EDITORIAL

ORIGINAL ARTICLES

• Diabetic symmetric peripheral neuropathy - Part 1: Diagnosis
• ‘Pre-diabetes’ - the risk it poses to South Africa and the role of lifestyle modification
• Carbohydrates, oxidative stress and low carbohydrate living
• The Role of Snacking in Diabetes
• The 16th Annual CDE Postgraduate Forum in Diabetes Management
The International Diabetes Federation theme for this past World Diabetes Day (14 November), and indeed the theme until 2016, is ‘Healthy living and diabetes’. In keeping with this, the final issue of SAJD for this year incorporates several relevant pieces.

What does healthy living actually mean? Dietitian, Hamish van Wyk writes on the practicalities of reducing risk by halting or delaying the progression of intermediate hyperglycaemia to diabetes using lifestyle approaches. He offers numerous solutions and simultaneously poses the valid question of whether or not this country has the ability and capacity to arrest the looming epidemic of diabetes. We continue our coverage of the concept of healthy living, but specifically with a nutritional slant, with a piece by Capetonian, Neville Wellington. His thoughts were well articulated in the annual ‘Ascending Star’ Lecture delivered at the 16th Annual CDE Postgraduate Forum. In a formal write-up of this lecture, Wellington puts forward his case for implementing a low carb lifestyle as an essential component in treating diabetes and obesity. Adding to the theme, dieticians Michelle Daniels and Liana Grobbelaar write on the role of snacking in diabetes. Accurate nutritional histories can often be difficult to elicit from people with diabetes, but by remembering to explore snacking behaviours, we will gain essential insights into the ‘goings-on’ in their lives. You may just unearth one reason for cryptic weight gain...

We offer an article by regular contributor David Webb on the under-appreciated topic of diabetic peripheral neuropathy. In the spirit of true clinical medicine, a good result often comes from accurate and insightful history taking followed by a thorough clinical examination.

Finally, Rosemary Flynn succinctly captures the essence of the most recent CDE Postgraduate Forum held in Gauteng this past August. We look forward to an even bigger and better meeting in 2015!

I wish to thank my tireless Sub-Editor, Michael Brown, for another great year behind the scenes. I am grateful for the dedication and wisdom you bring to our Journal. Michael and I are blessed to work with our Publisher, Angela Bell and Design and Layout Artist, Adèle Gouws. We thank you Angela for another bumper year and for your dogged determination and passion in supporting what we are trying to achieve. In Adèle, we could not wish for a more willing, fast, accurate and talented person to work with.

We wish you all an enjoyable and restful year’s end and a safe return to your homes and offices if you are going away.

Dr Stan Landau
Editor
email: Stan@cdecentre.co.za
“How can we extinguish a fire if we don’t first cut off the fuel that ignites the inferno?”  Arun Gandhi

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ORIGINAL ARTICLES
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The Role of Snacking in Diabetes
Ms Michelle Daniels and Ms Liana Grobbelaar

The 16th Annual CDE Postgraduate Forum in Diabetes Management - 8th to 10th August 2014
Weekend Overview
Mrs Rosemary Flynn
Diabetic symmetric peripheral neuropathy - Part 1: Diagnosis

Neuropathy is a common complication of type 1 and type 2 diabetes. The frequency increases with both age and duration of diabetes, such that neuropathy is prevalent in almost 10% of people with diabetes at time of diagnosis and in 40-50% after 10-20 years.

**Typical distal symmetric polyneuropathy**
The neuropathies of diabetes are classified into symmetric and asymmetric forms (Table 1). Typical distal symmetric polyneuropathy (DSPN) is the most common form, accounting for over 90% of cases. It is defined as "a symmetrical, length dependent sensorimotor polyneuropathy attributable to metabolic and microvessel alterations as a result of chronic hyperglycaemia exposure (diabetes) and cardiovascular risk covariates". Although it is primarily a sensory neuropathy and significant distal weakness is uncommon, subclinical motor involvement is usually demonstrable with nerve conduction studies.

**Diagnosis of DSPN**
In clinical practice, the diagnosis of DSPN is made by exclusion, based on history, the patient’s description of symptoms and clinical examination. Objective measures of nerve damage, which include nerve conduction studies, skin biopsy and corneal confocal microscopy may be used to confirm the diagnosis. Criteria for the diagnosis of DSPN are listed in Table 2.

**Clinical features of DSPN**
DSPN usually has an insidious onset with slow gradual progression of sensory symptoms starting in the toes and progressing proximally (stocking distribution). In more severe cases, once neuropathy is established in the legs, the upper limbs may also be involved with a similar progression of symptoms beginning in the proximal fingers. With advancing disease, motor symptoms and signs may include atrophy of the extensor and flexor muscles of the toe and small muscles of the hands, and limb weakness. In some patients, when sensory loss occurs without the patient being aware of it, foot ulceration, loss of balance, falls or other injuries related to loss of sensation may be the first presentation of DSPN.

Uncomfortable neuropathic symptoms may occur in the lower limbs in up to 50% of patients with DSPN. When taking a history from a patient with DSPN, it is important to differentiate between sensation loss (negative neuropathic sensory symptoms) and increased sensory (positive neuropathic sensory symptoms).
Table 3. Examples of positive neuropathic symptoms associated with dsPn

- Paraesthesia (uncomfortable tingling, skin crawling sensation).
- Pain and dysesthesias
  - burning
  - shooting
  - electric shocks
  - stabbing
  - crawling
  - aching
  - ‘numb-asleep’ sensation; e.g. like the sensation after lying too long on an arm
- Evoked pain
  - Allodynia (pain induced by light, moving stimuli, which are normally not painful)
  - Hyperalgesia (increased pain sensitivity to a stimulus that would normally be painful)
  - Summation (increasing pain sensation from repeated application of an identical single noxious stimulus. It may be produced by both mechanical and thermal stimuli)
  - Thermally evoked pain (cold and heat hyperalgesia)
- Unusual sensations
  - Feeling of swelling of the feet
  - Severe coldness of the legs
  - Sensations like walking on pebbles or hot sand

Table 2. Minimal criteria for defining DSPN

<table>
<thead>
<tr>
<th>Condition</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible DSPN</td>
<td>The presence of symptoms (decreased sensation, positive neuropathic sensory symptoms) predominantly of the toes, feet or legs, or signs (symmetric decrease of distal sensation or decreased or absent ankle reflexes) of DSPN.</td>
</tr>
<tr>
<td>Probable DSPN</td>
<td>The presence of symptoms or signs of DSPN, including any two of neuropathic symptoms, decreased distal sensation, or unequivocally decreased or absent ankle reflexes.</td>
</tr>
<tr>
<td>Confirmed DSPN</td>
<td>The presence of an abnormality of nerve conduction testing and a symptom / symptoms or a sign / signs of neuropathy. If nerve conduction is normal, a valid measure of small fibre neuropathy (e.g. skin biopsy or corneal confocal microscopy) may be used.</td>
</tr>
</tbody>
</table>

Adapted from Tesfaye S, et al. Diab Care 2010; 33: 2285-2293.

Table 1. Clinical classification of diabetic neuropathies

<table>
<thead>
<tr>
<th>Symmetric polyneuropathies</th>
<th>Asymmetric / focal and multifocal diabetic neuropathies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relatively fixed deficits (typical DPN)</td>
<td>• Diabetic lumbosacral radiculoplexopathy (DLSRP)</td>
</tr>
<tr>
<td>• Distal sensory polyneuropathy (DSPN)</td>
<td>• Truncal neuropathies</td>
</tr>
<tr>
<td>• Autonomic neuropathy</td>
<td>• Cranial neuropathies</td>
</tr>
<tr>
<td>Episodic symptoms (atypical DPN)</td>
<td>• Limb mononeuropathies</td>
</tr>
<tr>
<td>• Diabetic neuropathic cachexia (DNC)</td>
<td></td>
</tr>
<tr>
<td>• Hyperglycaemic neuropathy</td>
<td></td>
</tr>
<tr>
<td>• Treatment-induced diabetic neuropathy</td>
<td></td>
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</tbody>
</table>

phenomena (Table 3). Both are important in diagnostic questionnaires and rating scales and in making decisions about treatment.

