# Managing diabetes as an acute and long term condition

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#### Aims of the session

- 1. Describe the different metabolic disorders of glucose metabolism, the pathogenesis of these & their clinical characteristics
- 2. Discuss the differences between type 1 diabetes and type 2 diabetes in terms of clinical presentation, patient characteristics and diagnostic criteria.
- 3. Discuss treatment pathways for type 1 and type 2 diabetes.
- 4. Discuss acute complications and emergencies.
- 5. Discuss chronic complications of diabetes.
- 6. Explore Nursing interventions.
- 7. Explore principles of patient centred care and selfmanagement.

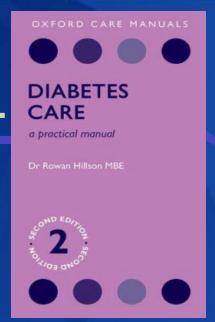
## Key Knowledge Sources used in this session

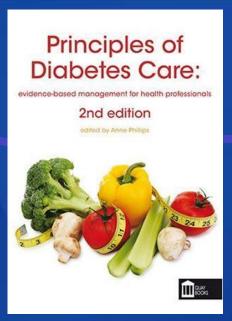
- NICE:
- Type 1 diabetes in adults: diagnosis and management (NG17) (Last update 2016)
   https://www.nice.org.uk/guidance/ng17

- Type 2 diabetes in adults: management (NG28) (Last Update May 2017)
  - https://www.nice.org.uk/guidance/ng28

### Knowledge Sources continued...

- National Diabetes Audit
  - http://content.digital.nhs.uk/nda
- Research @Hudd





Kitchanapaibul, S., Gillibrand, W. and Burton, R. (2017) 'Self-Management among the Ethnic Groups with Type 2 Diabetes Mellitus in Thailand' World Academy of Science, Engineering and Technology: International Journal of Social, Management, Economics and Business Engineering . ISSN 2010-376X

Rahman, R., Marler, J., Stephenson, J. and Gillibrand, W. (2017) 'Risk Factors for Elevated Intraocular Pressure on First Day Postoperative Review following Pars Plana Vitrectomy' Journal of Vitreoretinal Diseases . ISSN 2474-1264

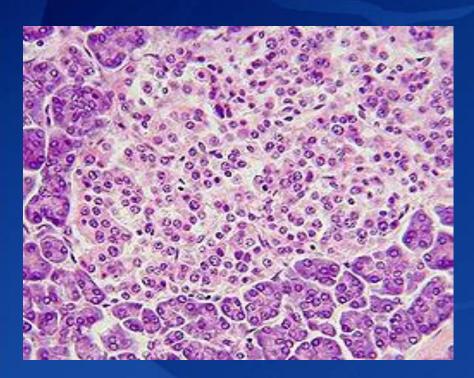
Gillibrand, W. and Holdich, P. (2017) 'Assessment of retinopathy'. In: Principles of Diabetes Care: evidence-based management for health professionals. Bournemouth, UK: Quay Books. pp. 217-226. ISBN 9781856425100

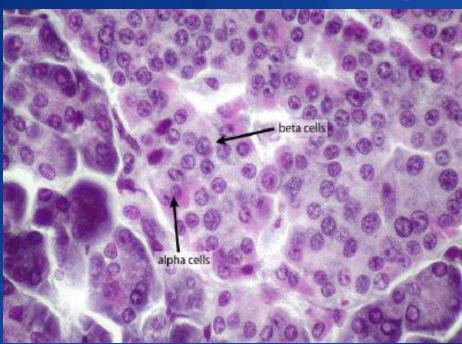
Levy, N., Gillibrand, W. and Kola-Palmer, S. (2017) 'Minor Amputation and Quality of Life: Is it Time to Give the Patient a Voice?' The Diabetic Foot Journal, 20 (4), pp. 228-234. ISSN 1462-2041

Youngs, W., Gillibrand, W. and Phillips, S. (2016) '<u>The impact of pre-diabetes diagnosis on behaviour change: an integrative literature review</u>' *Practical Diabetes International*, 33 (5), pp. 171-175. ISSN 1357-8170

Brooks, J., Kime, N., King, N., Wearden, A., Gillibrand, W. and Campbell, F. (2015) 'Exploring how young people think about and respond to diabetes in their peers' Diabetes Care for Children & Young People, 4 (1), pp. 14-18. ISSN 2050-1528

