia first detected during pregnancy** |
| Diabetes mellitus in pregnancy |
| Gestational diabetes mellitus |

**Diseases of the exocrine pancreas**

- Fibrocalculous pancreatopathy
- Pancreatitis
- Trauma/pancreatectomy
- Neoplasia
- Cystic fibrosis
- Haemochromatosis
- Others

DIABETES/METABOLISM RESEARCH AND REVIEWS
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RESEARCH ARTICLE

# Prevalence of diabetes mellitus secondary to pancreatic diseases (type 3c)

Ewald et al. (2012)

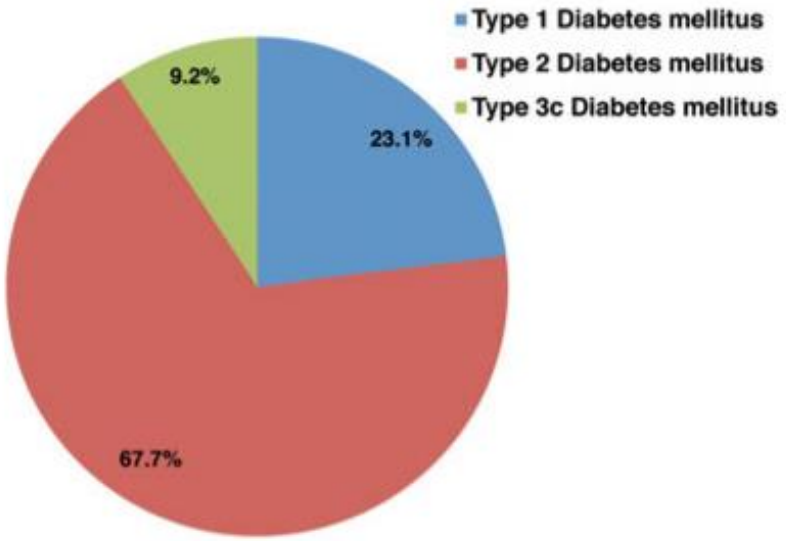



**Figure 1. Prevalence of diabetes type 1, type 2 and type 3c after reclassification**

Diabetes Metab Res Rev 2012; **28**: 338–342.
DOI: 10.1002/dmrr

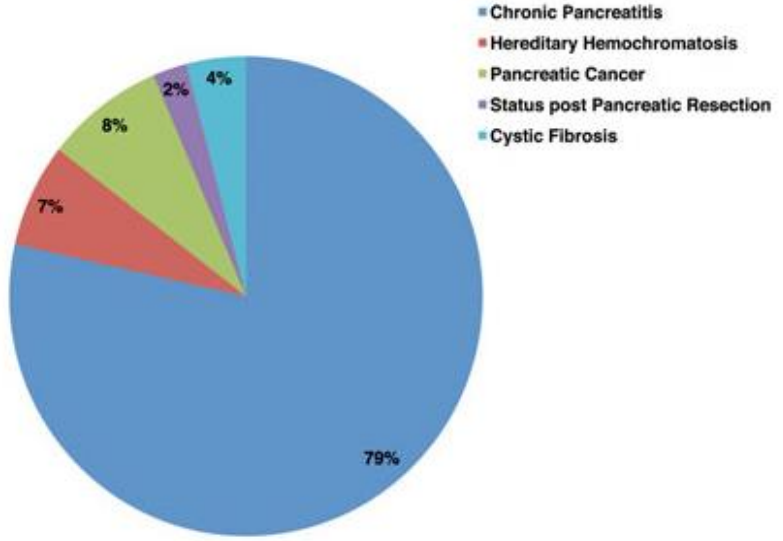
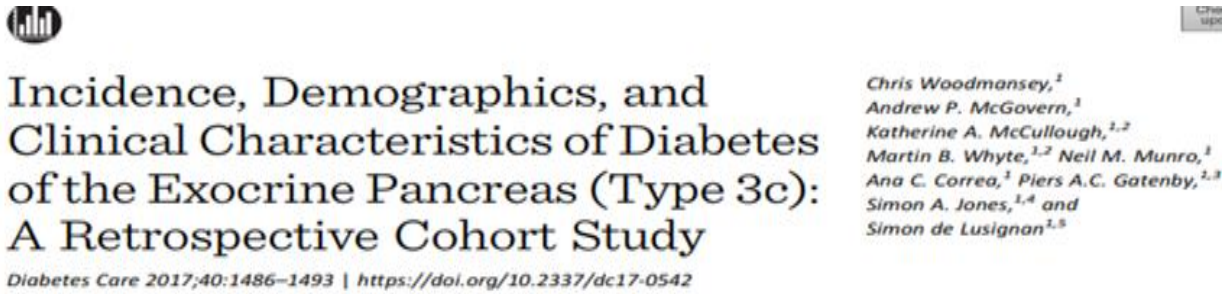