A recently published study showed that the prevalence of diabetic peripheral neuropathic pain (DPNP) among people with diabetes attending institutional and private clinics in South Africa was 30%. The risk of DPNP was significantly higher in:

- black patients (odds ratio [OR] 1.71; 95% CI 1.19-2.46),
- females (OR 1.58; 95% CI 1.18-2.12),
- the 50-64 year age group (OR 1.70; 95% CI 1.21-2.41) and
- those with duration of diabetes ≥10 years (OR 1.55; 95% CI 1.15-2.08).

Table 4 lists the prevalence of different symptoms reported by South African patients with DPNP.

Table 4. Frequency of symptoms reported by South African patients with type 1 or type 2 diabetes mellitus (n=1046)*

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burning</td>
<td>36.5 %</td>
</tr>
<tr>
<td>Pins and needles</td>
<td>35.4 %</td>
</tr>
<tr>
<td>Numbness</td>
<td>31.2 %</td>
</tr>
<tr>
<td>Tingling</td>
<td>26.7 %</td>
</tr>
<tr>
<td>Hypoaesthesia to touch</td>
<td>20.5 %</td>
</tr>
<tr>
<td>Itching</td>
<td>20.4 %</td>
</tr>
<tr>
<td>Painful cold</td>
<td>19.5 %</td>
</tr>
<tr>
<td>Hypoaesthesia to prick</td>
<td>16.4 %</td>
</tr>
<tr>
<td>Electric shocks</td>
<td>12.9 %</td>
</tr>
<tr>
<td>Brushing</td>
<td>9.4 %</td>
</tr>
</tbody>
</table>

DPNP may have a significant impact on the ability to function normally and on quality of life. It affects both mental and physical functioning. Associated limitations in mobility may restrict exercise and lead to weight gain, and mood may be severely affected leading to depression and anxiety. Nighttime pain often interferes with sleep. Consequently, the ability to carry out normal daily activities, to participate socially and to maintain work may be compromised.

Painful symptoms usually persist for years, although in some patients, but not all, symptoms may become less severe with worsening sensory loss.

**Screening tools**

Validated screening tools, which assess the subjective experience of neuropathic pain, are simple to use for both clinicians and patients.

The PainDETECT questionnaire relies only on interview questions and has been validated to differentiate nociceptive from neuropathic pain in the setting of lower back pain with a sensitivity and specificity of approximately 80%.

It is important to remember, however, that shooting pain and tingling sensations are reported by approximately 50% of patients with musculoskeletal pain and burning sensations by approximately 30% of patients with non-neuropathic pain. Furthermore, screening tools may be negative in approximately 10 to 20% of patients with clinician-diagnosed neuropathic pain. Therefore diagnostic questionnaires should not replace clinical judgement.

The *Douleur Neuropathique en 4 questions* (DN4), Standardised Evaluation of Pain (stEP) and Leeds assessment of neuropathic symptoms and signs (LANSS) tools combine both interview questions and clinical assessment tests, including hypoaesthesia to pin prick and sensitivity to brushing, which are clinical tests of small fibre neuropathy. The sensitivity and specificity of DN4 to detect neuropathic pain is 83% and 90%, respectively. stEP has been validated in patients with lower back pain, in whom it was able to differentiate neuropathic radicular pain from nociceptive pain with both specificity and sensitivity greater than 90%. The sensitivity and specificity of LANSS to detect neuropathic pain was 85% and 80%, respectively.

The screening tools are available online at:


LANSS: [http://www.meduniwien.ac.at/phd-iai/fileadmin/ISMED/Literaturhinweise/Bennett_LANSS_Pain_2001_92.pdf](http://www.meduniwien.ac.at/phd-iai/fileadmin/ISMED/Literaturhinweise/Bennett_LANSS_Pain_2001_92.pdf)

**Screening of protective sensation with a 10-g nylon monofilament**

The insidious and gradual sensory loss that occurs with DSPN is a risk factor for limb fracture, plantar ulcers and neurogenic arthropathy. Therefore, to allow for early intervention, where it is necessary, intensification of glycaemic control, and increased foot surveillance with a
plan for appropriate foot care, all patients with diabetes should be screened periodically for sensory loss with a 10-g nylon monofilament. These simple diagnostic devices consist of a nylon monofilament mounted in a hand unit for protection when not in use. They are calibrated to provide a 10 g force on the skin when applied perpendicular to the skin surface so that it just starts to bend.

With the patient’s eyes closed, each of the dorsal phalanges of the foot is tested, asking the patient to confirm when a stimulus is felt. The time between test stimuli should be varied between 2 and 5 seconds to determine whether the timing of the response is appropriate. A threshold of ≤4 out of 5 correct responses or inaccurate timing of responses is considered to be predictive for DSPN and indicates further examination and investigation.

Clinical examination
A thorough examination of the peripheral nervous system and vascular examination of the lower limbs is required to exclude other causes of neuropathic or leg pain (Table 5). Co-occurrence of retinopathy and/or nephropathy further suggests that the neuropathy is related to diabetes.

Nerve conduction studies may be useful to exclude other causes of neuropathy, such as nerve entrapment syndromes.

In contrast to nociceptive pain, patients with neuropathic pain almost always have areas of abnormal sensation or hypersensitivity in the affected area (Table 3). DPNP is characteristically worse at night, which differentiates it from inflammatory pain.

In more advanced DSNP, sensory deficits in perception of mechanical or vibratory stimuli indicate damage to large diameter afferent nerve fibres. Abnormalities that may be noted on clinical examination include stocking and glove sensory loss, impaired vibration and proprioception sense of the toes, reduced or absent Achilles tendon reflexes and weakness or atrophy of the intrinsic muscles of the foot. Sensory and motor symptoms are not confined to a single dermatome or motor nerve. Significant distal weakness of the ankle is uncommon. If profound upper and lower extremity weakness is present, other causes of neuropathy should be investigated.

In early DSPN, when nerve damage is still confined to small fibres, symptoms may be present without apparent abnormalities on clinical examination. However, quantitative sensory testing (QST) to assess psychophysical thresholds for cold and warm sensations, heat and cold pain, and pain to pin prick can help to identify patients with pure small fibre neuropathy. Skin blood flow as a marker of C fibre neurovascular dysfunction may also be measured using cutaneous vasomotor function (e.g. plethysmography or laser Doppler velocimetry).

Simple bedside tests that should be performed when examining a patient with neuropathic pain are listed in Table 6. Sensation of the skin over the area of maximum pain should be evaluated and compared to the contralateral area as a control, although it should be borne in mind that sensation on both sides of the body may be abnormal in patients with DPN.

Recording symptom intensity
The intensity and severity of pain and uncomfortable sensations should be recorded at every visit. A visual analogue scale or 11-point numerical scale (0=no pain; 10=worst pain imaginable) are simple for patients to use and provide both a record of symptoms and of response to treatment.

The Neuropathic Pain Symptom Inventory (NPSI) discriminates and quantifies and the individual symptoms of neuropathic pain (spontaneous ongoing pain, spontaneous paroxysmal pain, evoked pain, and paraesthesia/dysaesthesia) that are sensitive to treatment to.