## What is this?

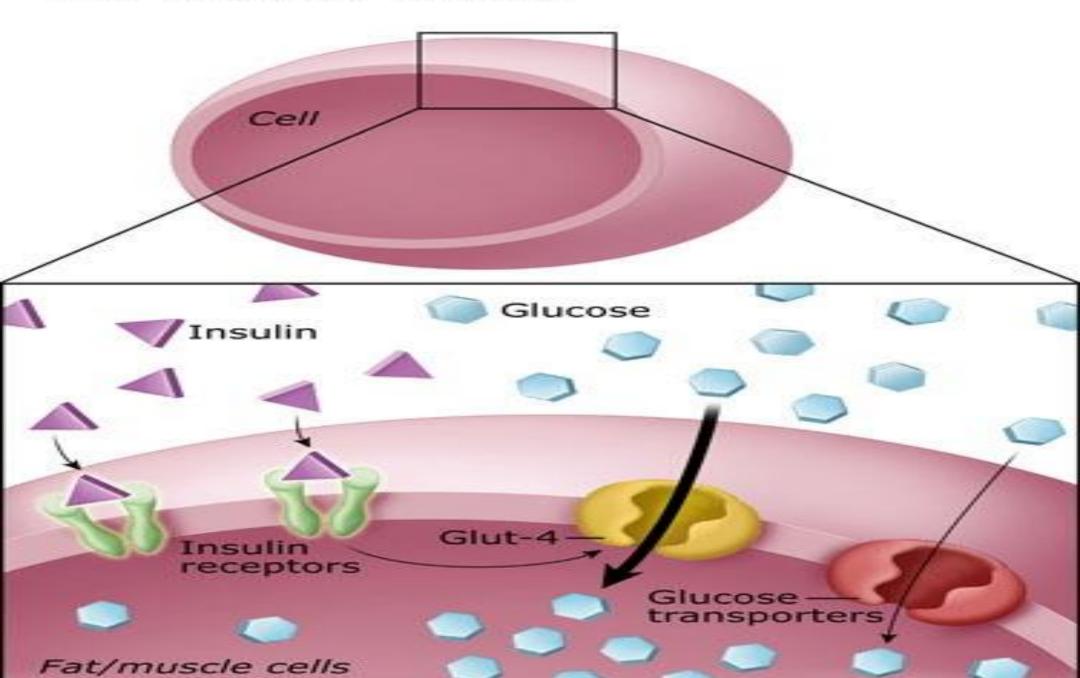


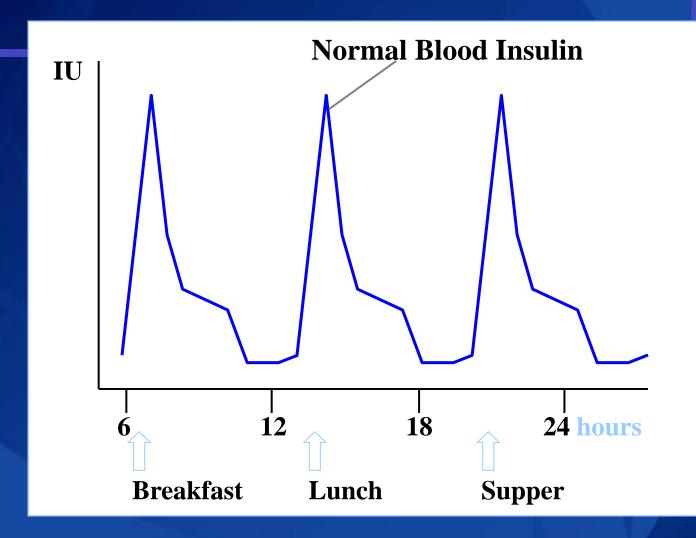


## Control of blood glucose

High blood glucose Raises blood sugar Glucagon stimulates the conversion of stored glycogen in the liver into glucose. Glucagon released by alpha cells of pancreas Glycogen Glucose Insulin Insulin released stimulates the by beta cells liver to remove of pancreas glucose from the blood and stores it as Tissue cells glycogen. take up glucose from blood. Lowers blood sugar Low blood glucose

#### The Effect of Insulin





#### What is diabetes?

## What is diabetes?

Diabetes is a chronic disease that occurs when the pancreas does not produce enough insulin, or when the body cannot effectively use the insulin it produces. Hyperglycaemia, or raised blood sugar, is a common effect of uncontrolled diabetes and over time leads to serious damage to many of the body's systems, especially the nerves and blood vessels.

WHO (2018) Diabetes Fact Sheet.

http://www.who.int/mediacentre/factsheets/fs312/en/index.html Accessed 16th January 2018

#### **Incidence and Prevalence**

Globally: The estimated diabetes prevalence worldwide for 2011 was 366 million and it is expected to affect 552 million people by 2030

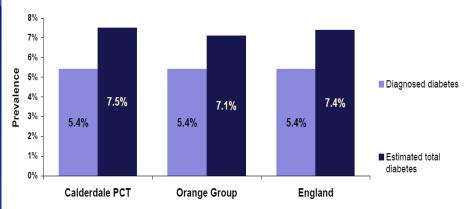
#### **United Kingdom**

- Prevalence % = England 5.5%; NI 3.8%; Scotland 4.3%; Wales 5.0%
- It is estimated that there are around 850,000 people in the UK who have diabetes but have not been diagnosed.
- 10% Type 1 adults. (15% if include children)
- 90% Type 2 adults. (85% including children)

Source - Diabetes in the UK (2018)

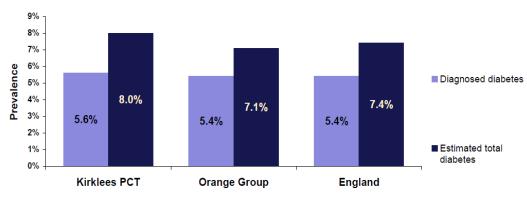
### Local prevalence

In 2009/10 there were 8866 people aged 17 years and older diagnosed with diabetes in Calderdale PCT. There is also an estimated 3379 adults with undiagnosed diabetes. The chart below compares the prevalence of diabetes in Calderdale PCT with the cluster group and England as a whole.



Source: Quality and Outcomes Framework, 2009/10 and APHO Diabetes Prevalence Mode

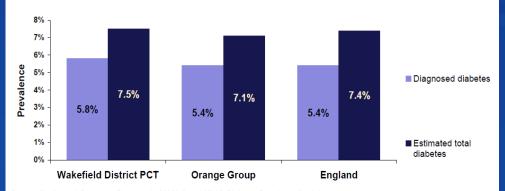
In 2009/10 there were 18059 people aged 17 years and older diagnosed with diabetes in Kirklees PCT. There is also an estimated 7292 adults with undiagnosed diabetes. The chart below compares the prevalence of diabetes in Kirklees PCT with the cluster group and England as a whole.



Source: Quality and Outcomes Framework, 2009/10 and APHO Diabetes Prevalence Model

#### Calderdale

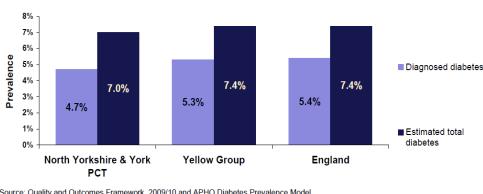
In 2009/10 there were 16148 people aged 17 years and older diagnosed with diabetes in Wakefield District PCT. There is also an estimated 3646 adults with undiagnosed diabetes. The chart below compares the prevalence of diabetes in Wakefield District PCT with the cluster group and England as a whole.



Source: Quality and Outcomes Framework, 2009/10 and APHO Diabetes Prevalence Model

#### Kirklees

In 2009/10 there were 30350 people aged 17 years and older diagnosed with diabetes in North Yorkshire & York PCT. There is also an estimated 16744 adults with undiagnosed diabetes. The chart below compares the prevalence of diabetes in North Yorkshire & York PCT with the cluster group and England as a whole.



Source: Quality and Outcomes Framework, 2009/10 and APHO Diabetes Prevalence Model

**Pathology & Classification of Diabetes** 

## **Case Study One**



Ben is 15 years old, complaining of increased urination, weakness and weight loss over 3 months.

Looks dehydrated and his random plasma glucose is 13.8mmol/L.

What is his diagnosis?

What leads you to suspect this?

Are there any tests or investigations you might carry out?

## **Case Study Two**



Mrs Sarah Palin is a 56 year old lady with a BMI of 31.

She presents with a UTI.

Her fasting glucose is 6.9mmol/L.

She has a sister with diabetes.

What is her likely diagnosis?

What would you do with her?

Why?

## Type 1 diabetes

- Auto-immune elimination of beta-cells (beta-cell antibodies)
- Genetic
- Clinical picture:
  - Absolute requirement for insulin
  - (without it: hyperglycaemia wasting, ketoacidosis)
  - Otherwise healthy
- Incidence
  - Two peaks:
    - a) infancy (1-4y)
    - b) early adolescence (8-12y)
  - May present at any time in life (if ~ type 2: LADA)



## Development of type 1 diabetes

#### **Causes - multifactorial**

Genetic predisposition

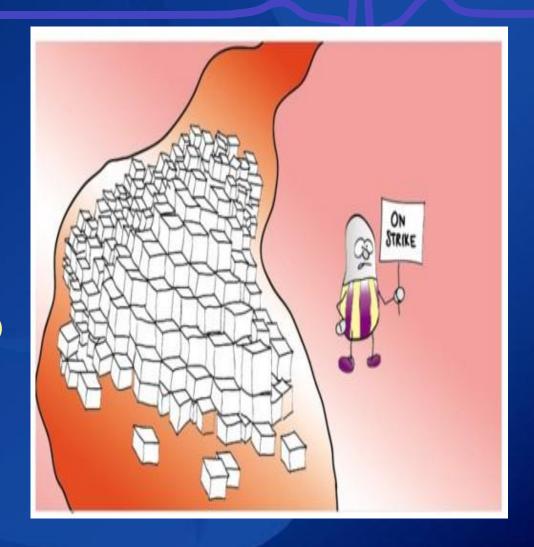


Diabetogenic trigger (environment)



