### Clinical **DIGEST 1**

## **Management of type 1 diabetes**

#### Type 1 and type 2 diabetes – what is the difference?



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ype 2 diabetes remains a challenge – we know that insulin resistance is important, but we also know that some individuals appear to be able to compensate by increasing insulin secretion whereas others do not. The debate continues as to why only some individuals

develop glucose intolerance. At least we feel we understand type 1 diabetes.

Glucose intolerance develops because of an autoimmune attack on the beta cell. Individuals develop the condition because of a genetic susceptibility and bad luck – they happen to be exposed to an antigen that triggers an immune response. We are all quite comfortable that it has nothing to do with insulin resistance or the continuing epidemic of overweight/obesity!

Three recent articles would challenge this simplistic view. The paper by Brookes-Worrell and colleagues (2004) demonstrated that autoimmune markers of type 1 diabetes can be seen in children recently diagnosed with what was thought to be type 2 diabetes, and that

markers of insulin resistance such as obesity or acanthosis nigricans do not distinguish between individuals with or without islet cell autoimmunity.

Schölin et al (summarised below) also suggest that measures of fatness are important in predicting the clinical course of type 1 diabetes. Being underweight resulted in a low remission rate. Being overweight resulted in a higher rate of remission but the length of remission was considerably shorter. They, like others, suggest that in an insulin resistant state, the beta cell is under stress and that a number of processes (including an autoimmune attack) may result in beta cell failure and diabetes (Wilkin, 2001). What this is saying is that there is really no clear distinction between type 1 and type 2 diabetes.

This may explain the parallel rise in both type 1 and type 2 diabetes seen around the world, which is illustrated in the paper by Haynes et al, summarised on the right.

Brooks-Worrell BM, Greenbaum CJ, Palmer JP, Pihoker C (2004) Autoimmunity to islet proteins in children diagnosed with newonset diabetes. *J Clin Endocrinol Metab* **89**(5):2222–27 Wilkin TJ (2001) The accelerator hypothesis: weight gain as the missing link between Type I and Type II diabetes. *Diabetologia* **44**(7): 914–22

# DIABETIC MEDICINE

# Remission promoted by normal weight

Readability 
Applicability to practice 
WOW! factor 
V 
V

- Previous studies have indicated that the recovery of beta cell function and improvement of insulin sensitivity are thought to underly clinical remission in newly diagnosed type 1 diabetes patients who have been initiated on insulin therapy.
- This study aimed to identify factors (clinical, biochemical and immunological) that promote remission and prolong its duration in a large sample of young patients with type 1 diabetes.

HbA<sub>1c</sub> levels and insulin dosage were recorded for 362 type 1 diabetes patients at local Swedish hospitals for an average of 5 years after diagnosis. The length of remission was also analysed in light of the conditions upon diagnosis.

Regression analysis showed that normal weight was the only consistent factor that significantly affected the likelihood of remission. Furthermore, among patients with islet antibodies, a low number of antibodies was associated with long remission periods.

Whilst a low number of islet antibodies was important in relation to remission duration, normal body weight was the most significant factor promoting remission.

Schölin A, Törnt C, Nyström L et al (2004) Normal weight promotes remission and low number of islet antibodies prolong the duration of remission in Type 1 diabetes. *Diabetic Medicine* **21**: 447–55

#### DIABETOLOGIA



# Continued increase in childhood-onset type 1 diabetes

An unexplained change in the epidemiology of childhood-onset type 1 diabetes has been previously documented in various countries.

Epidemiological studies are an important method of determining the aetiological factors that might help explain these changes.

This study aimed to elucidate the incidence of type 1 diabetes in Western Australian children aged 0–14 between 1985 and 2002.

Initial incidence data were gained from a prospective diabetes register set up in 1987. Further information was obtained from the Western Australia Hospital Morbidity Data System. The Australian Bureau of Statistics was the source for denominator data.

The incidence rates were analysed by calendar year, patient sex and age upon diagnosis using the Poisson regression techniques.

Out of 1144 cases, on average the incidence of the disease increased by more than 3 % year on year. No significant between-sex differences were observed, and an increase was seen in every age group.

The authors concluded that in Western Australia the incidence of childhood-onset type 1 diabetes increased significantly between 1985 and 2002. A higher rate of increase was not observed in the youngest age groups, in contrast to other studies.

Haynes A, Bower C, Bulsara MK, Jones TW, Davis EA (2004) Continued increase in the incidence of childhood Type 1 diabetes in a population-based Australian sample (1985–2002). *Diabetologia* **47**:866-70

### **Type 1 diabetes**



# Insulin detemir offers improved results over NPH

Readability	1	1	1	1	
<b>Applicability to practice</b>	1	1	1	1	1
WOW! factor	./	./	./	1	./

- Rapid-acting insulin analogues offer improvements in post-prandial glucose control, but are not ideal for use with NPH insulin. This is a significant barrier to achieving control targets.
- This study investigated whether insulin detemir (a long-acting soluble insulin analogue) offers improved glycaemic control over NPH insulin.
- A 16-week randomised clinical trial treated 408 type 1 diabetes patients with either insulin detemir or NPH insulin. Two different insulin detemir treatment regimens were tested (twice a day at 12 h intervals versus before breakfast and at bedtime).
- Fasting plasma glucose was lower in both insulin detemir patient groups compared with patients taking NPH insulin. Similarly, in the final 12 weeks of the trial, the risk of minor hypoglycaemic events was lower in insulin detemir patients compared to those taking NPH insulin. Furthermore, neither of the insulin detemir patient groups exhibited weight change over the trial, whereas those taking NPH insulin gained weight. Finally, for individual patients, the between-day variation in prebreakfast plasma glucose was lower for both detemir groups compared to those taking NPH insulin.
- Overall, the use of insulin detemir offered better glycaemic control compared to the use of NPH insulin.

Home P, Bartley P, Russel-Jones D et al (2004) Insulin deternir offers improved glycaemic control compared with NPH insulin in people with Type 1 Diabetes: a randomized clinical trial. *Diabetes Care* 27(5): 1081–87

#### DIABETIC MEDICINE

# Street drugs and diabetes control

- After a person with type 1 diabetes was admitted with ecstasy-related DKA, the researchers aimed to determine the level of street drug use amonst their patients.
- An anonymous questionnaire was sent to 158 young adults with type 1 diabetes to determine the frequency of drug use. Of the respondents who admitted using drugs, 72 % were unaware of possible adverse effects on their diabetes.
- 3 Drug use amongst young adults with type 1 diabetes is common and may cause serious disease complications.

Ng RSH, Darko DA, Hillson RM (2004) Street drug use among young patients with Type 1 diabetes in the UK. dysfunction. *Diabetic Medicine* **21**: 295-96

### NEW ENGLAND JOURNAL OF MEDICINE

# No causal link with vaccination

- A link between the development of type 1 diabetes and childhood vaccinations has been previously proposed.
- The researchers evaluated a cohort of Danish children. Using regresson analysis, rate ratios for type 1 diabetes comparing vaccinated and unvaccinated children were estimated.
- The data do not support a link between childhood vaccination and type 1 diabetes.

Hviid A, Stellfield M, Wohlfahrt J, Melbye M (2004) Childhood vaccination and Type 1 diabetes. *N Engl J Med* **350**(14): 1398–404