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The Brief Pain Inventory (BPI) is not specific to neuropathic pain, but includes numerical scales to evaluate and quantify pain severity and its impact on daily activities, work, sleep and quality of life. Other validated neuropathy-specific measures of quality of life include the NeuroQol and Norfolk Quality of Life Scale (NQLS). The NQLS is

<table>
<thead>
<tr>
<th>Table 5. Differential diagnosis of DSPN and DPNP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Other painful peripheral neuropathies: e.g. nerve transection pain, neurona, post-traumatic neuralgia, entrapment syndromes, ischaemic neuropathy (vascular disease), connective tissue disease</td>
</tr>
<tr>
<td>2. Generalised polyneuropathies</td>
</tr>
<tr>
<td>• Metabolic or nutritional (e.g. alcoholism, amyloidosis, hypothyroidism, beri beri, pellagra)</td>
</tr>
<tr>
<td>• Drug-related (e.g. antiretrovirals, antituberculosis treatments, metronidazole)</td>
</tr>
<tr>
<td>• Toxin-related</td>
</tr>
<tr>
<td>• Malignancy (e.g. myeloma)</td>
</tr>
<tr>
<td>• Infective or post infective, immune (e.g. HIV)</td>
</tr>
<tr>
<td>• Other polyneuropathies (e.g. idiopathic small fibre neuropathy)</td>
</tr>
</tbody>
</table>
sensitive to small fibre, large fibre and autonomic neuropathies associated with DPN.

The BPI and NPSI are available online at:

- **BPI (short form):** [http://www.painedu.org/Downloads/NIPC/Brief_Pain_Inventory.pdf](http://www.painedu.org/Downloads/NIPC/Brief_Pain_Inventory.pdf)
- **NPSI** (copyright protected review copy and validation study): [http://www.proqolid.org/instruments/neuropathic_pain_symptom_inventory_npsi](http://www.proqolid.org/instruments/neuropathic_pain_symptom_inventory_npsi)

**Laboratory tests**

If clinical examination suggests that it is necessary to exclude other causes of neuropathy, blood tests that may be considered include a full blood count, blood chemistry, liver and renal function, vitamin B12 level, folate, myeloma screen and immunofixation, antinuclear antibody (ANA), and VDRL.

**Nerve fibre assessment**

1. **Nerve conduction studies**

Nerve conduction (NC) studies provide an early and reliable objective indication of DSPN to confirm the clinical diagnosis and to define the neuropathy in a particular patient. An approach to estimating the severity of DSPN is as follows:

- **Grade 0:** no abnormality of NC.
- **Grade 1a:** abnormality of NC; e.g. Σ 5 NC normal deviates <95th percentile or another suitable NC criterion.
- **Grade 1b:** NC abnormality of grade 1a plus neurologic signs typical of DSPN, but without neuropathy symptoms.
- **Grade 2a:** NC abnormality of grade 1a with or without signs (but if present <2b) and with typical neuropathic symptoms.
- **Grade 2b:** NC abnormality of grade 1a, a moderate degree of weakness (i.e. 50 %) of ankle dorsiflexion with or without neuropathy symptoms.

2. **Skin biopsy and corneal confocal microscopy**

Formal neurological examination and other clinical tests for neuropathy, including nerve conduction studies, pressure and light touch perception with monofilaments, and vibration tests have limited utility in early DPN. These tests assess large myelinated (Aβ) nerve fibres, which account for only 10 % of peripheral nerves and are dysfunctional only in advanced DPN. In contrast, the majority of nerves are comprised of small unmyelinated C fibres and thinly myelinated Aδ fibres. In DPN, damage occurs to these nerves first, before any abnormalities in the larger fibres can

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**Table 6. Bedside tests for neuropathic pain**

<table>
<thead>
<tr>
<th>Tests for reduced perception</th>
<th>Non painful stimuli</th>
<th>Vibration</th>
<th>Painful stimuli</th>
<th>Heat or cold</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Touch skin with brush, cotton swab or gauze</td>
<td>Apply tuning fork to bone or joint</td>
<td>Single pinprick on skin</td>
<td>Objects at 10°C and at 40°C; e.g. glass of water; coolants (e.g. acetone)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Spontaneous sensations or pain</th>
<th>Non-painful ongoing sensations (e.g. paraesthesia)</th>
<th>Paroxysmal (shooting electrical shocks)</th>
<th>Painful ongoing sensations (e.g. burning)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Grade intensity and area (cm²)</td>
<td>Number per time period; grade intensity; threshold for evocation</td>
<td>Grade intensity and area (cm²)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Evoked pain</th>
<th>Allodynia</th>
<th>Hyperalgesia</th>
<th>Temporal summation</th>
<th>Mechanical deep hyperalgesia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Stroke skin with brush, cotton swab or gauze</td>
<td>Gentle mechanical pressure to skin</td>
<td>Prick skin with pin at intervals of less than 3 seconds for 30 seconds</td>
<td>Apply light manual pressure at joints or muscles</td>
</tr>
</tbody>
</table>

be demonstrated. The density of unmyelinated C fibres in the epidermis (intraepidermal nerve fibre [IENF]) is decreased early in the course of diabetes, when electrophysiology is still normal. IENF density can be quantified by examination of a skin (punch) biopsy taken from the foot and immunostained for PGP9.5.

It has been shown that measurement of small fibres in the cornea using corneal confocal microscopy (CCM) correlates with IENF density. The cornea is one of the most densely innervated tissues in the body. With this technique, it is possible to quantify small fibres in the sub basal nerve plexus between Bowman’s layer and the basal epithelial cell layer of the cornea. In a direct comparative study of IENF with CCM, IENF density, branch density and branch length, and corneal nerve fibre density and branch density all showed a progressive and significant reduction in patients with mild, moderate and severe neuropathy. In addition, patients with diabetes but not neuropathy also had significant reductions in corneal nerve-fibre branch density. Furthermore, IENF and corneal nerve fibre lengths were reduced in patients with painful compared with painless diabetic neuropathy.

Therefore, both IENF and CCM can be used to objectively and accurately quantify small nerve fibre damage in patients with diabetes. However, quantification of changes in the cornea is rapid, non-invasive and may be able to detect earlier stages of nerve damage than biopsy of IENF. Accordingly, although skin biopsy is still advocated as the ‘gold standard’, CCM has become the diagnostic modality of choice in early DPN.

**Conclusion**

The identification and quantification of the diverse symptoms and signs associated with DSPN requires meticulous history taking and careful, standardised examination procedures. The nature, severity and distribution of symptoms should be documented at every visit. Although there are currently no treatments to reverse the progression of neuropathy in diabetes, early identification of sensory deficits or discomfort can help to modify surveillance strategies and plans to prevent associated complications. Furthermore, painful and uncomfortable symptoms of DSPN can be treated, which may help to maintain or improve quality of life for these unfortunate patients.