Immune response (islet cell antibodies)





Type 2 Diabetes

- Decline in insulin production
- Insulin resistance

**Environment/Lifestyle factors:** 

- •Weight central (50-70%)
- Calorie dense diet
- Inactivity
- Thrifty gene
- Drug induced

Key study <u>UKPDS</u>



Age: peak 60 yrs

Prevalence: 85% PWD have

type 2

**Genetic susceptibility:** 

Family history = 40% patients

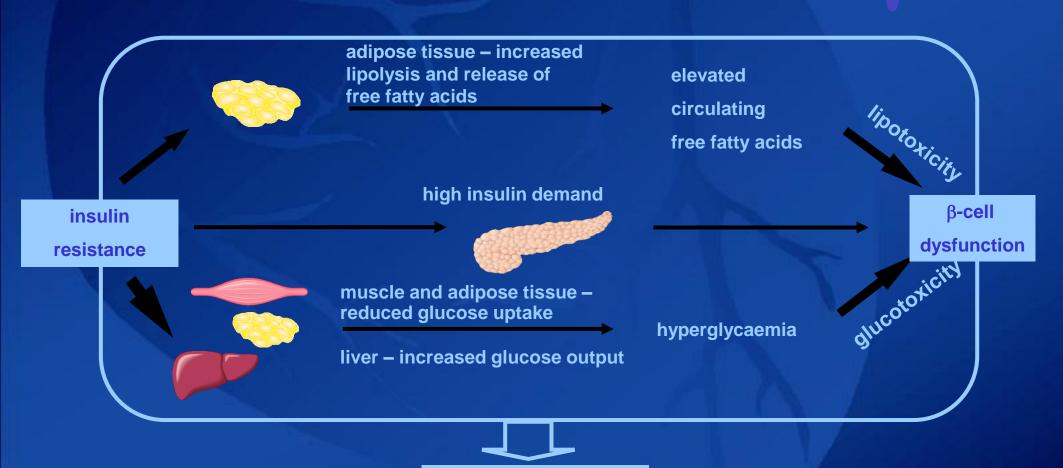
Twin = 60-90% concordance

Lifetime risk first degree relative with diabetes = five-fold

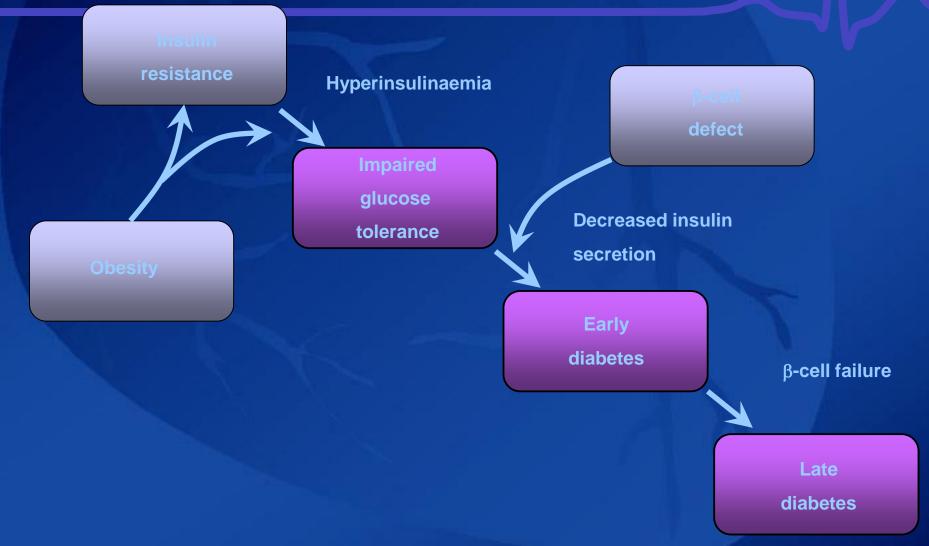
In-utero/infant malnutrition

Black (9%) & Asian (11%) compared to 1-3% UK white population

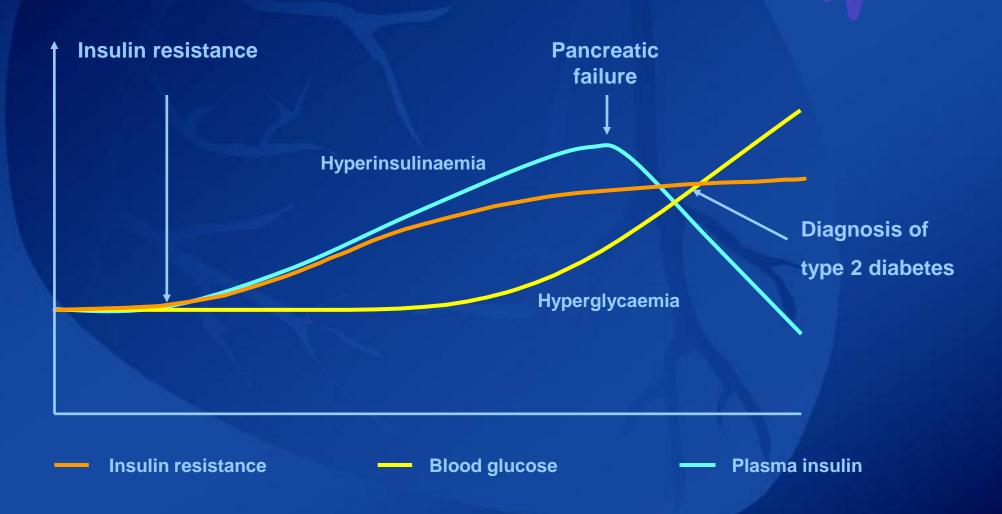
# Model of underlying factors in type 2 diabetes: insulin resistance and β-cell dysfunction



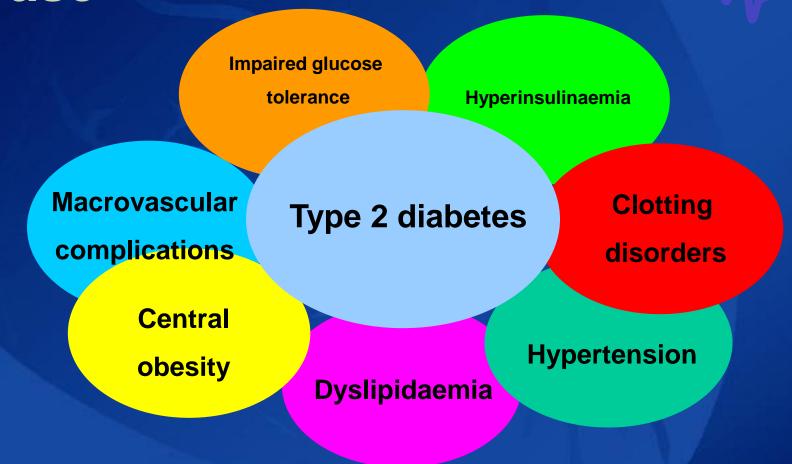
## **Underlying causes of Type 2 Diabetes**