**Figure 3. Aetiology of diabetes mellitus type 3c**



Incidence, Demographics, and Clinical Characteristics of Diabetes of the Exocrine Pancreas (Type 3c): A Retrospective Cohort Study

Chris Woodmansey,[1] Andrew P. McGovern,[1] Katherine A. McCullough,[1,2] Martin B. Whyte,[1,2] Neil M. Munro,[1] Ana C. Correa,[1] Piers A.C. Gatenby,[1,3] Simon A. Jones,[1,4] and Simon de Lusignan[1,5]

Diabetes Care 2017;40:1486–1493 | https://doi.org/10.2337/dc17-0542

- Adult-onset DM – Primary care records in the UK (n31780 NOD in adults).
  - Diabetes in pancreatic disease - 2.59 per 100 000 person years.
  - Type 1 -1.64 per 100 000 person years.
  - Type 2 - 142.89 per 100 000 person years.

- Type 3c misdiagnosed in 87.5% (n559) as type 2 diabetes
- Poorer glycaemic control
- Progressed to insulin use in 5 years:
  - Type 2 - 4.1%
  - Acute pancreatitis - 20.9%
  - Chronic pancreatic disease - 45.8%

# Implication of methods used to diagnose hyperglycaemia in pancreatic cancer patients

| | Patient history or medical records | Fasting blood glucose | HbA1c | OGTT 75g |
|---|---|---|---|---|
| **% diagnosed as having diabetes** | 12 – 29% | 47% | 41.7% | 77% (diabetes and glucose intolerance) |

# Diabetes diagnostic criteria and considerations

## American Diabetes Association 2022

**Table 2.2—Criteria for the diagnosis of diabetes**

| |
|---|
| FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.* |
| OR |
| 2-h PG ≥200 mg/dL (11.1 mmol/L) during OGTT. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.* |
| OR |
| A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.* |
| OR |
| In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L). |

DCCT, Diabetes Control and Complications Trial; FPG, fasting plasma glucose; OGTT, oral glucose tolerance test; WHO, World Health Organization; 2-h PG, 2-h plasma glucose. *In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples.

- Fasting BG lower in pancreatic patients with low glucagon.
- Maldigestion – Untreated PEI.
- HbA1c contraindicated during acute illness, steroids, post pancreatic resection.
- HbA1c less then 48mmol/mol does not exclude diabetes.

# Pathophysiology

- Islets of Langerhans – endocrine hormones
  - Alpha cells – glucagon
  - Beta cells – insulin
  - Delta – somatostatin
  - Epsilon – Ghrelin
  - PP cells – F cells producing pancreatic polypeptide

- **Absence of islets** – total pancreatectomy (absolute deficiency of insulin, glucagon and pancreatic polypeptide).

- **Partial absence of functional islets** – chronic pancreatitis, partial pancreatectomy, severe acute pancreatitis.

- **Paraneoplastic** – pancreatic ductal adenocarcinoma

- Glucagon deficiency 'brittle diabetes'

- PP deficiency – hepatic insulin resistance.