**REFERENCES AVAILABLE ON REQUEST**
Intentional weight loss and mortality among overweight individuals with type 2 diabetes

Approximately two-thirds of people with type 2 diabetes are overweight. 1 Weight reduction is regarded by many as a cornerstone of diabetes therapy because it improves glycaemia, lipoproteinemia and blood pressure and reduces medication costs. 3

Despite the physiological benefits of weight loss in patients with diabetes, the evidence that weight loss increases their longevity is equivocal. 2 In addition, no studies have failed to differentiate between intentional and unintentional weight loss. 1 This is important because unintentional weight loss may be associated with more severe disease or unrecognised health problems. 1

Williamson et al conducted the first study using data on weight loss intention to examine the relationship between weight change and mortality in overweight individuals with diabetes. 3 This prospective study with a 12-year mortality follow-up of 4,970 overweight individuals with type 2 diabetes compared overall death rates and death from cardiovascular disease or diabetes in individuals with or without intentional weight loss. 3

Intentional weight loss was reported by 34 % of the subjects. 1 In fully adjusted models, intentional weight loss was associated with −25 % reduction in mortality (RR = 0.75; 95 % CI 0.67-0.84), and a 28 % reduction in cardiovascular disease and diabetes mortality (RR = 0.72; 0.63-0.82). 1

The largest protective effect of intentional weight loss for total mortality was observed at a loss of 9.1-13.2 kg or 10-15 % of initial weight (RR = 0.67; 0.58-0.77). 1

These mortality reductions parallel the physiological improvements observed after weight reduction. 1 In contrast, losses of ≥ 31.8 kg or ≥ 30 % of initial weight were associated with small increases in mortality (Fig 1). 1 The majority of subjects with intentional weight loss reported fast weight loss (< 1 year), in sharp contrast to subjects who lost weight unintentionally (≥ 1 year). 1

A conservative estimate of the average weight loss patients can expect to achieve is 1.19 kg per week in the first 4 weeks and 0.56 kg per week in weeks 5-12. 4

Studies prove that faster initial weight loss is associated with a greater likelihood of achieving a 10 % weight loss, 5 which in turn translates into a 33 % reduction in mortality. 1

*plus 1000-calorie diet. Note: patients require medical review after a defined course of treatment, which ideally should not exceed 3 months.

Intentional weight loss in patients with type 2 diabetes may be easier said than done. It has been demonstrated clinically that people with type 2 diabetes find it more difficult to lose weight than overweight people without diabetes. 2 This may be due to the altered metabolism of diabetes rather than non-adherence. 2

Duromine, South Africa’s No. 1 prescribed weight loss medication, 3 helps patients achieve fast and significant weight loss. 4

Dosage and directions for use

15 mg or 30 mg capsule once daily at approximately 7 a.m. **

** Duromine is not indicated in children under 12 years old.

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Scheduling status: [a] Proprietary name (and dosage form): Duromine™ 15 mg and 30 mg Capsules. Compositions: Sustained action ion-exchange resinate granules, available as capsules containing fenfluramine 15 mg and 30 mg. Pharmacological classification: A 1.1.3 Aminorexics. Reference number: 15 mg 86672; 30 mg 86548 [Art 10517962]. Name and business address of applicant: Nova Pharmaceuticals (Pty) Ltd, Cape Town, 7100. For full prescribing information, refer to the package insert approved by the medicines regulatory authority. Further information is available on request from Nova Pharmaceuticals, IN465/13.
‘Pre-diabetes’ - the risk it poses to South Africa and the role of lifestyle modification

Hamish van Wyk
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Introduction
The focus of this article will be to evaluate the risk of developing diabetes with intermediate hyperglycaemia (I have used the term ‘pre-diabetes’) as well as the effectiveness of lifestyle interventions (LSI) in the prevention or delay of the conversion to diabetes. Lastly, attention will be given to possible solutions on how to manage pre-diabetes in South Africa.

The International Diabetes Federation lists five successful major diabetes prevention studies using lifestyle modification in patients with pre-diabetes (Table 1, International Diabetes Federation, 2007). However, the clinical implications of pre-diabetes are still heavily debated, especially the proposed American Diabetes Association (2014) criteria for impaired fasting glucose (IFG) (Yudkin & Montori, 2014).

Defining ‘pre-diabetes’
Currently, the Society of Endocrinology Metabolism and Diabetes of South Africa (SEMDSA, 2012) recognises two forms of pre-diabetes, IFG and impaired glucose tolerance (IGT). IFG is defined as a fasting blood glucose (FBG) between 6.1 and 6.9 mmol/l and IGT as a 2 hour post prandial blood glucose (2hPPBG) of between 7.8 and 11.0 mmol/l following a 75 g oral glucose tolerance test (OGTT).

Reproducibility of glycaemic tests for pre-diabetes
A challenge when assessing the relative risk for diabetes in patients with pre-diabetes lies in the reproducibility of a single glycaemic test. Data from an Agency for Healthcare Research and Quality Evidence Report (2005) showed that of patients across a number of studies who met the criteria for IGT with a first OGTT, only between 33 and 48 % met the criteria for IGT on a second test 6 weeks later. 39-46 % were reclassified as having normal glucose tolerance and 6-13 % met criteria for diagnosis of diabetes. In the US Diabetes Prevention Program (DPP) (1999) the inclusion criterion was based on a single OGTT. Could the incidence for developing diabetes have been higher if patients were...
only included following a confirmatory test? In the Indian Diabetes Prevention Program (IDDP), the inclusion criterion was two positive diagnoses for IGT (Ramachandran et al., 2006).

**Diagnostic criteria used in studies**
The DPP and IDDP defined diabetes in accordance with the current SEMDSA (2012) criteria, whereas the other three studies, seen in Table 1, defined diabetes according to the World Health Organization (1985) criteria of a FBG of ≥7.8 mmol/l or 2hPPBG of ≥11.1 mmol/l. Whether use of these alternative diagnostic criteria would affect the interpretation of the risk for diabetes is unclear, and thus this article will focus on the DPP and IDPP.

**Cumulative incidence of diabetes**
As seen in Table 2, the cumulative incidence varies substantially. One might be tempted to define the cumulative incidence for developing diabetes based on that of the control group. However, a pitfall of such an approach may be that the control groups in each of the five studies received lifestyle education, although less intense than the LSI cohort. The DPP control group received a similarly intense LSI compared to that given in the original LSI following the completion of the 3-year study. Thus, each group in the DPP received a LSI at some point over the 10 years (DPP Research Group, 2009). The former may result in cumulative incidence rates being lower than those in untreated patients with pre-diabetes were.

**Relative risk reduction through lifestyle modification**
Table 1 provides the relative risk reduction for each of five major studies. ‘Diet’ only and physical activity only were equally effective in reducing the risk for diabetes in the Da Qing study (Pan et al. 1997). The combination of diet and physical activity also did not offer a significantly greater effect than the former or the latter in isolation (Pan et al. 1999). However, subjects within the Da Qing study were generally less overweight (mean BMI of ±25.5 kg/m²) compared to subjects from the DPP (mean BMI of 34 kg/m²) and may therefore not have required as aggressive interventions in an attempt to lose large amounts of weight. Results from a diabetes prevention study by Mensink et al (2003) indicated that those who ‘complied’ only with the dietary management lost 0.9 kg. Those who only complied with the ≥150 minutes of physical activity lost 0.1 kg, whereas compliance with a

<table>
<thead>
<tr>
<th>Table 1: Diabetes prevention studies using lifestyle modification in patients with pre-diabetes</th>
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<tbody>
<tr>
<td>Relative risk reductions for diabetes compared to a control</td>
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<td></td>
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<tr>
<td></td>
</tr>
<tr>
<td>Da Qing (Pan et al. 1997; Li et al. 2008)</td>
</tr>
<tr>
<td>Diet</td>
</tr>
<tr>
<td>Exercise</td>
</tr>
<tr>
<td>Diet + exercise</td>
</tr>
<tr>
<td>Diabetes Prevention Program (DPP) (Knowler et al. 2002; DPP Research Group 2009)</td>
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<tr>
<td>LSI</td>
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<tr>
<td>Metformin</td>
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<tr>
<td>The Finish Diabetes Prevention Study (DPS) (Tuomilehto et al. 2001; Lindström et al. 2013)</td>
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<tr>
<td>LSI</td>
</tr>
<tr>
<td>Indian Diabetes Prevention Program (IDDP) (Ramachandran et al. 2006)</td>
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<tr>
<td>LSI</td>
</tr>
<tr>
<td>Metformin</td>
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<tr>
<td>LSI-Metformin</td>
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<tr>
<td>Kosaka, Noda, &amp; Kuzuya (2005)</td>
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<tr>
<td>LSI</td>
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combination of both the dietary management and physical activity resulted in a weight loss of 6.2 kg (p<0.01). As nutritional and physical activity interventions in combination may result in greater weight loss, it is reasonable to suggest both, especially in obese subjects with IGT, where weight loss is the most significant factor in preventing diabetes.

