



## Screening for type 2 diabetes

Tim Kenealey and colleagues' excellent article on screening for type 2 diabetes gives good guidance to general practitioners and wisely leaves options open to initiate an early therapeutic opportunity<sup>1</sup>.

As advocated for more than twenty years an excellent chance to estimate residual beta cell reserve function is when the patient turns fifty years of age. They can then be advised to have a really very large three course mid day birthday dinner (possibly with other older brothers and sisters?). They present at the laboratory exactly two hours after the meal has been finished. This time suits most people and laboratories. Values returned over 7.0mmol/L will suggest beta cell reserves less than 30% rather than the more likely 50% expected in the average New Zealander.

An oral glucose tolerance test giving 2 hour values 7.8 – 11mmol/L will allow a diagnosis of impaired glucose tolerance, always a forerunner of diabetes and low levels of beta cell residual capacity. Two hour values over 11.0mmol/L on the OGTT make an "official" diagnosis of type 2 diabetes.<sup>1</sup> In the United Kingdom there is usually a period of twelve years from onset to diagnosis.

Such two hour values relate much better to functioning residual beta cell mass, to future cardiovascular disease and to all cause mortality than do fasting blood glucose values alone – a late diagnostic feature.<sup>2-5</sup>

Diabetes NZ Inc, the Federation of thirty seven voluntary societies representing the 105 000 people with diabetes strongly supports earliest possible diagnosis set out in the Pricewaterhouse Coopers report of 2001.

Two other key issues emerge in respect to the widespread loss by midlife or earlier of non renewable beta cell function in so many people in New Zealand. It is now accepted that the earliest diagnosis is cost effective.<sup>6</sup> Evidence for this has been reviewed.<sup>7</sup> It is also now accepted<sup>8-10</sup> that 150 minutes of active exercise weekly and four visits yearly for dietetic advice on weight management and reduction of saturated fat intake prevents diabetes. Our high New Zealand weight and animal fat levels were not designed for the beta cells or liver enzymes of our huntergather ancestors.

Five fold increases in animal fat ingestion increases insulin demand and often body mass index and, unless modified, accelerate B cell destruction.

This 50% prevention of progression to diabetes with the above lifestyle changes is exciting but a less well known finding of the last five years.

Let us all now initiate action and do something modern about diabetes in our country.

Sue Benny,  
Diabetes Health Promoter.

Professor D W Beaven,  
Prof Emeritus,  
Diabetes Life Education and Patron Diabetes NZ,  
Christchurch.

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