- Pancreatic enzyme insufficiency and maldigestion
  - Relationship between endocrine and exocrine function –incretin secretion.
  - PERT management (improving and unmasking DM)

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REVIEW ARTICLE

# Diabetes of the exocrine pancreas

Katie Wynne,*,† Benedict Devereaux‡,§ and Anne Dornhorst¶,**

*Department of Diabetes and Endocrinology, John Hunter Hospital, †University of Newcastle, Newcastle, New South Wales, ‡Royal Brisbane and Women's Hospital, and §University of Queensland, Brisbane, Queensland, Australia; and ¶Imperial College Healthcare NHS Trust, Hammersmith Hospital, and **Imperial College London, London, UK

**Table 4** Secretory products of the cells in the islets of Langerhans and their endocrine effects

| Cell type | Frequency of cell type by pancreatic region (% islet volume) | | Hormone released | Action |
|---|---|---|---|---|
| | Posterior head | Anterior head, body, and tail | | |
| α cell | Very low (< 1%) | Moderate (15%) | Glucagon | Stimulates the breakdown of stored hepatic glycogen during fasting |
| β cell | Moderate (20 %) | High (80%) | Insulin | Promotes storage of nutrients in liver, muscle, and adipose tissue<br>Paracrine inhibition of α cells |
| PP cell | High (80 %) | Very low (< 1%) | PP | Potentiates the effect of insulin on liver |
| δ cell | Very low (< 1%) | Low (5%) | Somatostatin | Slowing of nutrient absorption from intestinal tract<br>Paracrine inhibition of glucagon and insulin |

PP, pancreatic polypeptide.

# Pathophysiology of diabetes specific to pancreatic cancer

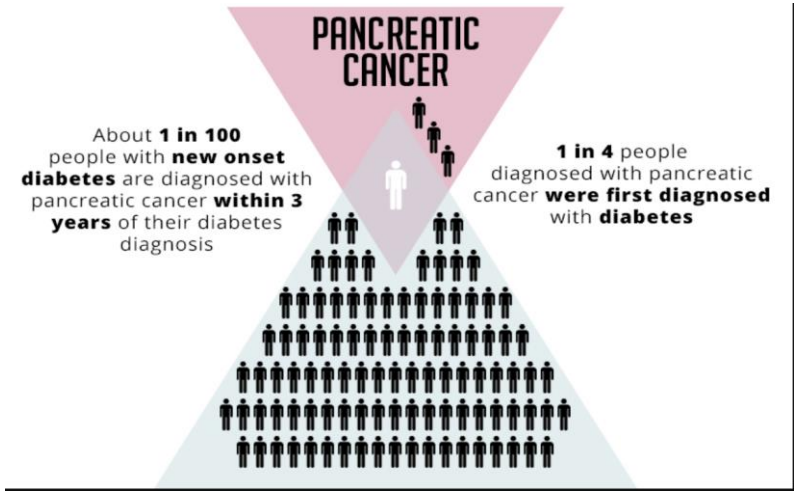
- Proposed mechanisms of hyperglycaemia

- Insulin deficiency due to beta cell loss/dysfunction
- Inflammatory mediators
- Paraneoplastic mechanism causing IR and beta cell dysfunction
- Immunopathogenesis
- Hepatic insulin resistance caused by reduction in pancreatic polypeptide
- Peripheral insulin resistance
- Reduced incretin effect
- Genetic
- Adrenomedullin and Vanin 1 as mediators of inflammation causing beta cell toxicity
- Gut microbiome

# Pancreatic Cancer Related Diabetes

- Risk factors:
  - long standing diabetes and obesity
  - Chronic pancreatitis

- New onset diabetes 2 – 3 years prior to diagnosis of pancreatic cancer
  - DETECT study
  - ENDPAC study
  - Biomarkers
  - Clinical prediction models




National Cancer Institute

## NOD type 2 vs. PCRD

- Sudden weight loss
- Lower BMI
- Deteriorating BG control
- Rapid on set of hyperglycaemia
- Lack of response to escalating diabetes medication
- Malnutrition
- GI symptoms

# Diabetes and cancer treatment pathway

- 2 – 3 years prior to diagnosis
- Post pancreatic surgery (metabolic response and loss of pancreatic tissue)
  - New onset
  - Exacerbation or resolution of PCRD
  - Coexisting type 2 and related to pancreatic surgery
- During chemo/radiotherapy
- Steroids
- Pancreatitis (acute and chronic)

