Diabetes mellitus

Health & Medicine, Diabetes

Diabetes Mellitus Diabetes Mellitus (DM) — “ a chronic, progressive disease characterized by the body’s inability to metabolize carbohydrates, fats, and proteins, leading to hyperglycaemia (high blood glucose level)" (Black & Hawks, 2009, p. 1062) Epidemiology: Pathophysiology Overview According to Canadian Chronic Disease Surveillance System, “[i]n 2008/09, close to 2. 4 million Canadians aged 1 yr and older were living w/ diagnosed diabetes (either type 1 or type 2)" making diabetes as one of the most common chronic diseases in Canada (Public Health Agency of Canada, 2011). most common chronic disease among children and youth, particularly Type 2 since it has been on the rise globally for the last two decades (Public Health Agency of Canada, 2011) In a decade (1998/99-2008/09), diabetes’ prevalence in Canada increased by 70% (Public Health Agency of Canada, 2011) More prevalent in males (5. 4%) than females (4. 4%) (Sanmartin & Gilmore, 2008) Prevalence of Type 2 diabetes in lowest income group is 4. 14X higher than in the highest income group in Canada (Dinca-Panaitescu et al., 2011) Prevalence of diabetes were more than 4X higher among First Nations than among non-First nations people (Dyck et al., 2010) Those w/ diabetes are over 3X more likely to be hospitalized w/ cardiovascular disease than individuals without diabetes (Public Health Agency of Canada, 2011) 11% of Canadian adults w/ diabetes were reported to have 2 or more other serious chronic conditions besides diabetes (Canadian Diabetes Association, 2008) Glucose-regulating hormones (secreted by the pancreas’ Islets of Langerhans): Alpha cells = secrete glucagon (â†‘ glucose) Î² —cells = secrete INSULIN (â†“ glucose) delta cells = secrete somatostatin (inhibit release of glucagon + insulin to extend time of nutrient absorption by tissues) Incretin

— GI hormone that â†‘ insulin release of Î²-cells Glucose transporters = “ special carriers" that help move glucose from blood into the cell Glucogenesis — breakdown of glycogen to form glucose Gluconeogenesis — breakdown of non-carbohydrate sources to form glucose Diabetes Overview TARGET CELLS Summary of comparisons between Type 1 & Type 2 diabetes: | Type 1 | Type 2 | Also known as: | “ juvenile-onset" or “ insulin-dependent diabetes mellitus" (IDDM) | “ adult-onset" or “ non-insulin-dependent diabetes mellitus" (NIDDM) | Description: | Destruction of pancreatic Î²-cells by Islet cell antibodies leading to insulin deficiencyType1A = “ autoimmune" — genetic predisposition or environmental factorsType1B = idiopathic | Insulin resistance — “ resistance to biological activity of insulin in both the liver and peripheral tissues" (Black & Hawks, 2009, p. 1066) ex. due to defective insulin receptors Insulin production of inactive insulin, or impaired release of insulin d/t exhausted Î²-cells etc. | Age of Onset | Typically < 30 years of age | < 40 years of age | Pancreatic Function | Little to none | Insulin may be in low, normal, or high amounts | Clinical Presentation | Acute or rapidly progressive hyperglycemia w/ symptoms of polyuria, polydipsia, fatigue, sudden weight loss, slow wound healing, â†‘ appetite, blurred vision, thrush or genital infections, neuropathy, and ketoacidosis | Similar to Type 1 except more chronic and progressive symptoms | Genetic Predisposition | Yes | Yes, heredity applies | Environmental factors: | Virus infection (triggers autoimmune process) | â†“ physical activity, obesity (particularly central obesity) — “ adipokines" (adipose tissue cells release these cytokines that have shown to cause cellular resistance to insulin) | (Porth, 2007; Black & Hawks, 2009; Thevenod, 2008) References Black, J. M., & Hawks, J. H. (2009).

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