## Underlying factors in type 2 diabetes are insulin resistance and β-cell dysfunction



## Type 2 Diabetes: A Complex Disease



### Other types of diabetes

- Genetic defects: effect on beta cell function or insulin action MODY, type A insulin resistance, leprechaunism, LADA
- <u>Diseases of the pancreas:</u> pancreatitis, trauma & infection, cancer, pancreatectomy
- <u>Endocrinopathies:</u> e.g.Cushing Syndrome (adrenal tumour)/Acromegaly antagonist effect of hormones - GH, glucagon, cortisol, adrenaline.
   Above syndromes may cause diabetes but typically when hormone levels reduced return to normoglycaemia
- Polycystic Ovarian Syndrome: ovary secretes high levels of oestregens and testosterone contributing to insulin resistance & weight gain (central) which may contribute to risk of type 2 DM
- Gestational Diabetes: type 2 diabetes in pregnancy carbohydrate intolerance leading to hyperglycaemia – OGTT @ 24-28 weeks (third trimester) - classification to type 2 diabetes 6 weeks after delivery with OGTT. Screen for those with higher risk
- Drug induced:

#### **DRUGS CAUSING DIABETES**

#### **Drugs That Affect Insulin Secretion**

Anticonvulsant

Phenytoin

Diuretics

Thiazides

Furosemide

Ethacrynic acid

Cations

Barium

Barium

Cadmium Lithium

Potassium

Zinc

Hormones

Somatostatin

Pesticides DDT

Fluoride

Pyriminil (Vacor)

Antihelminthics

Pentamidine

Antineoplastics

L-Asparaginase

Mithramycin

#### Drugs that affect insulin action

Hormones

Growth hormone

#### Drugs that affect both insulin secretion and insulin action

Hormones/Hormone Antagonists

Glucagon

Glucocorticoids

Octreotide

Adrenergic compounds

Epinephrine

Norepinephrine

Antihypertensive

Clonidine

Diazoxide

Prazosin

Blocking agents

β-Adrenergic blockers

Calcium-channel blockers

Histaminergic blockers

Psychopharmacologic agents

Benzodiazepines

Ethanol

**Opiates** 

Phenothiazines

# What are the likely presenting symptoms of diabetes?

# Presenting symptoms of type 1 and type 2 diabetes Thirst

- Hunger
- Fatigue Lethargic type 2
- Polyuria (type 1) Nocturia type 2
- Weight loss (type 1) May be less evident type 2
- Blurred Vision Less so in type 2
- Candidal infections
- Sores (ulcers) fail to heal or delayed
- May be no symptoms with type 2
- May present with complication related to diabetes e.g. retinopathy, CVD, foot problems

## Diagnosis- WHO (2018)

#### Methods and criteria for diagnosing diabetes

- 1.Diabetes symptoms (e.g. polyuria, polydipsia and unexplained weight loss for Type 1) plus:
  - 1. a random venous plasma glucose concentration ≥ 11.1 mmol/l or
  - 2. a fasting plasma glucose concentration ≥ 7.0 mmol/l (whole blood ≥ 6.1 mmol/l) or
  - 3. two hour plasma glucose concentration ≥ 11.1 mmol/l two hours after 75g anhydrous glucose in an oral glucose tolerance test (OGTT).
- 2. With no symptoms diagnosis should not be based on a single glucose determination but requires confirmatory plasma venous determination. At least one additional glucose test result on another day with a value in the diabetic range is essential, either fasting, from a random sample or from the two hour post glucose load. If the fasting random values are not diagnostic the two hour value should be used.

Consider further investigation in adults that involves measurement of C-peptide and/or diabetes-specific autoantibody titres (Type 1).

## Diagnosis of diabetes

For type 1 diagnosis likely to be based on presence of ketones and presenting symptoms especially in child. For type 2 tests on separate days if no symptoms.

**Diagnostic Guidelines** 

Symptoms eg thirst, polyuria, weight loss, fatigue, visual disturbances

**Diagnostic tests** 

•HbA1c > 48 mmol/mol (6.5%)

Plasma Glucose (Laboratory Test)

•Random ≥ 11.1 mmol/l

•Fasting ≥ 7.0 mmol/l

If asymptomatic, confirm diagnosis with repeat test on another day

**TEST URINE FOR KETONES** 

## Impaired Fasting Glucose & Impaired Glucose Tolerance

- IFG = elevated fasting glucose levels
   6.1-6.9 mmol/L
- IGT = Impaired Glucose tolerance
   7.8 mmol/L = 11.1mmol/L

In an adult with both tests abnormal the risk of developing diabetes after 10 years is 50%

#### Type 1 vs. type 2 diabetes

Lambert P, et al. Medicine 2006; 34(2): 47-51

Nolan JJ. Medicine 2006; 34(2): 52-56

#### Features of type 1 diabetes

- Onset in childhood/adolescence
- Lean body habitus
- Acute onset of osmotic symptoms
- Ketosis-prone
- High levels of islet autoantibodies
- High prevalence of genetic susceptibility
- Diagnosis on symptoms, history and presence of hyperglycaemia & ketones

#### Features of type 2 diabetes

- *Usually* presents in over-30s (but also seen increasingly in younger people)
- Associated with overweight/obesity
- Onset is gradual and diagnosis often missed (up to 50% of cases)
- Not associated with ketoacidosis, though ketosis can occur
- Immune markers in only 10%
- Family history is often positive with almost 100% concordance in identical twins
- Diagnosis typically HbA1c, FPG or OGTT with one or more symptoms

## **Treatment of Adults with type 1 diabetes**

- At the time of diagnosis (or if necessary after the management of critically decompensated metabolism), the diabetes professional team should develop with and explain to the adult with type 1 diabetes a plan for their early care.
- Offer all adults with type 1 diabetes a structured education programme of proven benefit, for example the <u>DAFNE</u> (dose-adjustment for normal eating) programme. Offer this programme 6–12 months after diagnosis.
- Dietary management
- Physical activity
- Blood glucose management
- Support adults with type 1 diabetes to aim for a target HbA1c level of 48 mmol/mol (6.5%) or lower, to minimise the risk of long-term vascular complications
- Advise routine self-monitoring of blood glucose levels for all adults with type 1 diabetes, and recommend testing at least
   4 times a day, including before each meal and before bed
- Consider real-time continuous glucose monitoring for adults with type 1 diabetes who are willing to commit to using it at least 70% of the time and to calibrate it as needed,