Frequent monitoring and long term follow up

Research and national guidelines

# Clinical example 1 – undiagnosed DM
# Setting – Pancreatic surgical outpatients.
# Pancreatic head adenocarcinoma

- Struggling with:
  - Poor appetite, lethargy, weight loss (8kg over previous 8 weeks), steatorrhoea, taste changes, polyuria and thirst.
- Random blood glucose - 20mmol/l
- BMI 23kg/m2
- Commenced pancreatic enzyme replacement
  - 75 000 with meals and 50 000 with snacks
- First line dietary advice – nutritional support avoiding high GI/sugary drinks and snacks
- Referred to oncology for assessment for chemotherapy
- Diabetic medication started by oncologist Friday pm – gliclazide 40mg bd

# Clinical example 2 – misdiagnosed DM
# Setting – Pancreatic surgical outpatients.
# Pancreatic head adenocarcinoma

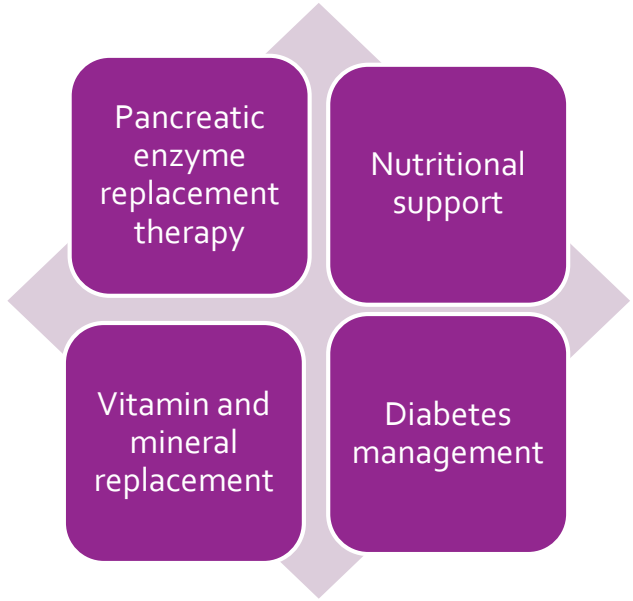
- Diagnosed Type 2 diabetes 8 months ago.
- Following healthy eating advice for weight loss
- Weight loss – 10kg, BMI 22kg/m2
- Appetite reasonable but reduced portion sizes and avoiding "sugary foods, desserts and choosing low fat options.
- Prescribed metformin by GP
- Struggling with abdominal pain, bloating, wind, urgency with slightly paler than usual stools.
- Not monitoring BG
- Offered group type 2 diabetes support session by GP practice.
- MDT – work up for possible Pylorus Preserving Pancreaticduodenectomy (PPPD)

- Unpick and explain type 2 diabetes and diabetes related to pancreatic cancer.
- Reframe nutritional goals.
- Start on PERT – review and assess considering GI side effects of Metformin and optimise PERT.
- Repeat BG and HbA1c (implication of starting PERT)
- Tertiary DM centre review depending outcome of BG results.
- Nutritional support – high protein/high kcal/low simple sugars and low GI.
- Exercise – Prehab service
- Goal – optimise nutrition, glycaemic control and physical function in preparation for pancreatic surgery.

# Nutritional aims

## In-line with cancer pathway and diabetes treatment

- Prevent hypoglycaemic events (<4mmol/L)
- Minimise hyperglycaemia
- Optimise nutritional status
- PEI management
- Monitor and correct any micronutrient deficiency
- Aim HbA1c <53mmol/mol
  - Minimise risks of longer-term complications
- Consider lifestyle factors
- Education and understanding
  - Challenging type of DM to manage - reassure and support.



# Care planning

- Hypoglycaemic awareness and treatment plan.
- Regular eating pattern, including starchy carbohydrates.
- Avoid missing meals.
- Small frequent meal pattern.
- Limit simple sugars and refined carbohydrates – especially sugary drinks and sweets.
- Nutritional drinks
  - Slow, avoid juice style
  - insulin
  - CHO 25 – 67g per bottle
- Lower glycaemic index foods and meal composition.
- Avoid 'diabetic foods'
- Monitor BG regularly
  - Intensive insulin regimen: 6 – 10 x per day or CGM
  - Monitor BG, diet, exercise and PERT 'brittle diabetes'
- Ensure adequate PERT and monitor for glycaemic consequences.
- Lifestyle changes – alcohol, smoking and exercise (prehab and beyond).