In assessing the risk reductions in Table 1, note that the mean risk reduction of an intervention is reflected and not the risk reduction of lifestyle modification per se - not everyone in either the control group, or the LSI group for that matter, complied with the lifestyle modification. Therefore, assessing the risk reduction in those who complied with the lifestyle advice may offer a better understanding of the effects of lifestyle modification. The former is conveyed by Hamman et al. (2006). Subjects in the DPP who lost 13.4 kg (≥7 % weight loss), engaged in ≥150 minutes of moderate intensity physical activity per week and whose fat intake was ≤25 % of total energy intake (TEI) had a 96 % relative risk reduction for diabetes over 3 years. Comparing subjects based on their ability to reach the five lifestyle goals set by DPS revealed a relative risk of 80 % compared to those who achieved none of the goals at 13 years (Lindström et al, 2013). Therefore, the relative risk reduction for diabetes through LSI’s is as much 96 % at 3 years and 80 % at 13 years with good adherence.

**Number needed to treat**

In the DPP, the number needed to treat (NNT) to prevent one case of diabetes was 6.9 in the lifestyle intervention group over 3 years. In the IDDP, the number needed to treat was 6.4 over 3 years. It appears that to treat subjects at an even higher risk (i.e. multiple risk factors) for diabetes lowers the NNT. Subjects from the DPS with a Finnish Diabetes Risk Score (FINDRISC) of ≥15 required only 3.7 subjects to be treated for 4 years compared to the usual 7.7. Furthermore, a FINDRISC score of <15 required a NNT of 24.8 over 4 years (Lindström et al. 2008).

### Table 2: Cumulative incidence of diabetes in five Diabetes prevention studies

<table>
<thead>
<tr>
<th>Study</th>
<th>3 year</th>
<th>4 year</th>
<th>6 year</th>
<th>10 year</th>
<th>13 year</th>
<th>20 year</th>
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</thead>
<tbody>
<tr>
<td><strong>Da Qing</strong>      (Pan et al. 1997; Li et al. 2008)</td>
<td></td>
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<td></td>
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<tr>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>93 %</td>
</tr>
<tr>
<td>Diet</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>43.8 %</td>
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<tr>
<td>Exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>41.1 %</td>
<td>80 %</td>
</tr>
<tr>
<td>Diet +exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>46.0 %</td>
</tr>
<tr>
<td><strong>DPP</strong> (Knowler et al. 2002; DPP Research Group 2009)</td>
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<tr>
<td>Control</td>
<td>28.9 %</td>
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<td></td>
<td></td>
<td>50 %</td>
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<tr>
<td>LSI</td>
<td>14.4 %</td>
<td></td>
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<td></td>
<td>40 %</td>
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<tr>
<td>Metformin</td>
<td>21.7 %</td>
<td></td>
<td></td>
<td></td>
<td>45 %</td>
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<tr>
<td><strong>DPS</strong> (Tuomilehto et al. 2001; Lindström et al. 2013)</td>
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<tr>
<td>Control</td>
<td>23 %</td>
<td></td>
<td></td>
<td></td>
<td>64 %</td>
<td></td>
</tr>
<tr>
<td>LSI</td>
<td>11 %</td>
<td></td>
<td></td>
<td></td>
<td>44 %</td>
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<tr>
<td><strong>Indian DPP</strong> (Ramachandran et al. 2006)</td>
<td></td>
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<tr>
<td>Control</td>
<td>55 %</td>
<td></td>
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<tr>
<td>LSI</td>
<td>39.3 %</td>
<td></td>
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<tr>
<td>Metformin</td>
<td>40.5 %</td>
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<td></td>
<td></td>
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<tr>
<td>LSI-Metformin</td>
<td>39.5 %</td>
<td></td>
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<tr>
<td><strong>Kosaka, Noda, &amp; Kuzuya</strong> (2005)</td>
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<td></td>
</tr>
<tr>
<td>Control</td>
<td>9.3 %</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>LSI</td>
<td>3 %</td>
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</tbody>
</table>
Legacy effect
The difference in physical activity was only statistically greater in the LSI relative to the control group until 3 years from baseline (p<0.001, DPP, 2012). The difference in weight was roughly 2 kg between the LSI and the control (93.2 vs. 91.8 kg), from year 4 until year 10 (DPP, 2012). Unfortunately, the difference in dietary intake between the control and LSI was limited to the first year. Despite this near converging of physical activity and weight between the two groups, the LSI group maintained a relative risk reduction of 34%. The former may suggest that early LSI may offer long-term protection against diabetes even if not maintained.

Delivering of diabetes
As seen in Table 2, approximately 40% of the patients from the LSI group developed diabetes over 10 years in the DPP. This may be rather disappointing, but the time to diagnosis may offer some perspective. The time to a diagnosis of diabetes in the DPP was delayed by 4 years relative to the control group over a 10 year period (DPP Research Group, 2009). Thus, even if diabetes is not prevented, the time to diagnosis may well be delayed.

Rates for regression to normoglycaemia
At the end of 3 years, 29.6 %, 19.7 % and 17.2 % for the DPP LSI, metformin and control groups respectively achieved normoglycaemia (Knowler et al. 2002). LSI was the most effective in restoring normal 2hPPBG. According to Snehalatha et al. (2009) 24.1 %, 35.8 %, 30.5 % and 31.4 % of subjects in the control, LSI, metformin and LSI-metformin groups returned to normoglycaemia by 2.5 years in the IDDP.

The impact of regression to normoglycaemia
Patients who achieved normoglycaemia at the end of the 3 years of the DPP were 56 % less likely to develop diabetes over the additional 7 year follow up period (Perreault et al. 2012). Remarkably, the impact of to which group the patient was previously assigned did not influence the results. This was reiterated at 10 years, where subjects from the LSI group who remained with pre-diabetes during the 3 year DPP had a 31 % greater risk of developing diabetes than control subjects who had achieved normoglycaemia (Perreault et al. 2012).

Weight loss and diabetes incidence
The link between weight loss and diabetes incidence varied between the major studies. In the IDDP and Da Qing, weight loss did not predict the risk reduction for diabetes (Pan et al., 1999; Ramachandran et al., 2006). In the DPP (Hamman et al. 2006) and DPS (Lindström et al. 2006) weight loss was the most important factor for preventing diabetes. The impact of weight loss on future diabetes incidence was dose dependant. This was noted by the relative risk reduction for diabetes of 16 % and 58 % for every 1 and 5 kg of weight loss over 3 years respectively (Hamman et al. 2006). Secondly, in those who lost ±15 kg, the incidence of diabetes was 1 per 100-person years, where those who gained +1.5 kg had a diabetes incidence of 16 per 100-person years (Hamman et al. 2006). Lindstrom et al. (2005) highlights the importance of weight loss in the DPS in that those patients who achieved a 10.4 % weight loss had a relative risk reduction of 71 % when compared to those who lost no weight. A smaller weight loss of 4.6 % conveyed a relative risk reduction of 50 % compared to those who did not lose weight. Even subjects who failed to lose weight, but were able to maintain their existing weight had a 1.5 times lower risk for diabetes compared to those who gained 3.3 % bodyweight (Lindstrom et al. 2005).