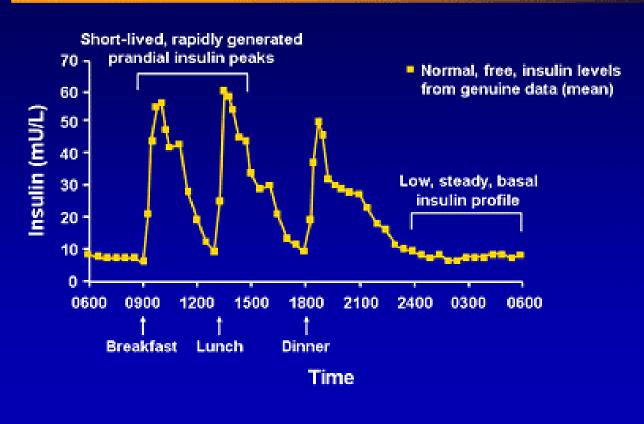
## Treatment of Adults with type 1 diabetes

- Insulin therapy
- Insulin regimens
- Offer multiple daily injection basal—bolus insulin regimens, rather than twice-daily mixed insulin regimens, as the insulin injection regimen of choice for all adults with type 1 diabetes. Provide the person with guidance on using multiple daily injection basal—bolus insulin regimens. [new 2015]
- Do not offer adults newly diagnosed with type 1 diabetes non-basal—bolus insulin regimens (that is, twice-daily mixed, basal only or bolus only). [new 2015]

Type of Insulin	Brand Name	Generic Name	Onset	Peak	Duration
Rapid-acting	NovoLog	Insulin aspart	15 minutes	30 to 90 minutes	3 to 5 hours
	Apidra	Insulin glulisine	15 minutes	30 to 90 minutes	3 to 5 hours
	Humalog	Insulin lispro	15 minutes	30 to 90 minutes	3 to 5 hours
Short-acting	Humulin R	Regular (R)	30 to 60 minutes	2 to 4 hours	5 to 8 hours
	Novolin R		50 to 60 minutes	2 to 4 flours	5 to 6 nours
Intermediate-acting	Humulin N	NPH (N)	1 to 3 hours	8 hours	12 to 16 hours
	Novolin N		T to 5 hours	o nours	12 to 16 hours
Long-acting	Levemir	Insulin detemir	1 hour	Peakless	20 to 26 hours
	Lantus	Insulin glargine	i nour	Peakless	20 to 26 nours
Pre-mixed NPH	Humulin 70/30	70% NPH and	30 to 60 minutes	Varies	10 to 16 hours
(intermediate-acting)	Novolin 70/30	30% regular			
and regular (short-	Humulin 50/50	50% NPH and	30 to 60 minutes	Varies	10 to 16 hours
acting)		50% regular			
Pre-mixed insulin lispro	Humalog Mix	_	10 to 15 minutes	Varies	10 to 16 hours
F	75/25	protamine and 25%			
(intermediate-acting) and insulin lispro (rapid-		insulin lispro	40. 45	. ·	40 . 40 !
	Humalog Mix 50/50	50% insulin lispro protamine and 50%	10 to 15 minutes	Varies	10 to 16 hours
		insulin lispro			
Pre-mixed insulin aspart	NovoLog Mix	70% insulin	5 to 15 minutes	Varies	10 to 16 hours
	70/30	aspart protamine			
(intermediate-acting)		and 30% insulin			
and insulin aspart (rapid-		aspart			
acting)					

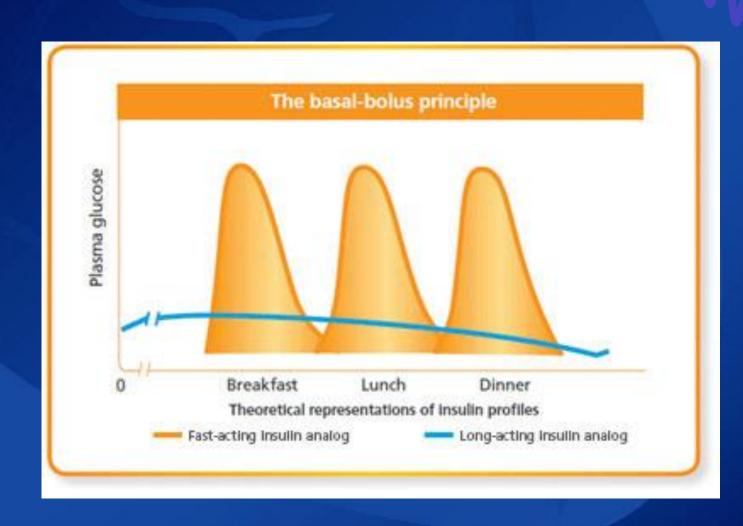
#### **Insulin Action**

### The Physiologic Insulin Profile



Adapted with permission from Polonsky KS. N Engl J Med. 1988;318:1231-1239.

## **Basal-Bolus principle**



## Treatment of adults with type 2 diabetes



SECOND INTENSIFICATION

- insulin-based treatment

- triple therapy with:

Consider:

If HbA1c rises to 58 mmol/mol (7.5%):

o metformin, a DPP-4i and an SU

o metformin, pioglitazone<sup>a</sup> and an SU

o metformin, pioglitazone or an SU, and an SGLT-2ib

Support the person to aim for an HbA1c level of 53 mmol/

#### Algorithm for blood glucose lowering therapy in adults with type 2 diabetes

- Reinforce advice on diet, lifestyle and adherence to drug treatment.
- Agree an individualised HbA1c target based on: the person's needs and circumstances including preferences, comorbidities, risks from polypharmacy and tight blood glucose control and ability to achieve longer-term risk-reduction benefits. Where appropriate, support the person to aim for the HbA1c levels in the algorithm. Measure HbA1c levels at 3/6 monthly intervals, as appropriate. If the person achieves an HbA1c target lower than target with no hypoglycaemia, encourage them to maintain it. Be aware that there are other possible reasons for a low HbA1c level
- Base choice of drug treatment on: effectiveness, safety (see MHRA guidance), tolerability, the person's individual clinical circumstances, preferences and needs, available licensed indications or combinations, and cost (if 2 drugs in the same class are appropriate, choose the option with the lowest acquisition cost).
- Do not routinely offer self-monitoring of blood glucose levels unless the person is on insulin, on oral medication that may increase their risk of hypoglycaemia while driving or operating machinery, is pregnant or planning to become pregnant or if there is evidence of hypoglycaemic episodes.