Adapted for pancreatic cancer patients from: Duggan et al (2017)

# Enteral tube feeding and insulin management

- Joint consultations
- Insulin profile and enteral feeding plan
- MDT communication
  - Alert – any interruption to feeding after insulin administered – hypo risk.
- Carbohydrate content of feeds
  - 1000kcal MCT/peptide feed contains 113 – 142g CHO
- Consider oral intake – hypo/hyper risk
- PERT
- Clinical condition –
  - stressed catabolic state vs. recovery
  - Steroids/chemotherapy
- Monitor, review and adjustment
- Risk assess HETF and insulin management

| BRAND NAME | GENERIC | DEVICE | | | TIME PROFILE | DOSING SCHEDULE |
|---|---|---|---|---|---|---|
| | | Vial | Disposable pen | Cartridge | | |
| **RAPID ACTING INSULIN ANALOGUES/SHORT ACTING SOLUBLE INSULINS** | | | | | | |
| Novorapid | Insulin Aspart | ✔ | FLEXPEN & FLEXTOUCH | ✔ |   | Usually THREE times a day<br>IMMEDIATELY before, or just after food<br>**Or**<br>When required for Hyperglycaemia |
| Humalog | Insulin Lispro 100 units/ml | ✔ | KWIKPEN | ✔ | | |
| | 200 units/ml | ✖ | KWIKPEN | ✖ | | |
| Apidra | Insulin Glulisine | ✔ | SOLOSTAR | ✔ | | |
| Fiasp | Insulin Aspart | ✔ | FLEXTOUCH | ✔ | | |
| Actrapid | Human soluble insulin | ✔ | ✖ | ✖ |   | Usually THREE times a day<br>**30 minutes** before, or just after food |
| Humulin S | | ✔ | ✖ | ✔ | | |
| Insuman Rapid | | ✖ | ✖ | ✔ | | |
| **LONG ACTING INSULIN ANALOGUES/INTERMEDIATE ACTING INSULINS** | | | | | | |
| Levemir | Insulin Detemir | ✖ | FLEXPEN & INNOLET | ✔ |   | ONCE or<br>TWICE a day |
| Abasaglar | Insulin Glargine | ✖ | KWIKPEN | ✔ | | |
| Lantus | | ✔ | SOLOSTAR | ✔ | | |
| Toujeo | Insulin Glargine 300 units/ml | ✖ | SOLOSTAR | ✖ | | |
| Tresiba | Insulin Degludec 100 units/ml | ✖ | FLEXTOUCH | ✔ | | |
| | 200 units/ml | ✖ | FLEXTOUCH | ✖ | | |
| Insuman Basal | Isophane insulin | ✔ | SOLOSTAR | ✔ |   | |
| Insulatard | | ✔ | INNOLET | ✔ | | |
| Humulin I | | ✔ | KWIKPEN | ✔ | | |
| **PRE-MIXED BIPHASIC INSULIN ANALOGUES/ PRE-MIXED BIPHASIC INSULIN** | | | | | | |
| Novomix 30 | Biphasic insulin Aspart | ✖ | FLEXPEN | ✔ |   | TWICE or THREE times a day<br>**15 minutes**<br>before, or just after food |
| Humalog Mix 25 | Biphasic insulin Lispro | ✔ | KWIKPEN | ✔ | | |
| Humalog Mix 50 | | ✖ | KWIKPEN | ✔ | | |
| Humulin M3 | Soluble and Isophane insulin | ✖ | KWIKPEN | ✔ | Time profile varies on the proportion of short acting insulin | TWICE daily<br>**30 minutes,** before food |
| Insuman Comb 15 | | ✖ | ✖ | ✔ | | |
| Insuman Comb 25 | | ✔ | SOLOSTAR | ✔ | | |
| Insuman Comb 50 | | ✖ | ✖ | ✔ | | |