Basic analysis of the dietary management
It is beyond the scope of this article to report on the intricate details of the dietary management used within the five major diabetes prevention studies. In general, the macronutrient goals were 10-15 % of TEI from protein, >50 % of TEI from carbohydrates and the remaining <25-30 % from fat. Interestingly, when looking at what the patients were able to achieve, the actual macronutrient distribution was somewhat different from that suggested in the protocol. The only long-term dietary intake data for all macronutrients comes from the DPS. At the 13-year follow up, the LSI group consumed 33 % of TEI from fat, 47 % of TEI from carbohydrates (186 g), 18.8 % of TEI from protein and 2 % of TEI from alcohol. Such data may provide some understanding as to what macronutrient range is sustainable long term.

Basic analysis of the physical activity
As with the dietary analysis, it is beyond the scope of this article to discuss physical activity in detail. However, it is interesting to note that in the DPS, total physical activity and walking for exercise was no different between groups. Rather there was a redistribution of the intensity and form of physical activity. Moderate-vigorous activity (p=0.0028) and strenuous structured activity (other than walking) (e.g. circuit training) (p<0.001) increased more so in the LSI group, while exercising at lower intensities decreased in both groups (Laaksonen et al. 2005).

Pre-diabetes and South Africa
IGT affects approximately 2 653 940 people in South Africa (SA) (8.3 % of the population between the ages 20-79) (IDF, 2013). The World Bank (2011) reported that 8.5 % of the total gross domestic profit of South Africa is spent on health, significantly more than the 5 % recommended by the WHO (2003). The mean expenditure per patient per annum for diabetes in South Africa for
2013 was US$935 (IDF, 2013), roughly 30% more than the previous year (IDF, 2012). An estimated 2,646,100 people have diabetes, and another 1,272,000 are unaware that they have diabetes (IDF, 2013). Thus, if all patients with diabetes were treated, it would cost an estimated $3,663,423,500 (± R$40,064,659,268) per annum, 30% of budget spent on health in the state sector for 2013/2014 (World Vision South Africa 2013). The number of patients with diabetes is said to increase to 3,860,400 by 2035 (IDF, 2013). Thus, we sorely need prevention efforts to reduce the cost of treating diabetes on an already strained healthcare system.

Possible solutions
The big question is how to prevent diabetes? Should every overweight or obese patient undergo vigorous lifestyle education? Possibly, but it is unlikely that the already strained health system would be able to pay for this. It would seem logical to focus efforts rather on patients at a high risk for diabetes, e.g., pre-diabetes. It is clear that IFG increases the risk for developing diabetes, but there is little evidence to suggest that LSI will prevent diabetes in these patients. Efforts should be directed at patients with IGT, for which there is evidence of the effectiveness of LSI. One could focus efforts towards targeting patients at even a higher risk for developing diabetes using the FINDDRISC score. This would reduce the amount of patients that would need to be treated, thus lowering the cost. However, even in this subgroup it is unlikely that there are enough dietitians to educate all of these patients, nor is there likely to be enough money to pay for the dietitians required. An approach, which might help resolve the issue in part, is group consultations. The Healthy Living Partnership Study to Prevent Diabetes (HELP PD) was a group-based version of the original DPP over 2 years. Weight loss and reduction in FBG were comparable to that of the original DPP (Katula et al. 2013). The direct medical costs were $850 per patient compared to $2,631 in the original DPP over 2 years (Lawlor et al. 2013). The direct medical costs of the DPP over 10 years was $4,572 per patient, and would have been $2,995 if completed in groups of 10 (DPP, 2012). Quality-adjusted life years (QALY’s) are often used to measure the cost effectiveness of a treatment (Li et al. 2010). This simply measures the costs for every gain in quality of life. Li et al. (2010) mentions that according to the literature, very cost-effective is < $25,000 per QALY, cost-effective $25,001 to $50,000 per QALY, marginally cost-effective $50,001 - $100,000 per QALY and not cost effective > $100,000 per QALY. Over 10 years, the QALY was $12,878 in the LSI relative to the control and thus very cost effective. Interesting, if the education was conducted in a group of 10, the QALY would have only been $1,478 (DPP, 2012).

A second lesson learnt from the HELP PD study was the use of community health workers. The community health workers were responsible for seeing the patients in groups with the aid of standardized educational DVDs. Dietitians acted as trainers of the community healthcare workers, programme managers, and offered 3 individual education sessions over the 2 years, taking on the role of a nutrition consultant. Such an approach would provide career advancement for dietitians whilst empowering the currently available community healthcare workers. The overall cost of such an intervention in South Africa would be far less, as the salaries required to pay community healthcare workers are substantially lower. Secondly, community healthcare workers are available, whereas to train the equivalent in the form of dietitian’s takes time, which South Africa may not have. Roughly 200 dietitians graduate each year in South Africa, which is simply not going to be enough to tackle the explosion of pre-diabetes and diabetes.

Is education, support and motivation going to be enough? It is most unlikely. The price and availability of food influences peoples food choices, possibly even more so in low-income households. Buying low-cost refined carbohydrate foods such as mielie meal will feed a family, whereas the same of amount of money will only serve to decorate the plates with a few vegetables. Would it not be difficult for you, the reader, to maintain a healthy weight, if all you could afford to eat was low-cost refined carbohydrates, which have a poor ability to ensure satiety at smaller ‘normal’ portion sizes? To buy and eat healthy food is a case of swimming upstream. One only needs to walk into the many fast food take-outs or your local petrol station to realize that trying to find a healthy meal is often a game of hide-and-go-seek. Could taxing unhealthy food such as sugar containing beverages and using that money to make healthy food cheaper be part of the solution, as proposed by Manyema et al. (2014)? Part of the solution could very well be to make it easier to eat healthy foods than it is to eat unhealthy food. This is one of the key messages of the 2014 World Diabetes Day, ‘Healthy Living and Diabetes’, ‘Off to the right start’ healthy breakfast Campaign.

‘Diabetes is an all-too-personal time bomb which can go off today, tomorrow, next year, or 10 years from now - a time bomb affecting millions like me and the children here today’ Mary Tyler Moore

If type 2 diabetes is like a time bomb, then pre-diabetes is most likely the sound of the bomb counting down. Will South Africa, be able to defuse this bomb in time?

REFERENCES AVAILABLE ON REQUEST
Carbohydrates, oxidative stress and low carbohydrate living

In 1963, Dr George Campbell wrote an article, published in the South African Medical Journal, in which he described his experiences working among the Indian and Zulu populations of Natal. He noted an increased incidence in the development of diabetes amongst these populations, which correlated with an increase in their sugar consumption. He, and other researchers around the world, came to recognize that it took about 20 years of a high sugar load of between 36-38 kg/year in a sugar naïve patient to develop diabetes (i.e. about 21 teaspoons of sugar per day). He also recognized that patients could control their diabetes by limiting their intake of carbohydrates (see his recommendations in Figure 1) as an adjuvant to biguanide therapy, although many patients were not compliant with the recommendations.

While some of these recommendations are probably still appropriate for contemporary diabetes clinics, a greater emphasis has been placed on low-fat approaches at the expense of a reduction in carbohydrate consumption, as detailed in recent publications by Gary Taubes and more recently in “The Big Fat Surprise” by Nina Teicholz. One of the plausible pathways through which hyperglycaemia manifests its harm is through the generation of reactive oxidative species. This article offers insights into how limiting carbohydrate consumption might minimise or prevent vascular disease associated with diabetes.

Figure 1: Dietary recommendations at The King Edward VIII Hospital Diabetic Clinic in 1963.

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Dr Neville Wellington
CDE Branch: Kenilworth Medical and Diabetes Centre, Cape Town
**Glucose and insulin**

All carbohydrates are digested in the gastro-intestinal tract to their component monosaccharides, the most common of these being glucose, fructose and galactose. After a carbohydrate-rich meal, glucose levels rise in the bloodstream. The extent of this rise is determined by the amount of carbohydrate consumed. The rise in glucose levels in turn triggers the release of insulin.