If the person is symptomatically hyperglycaemic, consider insulin or an SU. Review treatment when blood glucose control has been achieved. II the person is symptomatically hypothysical management of the second s ADULT WITH TYPE 2 DIABETES WHO CAN TAKE METFORMIN If HbA1c rises to 48 mmol/mol (6.5%) on lifestyle If standard-release interventions: metformin is not Offer standard–release metformin tolerated, consider a • Support the person to aim for an HbA1c level of 48 mmol/ trial of modified-release metformin If triple therapy is not FIRST INTENSIFICATION effective, not tolerated If HbA1c rises to 58 mmol/mol (7.5%): or contraindicated, Consider dual therapy with: consider combination - metformin and a DPP-4i therapy with metformin, - metformin and pioglitazone an SU and a GLP-1 - metformin and an SU mimetic<sup>c</sup> for adults with - metformin and an SGLT-2ib type 2 diabetes who: Support the person to aim for an HbA1c level of 53 mmol/ have a BMI of 35 kg/m<sup>2</sup> mol (7.0%) or higher (adjust accordingly for people from black, Asian and other minority ethnic groups)

METFORMIN CONTRAINDICATED OR NOT **TOLERATED** If HbA1c rises to 48 mmol/mol (6.5%) on lifestyle interventions:

- Consider one of the following<sup>a</sup> - a DPP-4i, pioglitazonea or an SU
- an SGLT-2ib instead of a DPP-4i if an SU or pioglitazone is not appropriate
- Support the person to aim for an HbA1c level of 48 mmol/mol (6.5%) for people on a DPP-4i, SGLT-2i or pioglitazone or 53 mmol/mol (7.0%) for people on an SU

#### FIRST INTENSIFICATION If HbA1c rises to 58 mmol/mol (7.5%):

- Consider dual therapy<sup>e</sup> with:
- a DPP-4i and pioglitazone
- a DPP-4i and an SU - pioglitazone<sup>a</sup> and an SU
- Support the person to aim for an HbA1c level of 53 mmol/mol (7.0%)

#### SECOND INTENSIFICATION

- If HbA1c rises to 58 mmol/mol (7.5%): Consider insulin-based treatment
- Support the person to aim for an HbA1c level of 53 mmol/mol (7.0%)

#### Insulin-based treatment

- When starting insulin, use a structured programme and continue metformin for people without contraindications or intolerance. Review the continued need for other blood glucose lowering therapies<sup>f</sup>
- Offer NPH insulin once or twice daily according to
- Consider starting both NPH and short-acting insulin either separately or as pre-mixed (biphasic) human insulin (particularly if HbA1c is 75 mmol/mol (9.0%) or
- Consider, as an alternative to NPH insulin, using insulin detemir or glargineg if the person; needs assistance to inject insulin, lifestyle is restricted by recurrent symptomatic hypoglycaemic episodes or would otherwise need twice-daily NPH insulin in combination with oral blood glucose lowering drugs.
- Consider pre-mixed (biphasic) preparations that include short-acting insulin analogues, rather than pre-mixed (biphasic) preparations that include shortacting human insulin preparations, if: the person prefers injecting insulin immediately before a meal. hypoglycaemia is a problem or blood glucose levels rise markedly after meals.
- Only offer a GLP-1 mimetic<sup>e</sup> in combination with insulin with specialist care advice and ongoing support from a consultant-led multidisciplinary teamh.
- Monitor people on insulin for the need to change the
- An SGLT-2i in combination with insulin with or without other antidiabetic drugs is an option<sup>b</sup>.

Abbreviations: DIPP-4|Dipeptidyl peptidase-4 inhibitor, GLP-1 Glucagon-like peptide-1, SGLT-2|Sodium-glucose cotransporter 2 inhibitors, SUSulfonylurea. Recommendations that cover DPP-4 inhibitors, GLP 1 mimetics and sulfonylureas refer to these groups of drugs at a class level.

a. When prescribing ploglitazone, exercise particular caution if the person is at high risk of the adverse effects of the drug. Pioglitazone is associated with an increased risk of heart failure, bladder cancer and bone fracture. Known risk factors for these conditions, including increased age, should be carefully evaluated before treatment: see the manufacturers' summaries of product characteristics for details. Medicines and Healthcare products Regulatory Agency (MHRA) guidance (2011) advises that 'prescribers should review the safety and efficacy of pioglitazone in individuals after 3-6 months of treatment to ensure that only patients who are deriving benefit continue to be treated b. See NICE technology appraisal guidance 288 & 418, 315 and 336 on dapagliflozin, canagliflozin and empagliflozin, respectively. All three SGLT-2 inhibitors are recommended as options in dual therapy regimens with metformin under certain conditions, as options in triple therapy regimens and in combination with insulin. All three are also options as monotherapies in adults in whom metformin is contraindicated or not tolerated. Serious and life-threatening cases of diabetic ketoacidosis have been reported in people taking SGLT-2 inhibitors (canagliflozin, dapagliflozin or empagliflozin) or shortly after stopping the SGLT-2 inhibitor. MHRA guidance (2015) advises testing for raised ketones in people with symptoms of diabetic ketoacidosis, even if plasma glucose levels are near normal.

c. Only continue GLP-1 mimetic therapy if the person has a beneficial metabolic response (a reduction of HbA1c by at least 11 mmol/mol [1.0%] and a weight loss of at least 3% of initial body weight in 6 months).

and specific psychological

or other medical problems

associated with obesity or

insulin therapy would have

implications, or weight loss

significant obesity-related

kg/m<sup>2</sup>, and for whom

would benefit other

comorbidities

significant occupational

have a BMI lower than 35

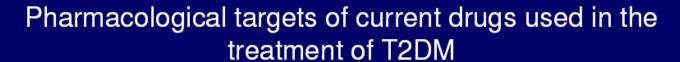
d. Be aware that, if metformin is contraindicated or not tolerated, repaglinide is both clinically effective and cost effective in adults with type 2 diabetes. However, discuss with any person for whom repaglinide is being considered, that there is no licensed non-metformin-based combination containing repaglinide that can be offered at first intensification.

e. Be aware that the drugs in dual therapy should be introduced in a stepwise manner, checking for tolerability and effectiveness of each drug.

f. MHRA guidance (2011) notes that cases of cardiac failure have been reported when pioglitazone was used in combination with insulin, especially in patients with risk factors for the development of cardiac failure. It advises that if the combination is used, people should be observed for signs and symptoms of heart failure, weight gain, and oedema. Pioglitazone should be discontinued if any deterioration in cardiac status occurs

g. The recommendations in this guideline also apply to any current and future biosimilar product(s) of insulin glargine that have an appropriate Marketing Authorisation that allows the use of the biosimilar(s) in the same indication h. A consultant-led multidisciplinary team may include a wide range of staff based in primary, secondary and community care.