V1.0 22.02.18

| Medication class | Name | Mode of action | Pros/Cons and considerations |
|---|---|---|---|
| Biguanides | Metformin<br>*First line | Decreasing gluconeogenesis by opposing the action of glucagon.<br>Increasing peripheral use of glucose.<br>Requires some residual functioning pancreas.<br>Insulin resistance | Reduces risk of pancreatic cancer<br>First line if hyperglycaemia mild<br>Sensitisation to chemo - gemcitabine<br>GI side effects and weight loss<br>Avoid with ongoing alcohol excess – lactic acidosis risk<br>B12 malabsorption |
| Sulfonylureas<br>(SUs) | Gliclazide,<br>Glibenclamide,<br>Glimepiride<br>Glipizide<br>Gilclazide | Stimulate beta cells in the pancreas to produce more insulin. | Hypoglycaemia risk<br>Hyponatraemia<br>Avoid in severe renal and hepatic impairment<br>Absorption reduced by colesevelam |
| Glinides | Nateglinide,<br>Repaglinide | Stimulate beta cells in pancreas to produce more insulin | Useful in milder hyperglycaemia prior to starting insulin<br>Hypoglycaemia risk<br>Shorter half life then sulfonylureas |
| Thiazolidinediones<br>(Glitazones)<br>(TZDs) | Rosiglitazone,<br>Pioglitazone | Binds to a receptor which promotes deposition of fat cells into peripheral tissue which improves a person's sensitivity to insulin. | Should be avoided due to osteoporosis, fluid retention, congestive heart disease.<br>Weight gain due to increase in peripheral fat mass.<br>Avoid in patients with chronic pancreatitis |
| Alpha-glycosidase inhibitors<br>(AGIs) | Acarbose<br>Miglitol | Block and slow down the absorption of carbohydrates from the GIT. | Increases PEI and weight loss<br>DKA and acute pancreatitis risk<br>Little or no evidence - should be avoided |
| Incretin based therapies<br>GLP-1 analogues - injection<br>DPP-4 inhibitors - tablet | Exenatide<br>Liraglutide<br>Sitagliptin<br>Saxagliptin<br>Linagliptin | GLP-1 incretin mimics – increases levels of incretins.<br>DPP-4 works by blocking the action of the enzyme which destroys the hormone incretin. Incretin signals the pancreas to produce more insulin and reduce hepatic glucose production. | GI side effects<br>Increased risk of acute pancreatitis<br>Pancreatic cancer risk<br>Current recommendation is to avoid in type 3cDM |
| Sodium glucose co-transporter-2 (SGLT-2) | Dapagliflozin<br>Canagliflozin<br>Empagliflozin | Reduce renal reabsorption of glucose without stimulating insulin release | DKA in insulin deficient patients and should not be prescribed |
| Insulin | | Insulin replacement therapy | Anabolic effect for treating malnutrition<br>Hypoglycaemic risk<br>Carcinogenic |

# LTHT Algorithm for the Treatment of Hypoglycaemia in Adults with Diabetes

## Hypoglycaemia is defined as capillary blood glucose (CBG) less than 4mmol/L

(if not less than 4mmol/L but symptomatic give a small carbohydrate snack)

| Mild | Moderate | Severe |
|---|---|---|
| **Patient conscious, orientated, able to swallow and able to self-help** | **Patient conscious, able to swallow but disorientated or unable to self-help** | **Patient unconscious/ fitting/ aggressive or nil by mouth (NBM)** |

**For patients on IV insulin: In the event of hypoglycaemia (CBG less than 4mmol/L), reduce IV insulin infusion to 0.1 unit/hour. Correct hypoglycaemia as below. IV insulin MUST be restored to an appropriate rate once patient's CBG greater than 4mmol/L.**

**For enterally fed patients with mild or moderate hypoglycaemia: Please refer to Leeds Health Pathways Hypoglycaemia guidelines for details.**

**Give 15-20g of quick acting carbohydrate e.g. 1 bottle of glucose liquid** (e.g. GlucoJuice), or 4-5 glucose tablets (e.g.Gluco-Tabs)

**Test CBG after 15 minutes**. If still less than 4mmol/L, can repeat above up to 3 times.

If this has been repeated 3 times and patient not responding to treatment or clinically deteriorating, consider IV 10% glucose at 100ml/hr (volume according to clinical circumstance) or 1mg IM glucagon*