Practically, patients can measure these glucose changes post-prandially to assess how carbohydrate ingestion influences their blood glucose levels.

**How insulin can make us fat**

From experience, we have seen patients who gain weight while using insulin or insulin secretagogues. As mentioned, glucose from carbohydrates is the main reason why insulin levels rise. Insulin works on three cell types, the muscle (storing glycogen), and the liver and fat cells through glucose transporter 4 (GLUT4). In fat cells, it not only stores fat, but also is anti-lipolytic. Our physiology attempts to maintain glucose levels at a narrow range of 3.7-6.0 mmol/l pre-prandially and less than 7.8 mmol/l post-prandially. Insulin is the main driver of keeping glucose levels in this range, because levels beyond this are damaging to our cells.

**How hyperglycaemia causes damage**

Evidence from many studies shows that people with diabetes experience mortality rates 2 to 4 times higher than those who do not have diabetes do.

Other studies have shown that even patients who have levels of glycated haemoglobin (HbA1c) lower than that accepted for a diagnosis of diabetes (i.e. <6.5 %) have an increased risk of cardiovascular disease (CVD), and worsening coronary artery disease.

At a cellular level, hyperglycaemia has been shown to induce four major pathways through which excess substrates of glucose metabolism will flux viz. the polyol pathway, the hexosamine pathway, the protein kinase C pathway and the advanced glycation end products (AGEs) pathway. These pathways induce the production of pro-inflammatory cytokines such as tumour necrosis factor-α (TNF-α) and C-reactive protein (CRP) amongst others. Ultimately, these processes culminate in reduced nitric oxide (NO) formation. This occurs predominantly at an endothelial level.

In the arteries, the inflammatory milieu of hyperglycaemia causes changes to the endothelial cells, which promote coagulation and thrombosis in the arteries. Platelets are activated, NO is depleted and angiotensin II increases which increases vasoconstriction. The first step in the formation of atherosclerotic plaques is the uptake of oxidised low-density lipoprotein (LDL)-cholesterol simultaneously with the adhesion and uptake of monocytes. In the sub endothelial space, monocytes change to macrophages that then ingest oxidised LDL-cholesterol and eventually form foam cells. Cytokines are released which promote the translocation of smooth muscle cells to the area and further increase growth of the plaque. Stable plaques are usually formed, but ongoing inflammation can destabilise them. Macrophages release matrix metalloproteinases, which appear to thin the fibrous cap and make it susceptible to rupture. This helps to explain the proliferation of plaques that occur in diabetes and the propensity to peripheral vascular disease, coronary artery disease, cerebral artery disease and other vascular disease processes.

‘Spikes’ or excursions of glucose that last for more than 16 hours to levels >8.6 mmol/l have been shown to cause epigenetic changes in the promoter of NF-κβ subunit p65 in the aortic endothelial cells. Hyperglycaemia induced reactive oxygen species (ROS) formation in the mitochondria causes these effects. Increased levels of NF-κβ p65 cause increased levels of MCP-1, which recruits plasma monocytes in the early stages of atherosclerosis, and VCAM-1, which promotes monocyte adhesion to the arterial endothelial cells. These changes can persist for 6 days after normalisation of glucose levels - this highlights the prolonged effects even relatively short-term hyperglycaemic excursions can have on cells. **Editor:**

The DECODE Study demonstrated the significance of postprandial glycaemic excursions in terms of adverse cardiovascular outcomes.

**The Problem of Fructose**

It is seldom that you find glucose without fructose in the modern lifestyle, whether in the ubiquitous high fructose corn syrup added to many commercial foods and drinks, or in the over consumption of fruit juices and even fruits...
Essentially, it is a low carbohydrate, high fat (LCHF) diet based around the premise that carbohydrates drive the problem of hyperglycaemia, diabetes, hypertension, the metabolic syndrome, CVD, and many other associated diseases.

The Metabolic Syndrome
The Metabolic Syndrome describes a syndrome made up of a cluster of cardiovascular risk factors, which are present in a single individual. Gerald Reaven coined the term ‘Syndrome X’ in the 1988 Banting lecture where he described insulin resistance in response to glucose uptake, and its associated abnormalities and clinical phenomena. Since then, various attempts have been made to define what has subsequently become known as the metabolic syndrome. The three most notable attempts to define the syndrome are those by the World Health Organisation (WHO) in 1999, the National Cholesterol Education Program - Third Adult Treatment Panel (NCEP ATP III) in 2003 and by the International Diabetes Federation (IDF) in 2005. The IDF definition is still widely used.

Clinicians typically see these characteristics in diabetes clinics and they are common changes we see in our overweight population of patients. As has been noted by some authors, obesity and the metabolic syndrome is a response to a diet high in carbohydrates, and to hyperinsulinaemia.

What is a low carbohydrate lifestyle?
• A lifestyle low enough in carbohydrates, not to cause or to reverse these problems.

Essentially, it is a low carbohydrate, high fat (LCHF) diet based around the premise that carbohydrates drive the problem of hyperglycaemia, diabetes, hypertension, the metabolic syndrome, CVD, and many other associated diseases. For me, the overriding theme has been the improvements in CVD markers in people who have followed these lifestyle changes. Even before I started advocating this lifestyle, a number of my patients had been hearing about the lifestyle and had themselves started following it. Many had shown some quite dramatic changes.

a ~42:55 ratio. Compared with glucose, the body handles fructose quite differently. It is taken up by GLUT-5 in the intestine and metabolized largely (50-75 %) in the liver, while the rest is metabolized by the kidneys and adipocytes. Then it is absorbed by cells, again via GLUT 5 and 2, and metabolised by fructokinase (also known as ketohexokinase (KHK)). Two isoforms exist, but the dominant KHK-C isoform is the main enzyme involved in metabolism of fructose. It is mainly found in the
• Liver
• Intestinal cells
• Proximal tubules of the kidneys
• Adipocytes
• Possibly the endothelium.

Fructose has the following properties that differ from those of glucose:
• Fructose uses different transporters - GLUT 2 and 5 vs. GLUT 1 and 4.
• It is metabolised by different enzymes viz. fructokinase vs. glucokinase and phosphofructokinase.
• Regulation of fructose metabolism is poor. Fructokinase phosphorylates all fructose rapidly causing depletion in adenosine triphosphate (ATP). This has been shown in liver cells, endothelial cells and human proximal tubular cells. This ATP depletion can temporarily reduce protein synthesis and increase inflammatory markers, endothelial dysfunction and oxidative stress.
• Fructose is highly lipogenic, stimulating the production of triglycerides (TG’s), by increasing fatty acyl coenzyme A and diacylglycerol (DAG). It therefore causes a greater production of TG’s than the same amount of glucose, and generates higher levels of apolipoprotein B. Fructose feeding causes fatty liver, sometimes used by animals in hibernation.
• Fructose metabolism rapidly causes an increase in uric acid, because as ATP is consumed, adenosine monophosphate (AMP) increases, which is then metabolised by AMP deaminase to uric acid.
• Fructose has a positive feedback loop that up-regulates GLUT 5. Thus, people who have been on a high fructose diet will show enhanced production of uric acid for a standard load of fructose vs. those on a regular diet.
• Fructose does not signal insulin release.

Ultimately, the lipogenic characteristics, the ATP depletion effects and the increase in uric acid seem to be the precursor for the metabolic syndrome that we see.
which really piqued my interest. One patient, in the course of 3 months, dropped his TG’s from 33 to 1, lost 17 kg and dropped his HbA1c from 10.4-7.7 % while following an Atkins type diet. We are looking for reversal of many of the features of the metabolic syndrome. This can be attained through lifestyle changes that result in reductions in weight, TG’s, an increase in HDL-cholesterol, falls in blood pressure (and reduction in medication), and improvements in glucose control (with a reduction in medication use). For me, these positive outcomes have been sufficient to amend my practice.