## **Type 2 Diabetes Treatment**



#### Incretin mimetics (exenatide injectable)

Improves glucose-dependent insulin secretion, suppresses glucagon secretion, slows gastric emptying

#### Biguanide (metformin)

Decreases insulin resistance

#### Sulphonylurea

Increase insulin secretion from pancreatic β-cells

#### Meglitinides

Increase insulin secretion from pancreatic β-cells

Liraglutide (injectable) is the first human GLP-1 analogue. Stimulates insulin secretion, inhibits glucagon secretion

#### **DPP-4** inhibitors

Increase endogenous GLP-1, stimulate insulin secretion, suppress glucagon release in a glucose dependent manner

#### Glitazones

Decrease insulin resistance

#### α-glucosidase inhibitors

Delay intestinal carbohydrate absorption

DDP-4=dipeptidyl peptidase-4; GLP-1=glucagon-like peptide-1 Adapted from Cheng AY, Fantus IG. CMAJ. 2005; 172: 213–226

## **Initial Management**

- History FBC lipids, FPG, U & E's
- Examination complications
- Explanation
- Explore current lifestyle
- Diet
- Activity
- ? Medications oral/injectable
- ? Monitoring glucose/ketones
- ? Follow up
- Education for self-management





## Diabetes Emergencies (Acute Complications)

- Hypoglycaemia
- "low blood glucose level : <3.5 mmols/l
- Can affect people treated with insulin (type 1 and 2) and those taking sulphonylureas Hospital management-

https://diabetes-resources-production.s3-eu-west-1.amazonaws.com/diabetes-storage/migration/pdf/JBDS%2520hypoglycaemia%2520position%2520%282013%29.pdf

- 1.Oral Glucose (short acting CHO)
- 2.Glugagon IM
- 3.IV Glucose

### **Diabetes Emergencies**

## Hypergylcaemia

- Polydypsia (excessive thirst)/ dry mouth
- Polyuria (passing urine frequently)/ incontinence
- Tiredness
- Blurred vision

### Diabetic Ketoacidosis

- Usually type 1
- ? Infection
- Rapid onset (hours to a few days)
- Polydypsia
- Weakness/lethargy
- Urine output high or low
- Deep and rapid respirations
- Ketonuria
- Ketone smell to breath
- Nausea, vomiting, abdo pain

Dehydrated – Tachycardia, hypotensive, flushed, dry mucous membranes) Alert or unconscious Weight loss Electrolyte imbalance

### **Diabetic Ketoacidosis**

- Ketone self-monitoring for prevention of DKA
- 1.11.1 Consider ketone monitoring (blood or urine) as part of 'sick-day rules' for adults with type 1 diabetes, to facilitate self-management of an episode of hyperglycaemia. [new 2015]
- Ketone monitoring in hospital
- 1.11.2 In adults with type 1 diabetes presenting to emergency services, consider capillary blood ketone testing if:
- DKA is suspected or
- the person has uncontrolled diabetes with a period of illness, and urine ketone testing is positive. [new 2015]
- 1.11.3 Consider capillary blood ketone testing for inpatient management of DKA in adults with type 1 diabetes that is incorporated into a formal protocol. [new 2015]

### Management of DKA

- 1.11.4 Professionals managing DKA in adults should be adequately trained, including regular updating, and be familiar with all aspects of its management which are associated with mortality and morbidity. These topics should include:
- fluid balance
- acidosis
- cerebral oedema
- electrolyte imbalance
- disturbed interpretation of familiar diagnostic tests (white cell count, body temperature, ECG)
- respiratory distress syndrome
- cardiac abnormalities
- precipitating causes
- infection management, including opportunistic infections
- gastroparesis
- use of high dependency and intensive care units

### **DKA** treatment

- For primary fluid replacement in adults with DKA, use isotonic saline, not given too rapidly except in cases of circulatory collapse. [2004]
- 1.11.6 Do not generally use bicarbonate in the management of DKA in adults. [2004, amended 2015]
- 1.11.7 Give intravenous insulin by infusion to adults with DKA. [2004]
- 1.11.8 In the management of DKA in adults, once the plasma glucose concentration has fallen to 10–15 mmol/litre, give glucose-containing fluids (not more than 2 litres in 24 hours) in order to allow continued infusion of insulin at a sufficient rate to clear ketones (for example, 6 units/hour monitored for effect). [2004, amended 2015]
- 1.11.9 Begin potassium replacement early in DKA in adults, with frequent monitoring for the development of hypokalaemia. [2004]
- 1.11.10 Do not generally use phosphate replacement in the management of DKA in adults. [2004, amended 2015]
- 1.11.11 In adults with DKA whose conscious level is impaired, consideration should be given to inserting a nasogastric tube, monitoring urine production using a urinary catheter and giving heparin. [2004]
- 1.11.12 To reduce the risk of catastrophic outcomes in adults with DKA, ensure that monitoring is continuous and that review covers all aspects of clinical management at frequent intervals. [2004, amended 2015]

## Diabetes as a long term condition

- Principle- Individualised Shared Care Plan
- Self –management/ self care.
- Service level interventions.
- Surveillance.
- Screening.
- -Education

Via- Primary Care Led Services.

### **Education**

http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD005268.pub2/full

### **Self-management**

Self-management education programmes by lay leaders for people with chronic conditions http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD005108.pub2/full

### **Service Interventions:**

Interventions to improve the management of diabetes mellitus in primary care, outpatient and community settings http://onlinelibrary.wiley.com/doi/10.1002/14651858.CD001481/full Cochrane Library 2017

### **Care Plan Mutual Goals**

- Height and weight (BMI= kg/m²)
- Blood pressure
- Blood glucose control
- HbA1c and cholesterol levels
- Discuss any issues
- Advise any change in regime, lifestyle or medication including any side effects
- People who take <u>insulin</u> should also have their injection or <u>infusion sites</u> checked.
- Depression and <u>sexual dysfunction</u> questionnaire.

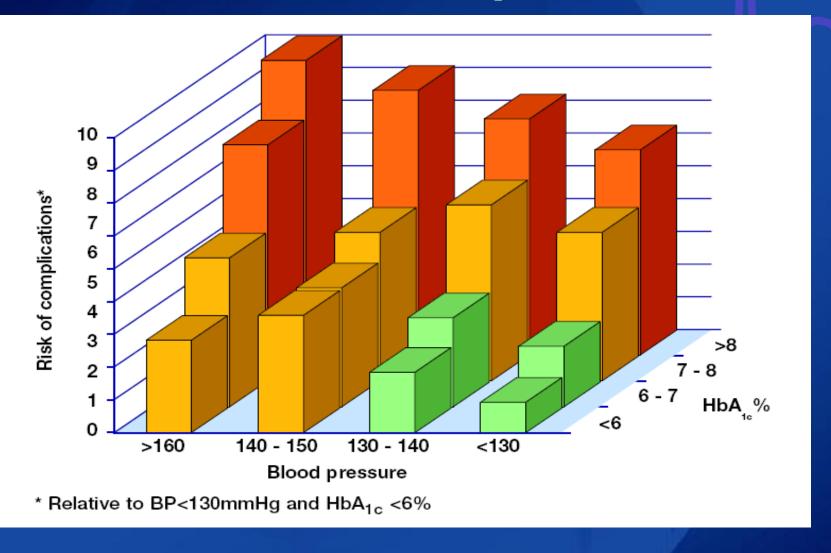
### **Care Plan Mutual Goals**

- HbA1c targets
- People with diabetes are generally advised to keep their HbA1c values below 48 mmol/mol (6.5%)
- However, an individualised target should be set to take into account factors such as your daily activities, likelihood of developing complications and risks presented by <a href="https://hypoglycemia.">hypoglycemia</a>.
- Ideally, the lower the HbA1c value you can achieve without increasing instances or severity of hypoglycemia, the better.
- For comparison purposes, people without diabetes tend to get HbA1c readings in the 15 to 37 mmol/mol (3.5 to 5.5%) range.
- Blood pressure (hypertension) guidelines
- Blood pressure targets for type 1 diabetes:
- Below 135/85 mmHg
- Or **below 130/80 mmHg** if abnormal presence of albumin in the urine (a sign of diabetic kidney disease) or if there are signs of metabolic syndrome
- Blood pressure targets for type 2 diabetes:
- Below 140/80 mmHg
- Or below 130/80 mmHg if you have kidney disease, retinopathy or have cerebrovascular disease (including stroke)
- Total cholesterol: under 4.0 mmol/l
- LDL levels: below 2.0 mmol/l
- **HDL levels:** at least 1.0 mmol/l (men) or 1.2 mmol/l (women)
- Triglyceride levels: less than (or equal to) 1.7 mmol/l
- Kidney function guidelines for diabetic patients are as follows:
- Albumin/creatinine ratio: less than or equal to 2.5 mg/mmol (men) or 3.5 mg/mmol (women)