**Give 15-20g of quick acting carbohydrate** e.g. **40% Glucose oral gel** (1.5-2 tubes) or consider 1mg IM glucagon*

**Test CBG after 15 minutes**. If still less than 4mmol/L, can repeat 15-20g of quick acting carbohydrate up to 3 times. Continue to test every 15 minutes

If this has been repeated 3 times and patient not responding to treatment or clinically deteriorating consider IV 10% glucose at 100ml/hr (volume according to clinical circumstance)

**Check airway, breathing and circulation and fast bleep a doctor for urgent review**

**If IV access secured** give 150ml of 10% glucose over 15mins (rate 600ml/hr)

**If unable to quickly secure IV access** give 1mg IM glucagon*.

**Test CBG every 15 minutes** till CBG greater than 4mmol/L. If CBG less than 4mmol/L and patient still unwell, 150ml of IV 10% glucose over 15 mins can be repeated up to 3 times, dependent on clinical response.

**Blood glucose level should now be above 4mmol/L.** If able to swallow, give 20g of long-acting carbohydrate (e.g. 2 biscuits/ 1 piece of toast/ or next meal if due)

If IM glucagon has been used give 40g carbohydrate to replenish glycogen stores.

**For patients who are NBM and on IV insulin :** Review and adjust IV insulin infusion. When CBG greater than 4mmol/L commence 10% glucose at 100ml/hr (volume according to clinical circumstance)

**For enterally fed patients:** Restart feed, or give bolus feed as per guideline or IV 10% glucose at 100ml/hr.

**DO NOT** OMIT SUBSEQUENT DOSES OF INSULIN, CONTINUE REGULAR CBG MONITORING FOR 24-48 HOURS AND GIVE HYPO EDUCATION. DOSE ADJUSTMENT OF INSULIN MAY BE REQUIRED. REFER TO DIABETES TEAM IF HYPO UNEXPLAINED, SEVERE OR RECURRENT.

In all cases: IV Glucose and IM Glucagon must be prescribed on charts, and hypoglycaemia event documented in patients notes, with completion of audit form

(*NB Give once only. Certain patients may respond poorly to glucagon i.e. those with malnourished, severe liver disease, glucocorticoid deficiency)

# NICE guidelines NG28 Type 2 diabetes in adults management (updated 31 March 2022)

Real time Continuous Glucose Monitor (rtCGM)
Intermittently scanned CGM 'Flash' monitor (isCGM)

## Type 1 adults:

- Everyone is offered a rtCGM or isCGM.

## Type 2 adults:

- Everyone who has multiple daily injections (two or more daily insulin injections) and at least one of the following:
    1. They have recurrent hypoglycaemia or severe hypoglycaemia
    2. They have impaired hypoglycaemia awareness
    3. They have a condition or disability which means they are unable to self-monitor their blood glucose.
    4. They would otherwise be advised to self monitor 8 times per day.


FreeStyle Libre
Dexcom
Glucomenday
Guardiam connect
GlucoRx Aidex

FreeStyle Libre

# Supportive information

Pancreatic Cancer UK

## Diabetes if you have pancreatic cancer

### Information about type 3c diabetes

If you have pancreatic cancer or have had surgery to remove the cancer, you may have a type of diabetes called type 3c diabetes. This information is for people with type 3c diabetes. It explains what type 3c diabetes is, and how to manage it.

Managing diabetes if you have pancreatic cancer can be complicated. Speak to your medical team for help with managing diabetes, and ask them any questions you have.

Nutrition Interest Group of the Pancreatic Society (NIGPS)

Supported by Pancreatic Cancer UK

## Type 3c diabetes and reduced appetite

This booklet has been produced for people who have a particular type of diabetes that is caused by having all or part of the pancreas removed (surgically) or the pancreas being damaged, (for example by pancreatitis or pancreatic cancer). **This is called Type 3c Diabetes.**

This booklet is for people with a reduced appetite or who have lost weight, who are aiming to put weight back on, and/or recover from surgery.

Our other publication 'Type 3c diabetes and healthy living' provides advice for people with type 3c diabetes who are aiming to maintain or reduce their weight and are not recovering from surgery.

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