Week 3

- **Endocrine & Diabetes**

- **Normal insulin secretion**
- insulin is a hormone produced by the beta cells in the islets of Langerhans of the pancreas
- released continuously into blood stream in small increments (basal) and large releases (bolus) when food is ingested
- it moves glucose from food into cells and therefore lowers BSL
- normal without diabetes is <6.0mmol/L fasting and <7.8mmol/L, 2 hours after food
- with diabetes 6.0-8.0 fasting, 6.0-10.0 2hr after food

- **Ketones**
- Ketones are produced during oxidation of fatty acids due to insufficient insulin to help fuel the body's cells
- ketones attack myelinated sheath around neurone (nerve cell) making it brittle and snap off, breaking signal along nerve cell—> effecting peripheral nervous system—> peripheral neuropathy. Heart has no nerves so pain during MI is referred to jaw/arm, causing silent MI in some diabetics
- Ketones tested when
  - BSL >15
  - hrly when monitoring DKA
- **Blood test**: Abbott Freestyle Optium meter (BSL test)
- tests for betahydroxybuterate, main ketone body in DKA, more accurately reflects risk of and recovery from DKA
- **Urine test:** tests acetoacetate (only use if blood test strips are unavailable)
- When acidosis is improving betahydroxyburate converts to acetocaetate and is excreted in urine —> elevated level in urine when condition is actually improving, less accurate when correlated with blood pH

- **Diabetic ketoacidosis**
- life threatening metabolic situation resulting from high BSL
- cells are unable to get glucose needed for energy due to lack of insulin—>increase in counter-regulatory hormones (glucagon, cortisol, growth hormone, epinephrine), body begins to breakdown fat/muscle for energy producing ketones in blood causing metabolic acidosis (DKA)
- Biochemical criteria:
  - BSL >11mmol/L
  - Venous pH <7.3 serum Bicarbonate < 15mmol/L

- **Diabetes Mellitus**
- metabolic disorder of multiple aetiologies, characterised by chronic hyperglycaemia with disturbances of carbohydrate, fat and protein metabolism, resulting from defects in insulin secretion and/or action
- type , type 2, gestational, secondary (develops from other condition)
  - **Type 1:**
  - mostly diagnosed in childhood/adolescence but can be in adulthood
  - must have insulin
  - autoimmune process leading to destruction of insulin producing beta cells of the Islets of Langerhans in pancreas. When Beta cells are destroyed, insulin is not produced—> inability to turn glucose, to glycogen to be used by body through glycolysis—> body burns fat instead —> ketosis
  - 10 to 15% of the total number of people with DM Australia

  - **Cause**
  - genetic susceptibility (associated with histocompatibility leukocyte antigen HLA)
    - viral or chemical trigger causes auto antibodies within the islet cells. These antibodies respond to normal islet beta cells as though they were foreign substances, destroying them

- **Signs/symptoms**
- **(early)**
- high BSL >11.1mmol/L
- polyuria
- polydipsia
- thrush
- vomiting, chronic weight loss
- irritability
- recurrent skin infections
- **(late)**
- severe dehydration
- frequent vomiting/abdo pain
- polyuria despite dehydration
- weight loss
- flushed cheeks
- acetone breath
- hyperventilation
- disorientated
- shock: rapid pulse, poor peripheral circulation
- hypotension (rare)

- **Diagnosis**
- BSL elevated > 11.1mmol/L
- ketones elevated > 0.6mmol/L
- markers of immune destruction (autoantibodies)
- islet Cell (ICA), Glutamic acid decarboxylase (GAD), IA-2, Insulin (IAA) = positive (results take 2 weeks)
- HbA1c (glycated haemoglobin) usually > 6.0%
- fasting insulin or C-Peptide= low

- **Type 2:**
- pancreas produces insulin but insufficient amount or is poorly utilised by tissues (insulin resistance)
- usually oral meds, some require insulin
- any age, most common >40 years
- BSL rises when the glucose cannot move into the cells

- **Risk factors**
- family history
- obesity
- inactivity
- women with previous gestational diabetes
- Aboriginal, Torres Strait Islander, Asian, African

- **Signs/symptoms**
- slow onset of symptoms
- polyuria
- polydipsia
- lethargy
- recurrent infections
- thrush
- glycosuria
- blurred vision
- often no symptoms: random BGL elevated, or glucose in urine
- diabetic Ketoacidosis (rare)