## **Self-Management**

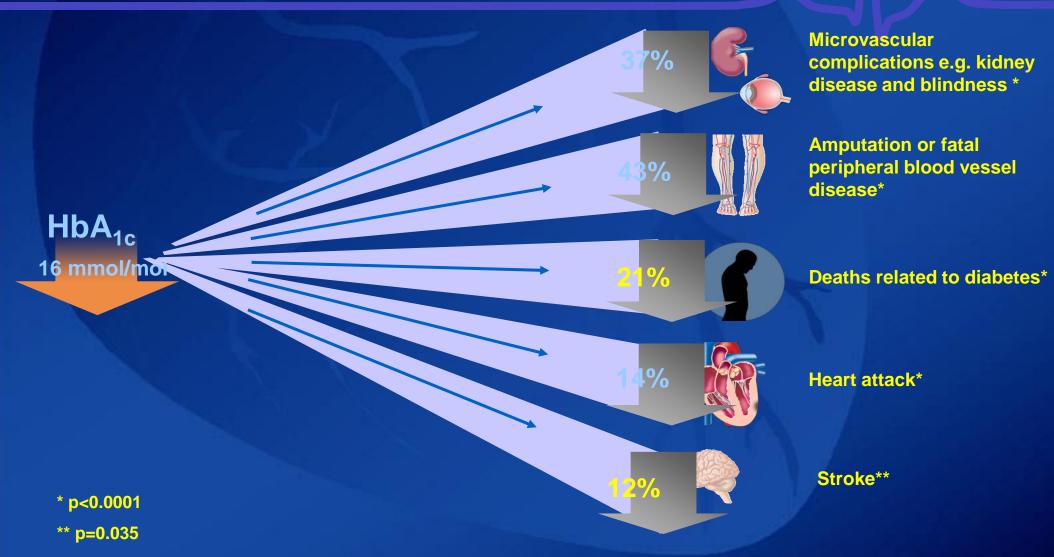
- Physical Activity/ Exercise.
- Dietary management.
- Mediation compliance.
- Psychological well-being.
- Control- (Blood Glucose Monitoring/ Dose adjustment/ Nutritional adjustment).
- Education
- Dose Adjustment for Normal Eating (DAFNE)- Type 1http://www.dafne.uk.com/
- -Diabetes Education and Self Management for Ongoing and Diagnosed (DESMOND)- Type 2
- http://www.desmond-project.org.uk/index.php

## Risk of diabetes complications

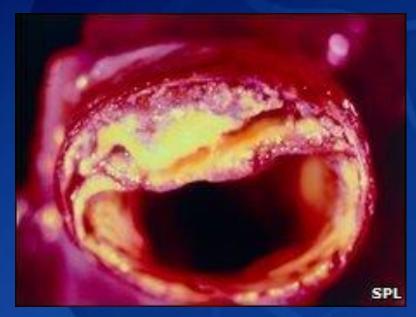


The risk of diabetes complication based on the UKPDS Study. From Mogensten C-E. Diabetic nephropathy:evidence for renoprotection and practice. *Heart* 2000; 84(suppl): i26 -28. Reproduced with permission from the BMJ Publishing Group.

## UKPDS: 1% (16 mmol/mol) decrease in HbA<sub>1c</sub> is associated with a reduction in complications



## Macro-vascular complications of diabetes





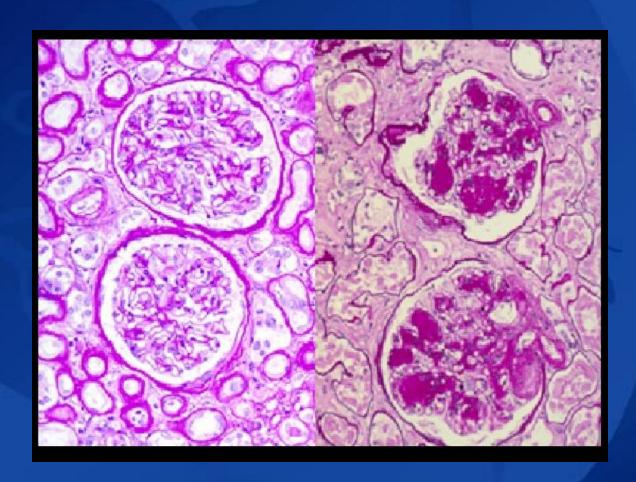


## Micro-vascular complications

- Diabetic Retinopathy
- <a href="https://www.youtube.com/watch?v=mHBRxQD4ef4">https://www.youtube.com/watch?v=mHBRxQD4ef4</a>



## Micro-vascular complications



Kidney disease

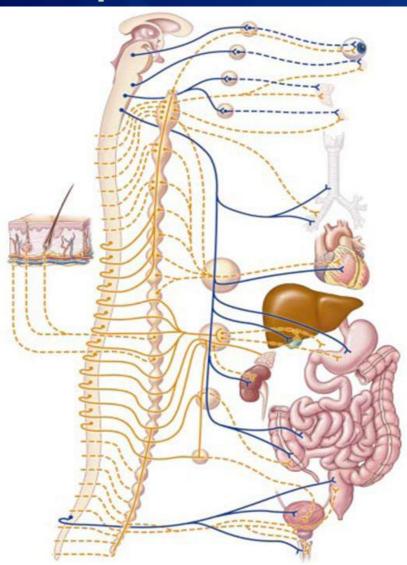
Diabetic Nephropathy

## Micro-vascular complications



Diabetic Neuropathy

## Other complications



# Diabetic autonomic neuropathy:

### Pupillary

Decreased diameter of darkadapted pupil Argyll-Robertson type pupil

### Metabolic

Hypoglycemia unawareness Hypoglycemia unresponsiveness

### Cardiovascular

Tachycardia, exercise intolerance Cardiac denervation Orthostatic hypotension Heat intolerance

### Neurovascular

Areas of symmetrical anhydrosis Gustatory sweating Hyperhidrosis Alterations in skin blood flow

### Gastrointestinal

Constipation
Gastroparesis diabeticorum
Diarrhea and fecal incontinence
Esophageal dysfunction

### Genitourinary

Erectile dysfunction Retrograde ejaculation Cystopathy Neurogenic bladder Defective vaginal lubrication