Low carbohydrate diets have been defined as follows:

- **Very Low-Carbohydrate Ketogenic diet (VLCKD)**
  - 20-50 g/day or <10 % of a 2000 kCal diet. Usually the level of carbohydrates that may induce ketosis in most people (>0.5 mmol/l of ketones).

- **Low Carbohydrate Diet**
  - <130 g/day or less than 26 % of total energy. The American Diabetes Association accepts this as the lower limit.

- **Moderate Carbohydrate Diet**
  - 130-225 g/day or 26-45 % of a 2000 kCal diet. This was the usual upper limit of carbohydrates before the obesity epidemic.

- **High Carbohydrate Diet**
  - >225 g/day or >45 % of total calorie consumption.

### Clinical trials

Despite numerous assertions that few studies show the effectiveness of low carbohydrate diets, at least 24 studies show benefits compared to low fat studies. Typically, these studies reflect the heterogenous nature of the populations investigated, and have spanned periods varying from a few weeks to 2 years. Three of the studies included patients with type 2 diabetes, but only one 24-week study by Westman et al, showed significant changes in weight loss, HbA1c and reductions in medication usage. One lasted for 3 months, and only showed a big difference in weight loss, while the other, lasted two years, but only showed glycaemic improvements in the first 6 months. The problem with this last trial was that the adherence to both diets worsened as the trial continued, and the low carb group were allowed to increase their carb intake. Unfortunately, very few trials actually achieved sustained levels of carbohydrate restriction below the lower limit of a ‘moderate’ carb intake (130 g/day), although most were able to lower carbohydrate consumption below previous levels.

Overall, however, all the trials showed improvements in **weight loss** and in **lipid profiles** (in those trials that reported these). LDL-cholesterol levels did not worsen in these trials, although I have seen this occur in some of the patients I have treated. HDL-cholesterol levels improved, and so did TG’s.

A number of reviews have shown improvements in patients with diabetes and some do recommend the use of low carb lifestyles in improving diabetes control. There are no long-term low carbohydrate studies showing hard cardiovascular beneficial endpoints; but neither is there any long-term studies showing cardiovascular benefits from low fat diets. At this stage, the studies only demonstrate reductions in markers related to cardiovascular disease.

A recent study by Maekawa et al also showed the effectiveness of low carb lifestyles in preventing patients with impaired fasting glucose from developing diabetes over a 12-month period (0 % vs. 13.9 %).

This brings us to the practical aspect of recommending low carbohydrate lifestyles for patients who are obese, those with metabolic syndrome and people with diabetes.

### So what do I tell patients?

Firstly, I describe what carbohydrates, proteins and fats are and how the body uses these. I explain how insulin works and how it is primarily increased by carbohydrate consumption, but also causes fat production. I then go through a list of common carbohydrates.

### Commonly consumed carbohydrates and their approximate values (g)

- 1 tsp sugar = 4-5 g of carbohydrates
- 1 slice bread = 15 g
- 1 plate cereal (e.g. 2 Weetbix biscuits) = 23-30 g
- 250 ml (1 cup) Coca cola = 28 g
- 250 ml fruit juice = 32 g
- 100 g spaghetti = 70 g
- 100 g potato/sweet potato = 20 g
- 1 cup cooked rice = 20 g
- 1 medium apple = 20 g
- 1 large banana = 30 g (note this is equivalent to about 2 slices of bread)
- 100 ml sweetened yoghurt = 15 g
- 250 ml sweetened yoghurt = 42.5 g
- 100 g slab chocolate = 60 g

Unfortunately, very few trials actually achieved sustained levels of carbohydrate restriction below the lower limit of a ‘moderate’ carb intake (130 g/day), although most were able to lower carbohydrate consumption below previous levels.
I explain where they can get resources. More lists of carbohydrates can be found at www.carbohydrate-counter.org, and on all nutrition labels (see examples in Figure 2 below).

For those with diabetes and those trying to lose weight, I advocate that they eat no more than 50 g of carbohydrate per day, and for some, even less. Each person needs to find his or her optimum level. For patients with diabetes, it is critical to check their glucose levels regularly before and 2 hours after meals. These results should help to guide medication amendments such as altering future insulin doses. This will need to be done in conjunction with their doctor or diabetes educator. For dietary advice and help with planning meals, I recommended that they consult with a registered dietician who is familiar with low carbohydrate lifestyles.

What should patients avoid and what can they eat?
As suggested above, avoid all sugars, sweets, confectionaries, breads, cereals and certain low fat foods. Some low fat foods (e.g. low fat yoghurts) have sugar added to make them taste better. Avoid all processed foods, which generally means foods that come in a box. While whole foods like fruit and vegetables contain micronutrients that your body can use, it is advisable to reduce their intake or avoid them if it their consumption will exceed the personal daily target of carbohydrate intake.

Foods to eat include the following:
- Eggs
- Full fat meats, although there are controversies in some studies about the association between red meat and increasing incidence of CVD development
- Chicken with the skin
- Fish
- Leafy green vegetables
- Avocados
- Olive oil
- Coconut and coconut oil
- Fatty nuts like almonds and macadamia nuts
- Cheese (unprocessed)
- Full cream milk
- Cream
- Butter
- Mushrooms
- Berries
- Small amounts of rice and potatoes (in diabetes, check glucose)

Again, the dietician can help with these choices.

Conclusion
All carbohydrates are digested to glucose and fructose. High glucose levels in cells cause oxidative stress, damage, and are the precursor to atherosclerosis in endothelial cells. In those with impaired glucose tolerance or diabetes, excess glucose and fructose are metabolised to fat although the main culprit in this is really fructose. High consumption of both may eventually lead to obesity and insulin resistance, and I contend that high insulin levels are also damaging to the arteries and other organs. Numerous clinical trials and reviews confirm the safety of low carbohydrate lifestyles and recommend their use in patients with diabetes, obesity and metabolic syndrome. Practitioners need to familiarise themselves with these lifestyles so as to better inform their patients of the options that low carbohydrate lifestyles may afford them.

REFERENCES AVAILABLE ON REQUEST
The Role of Snacking in Diabetes

For people with diabetes, healthy eating is not merely a case of what one eats, but also when one eats.

Historically, earlier oral hypoglycaemic agent and insulin regimens often dictated rigid nutritional prescriptions including a need for regular snacking. This was to counter a higher risk of iatrogenic hypoglycaemia due to the prolonged modes of action of therapy. Unfortunately, this historical insight is largely forgotten and snacking for people with diabetes is still routinely recommended without first assessing the reason or need.

With current therapies, the need for snacking should be individualised by taking into consideration treatment options, glycaemic control and patient preference.

Eating frequently is presumed to improve hunger and thus reduce energy intake, but the effect of meal frequency on human health and longevity are still unclear in current literature. We know too that frequent snacking could encourage weight gain and this is something that we need to caution against, given the current obesogenic climate.

Why do patients snack in the context of diabetes?
Most commonly, patients snack because of:
- Previous education (they have been told to snack)
- A fear of hypoglycaemia / defensive eating
- Habit
- Physiological hunger*
- Emotional need
- A belief that it aids weight loss
- A belief that it stabilises blood glucose levels

Valid Reasons that may necessitate snacking in the context of diabetes
- Medication
  - Some oral hypoglycaemic agents
  - Some insulin regimens
- Exercise
- Physiological hunger*

*Physiological hunger is recognised by stomach sensations such as ‘stomach rumbling’. Generally, there is a feeling of emptiness and even pain in the stomach. One may feel light headed and tired if it has been about 4-5 hours since the last meal.

Psychological or emotional hunger on the other hand is not caused by an actual physical need for food. It is a desire to eat from habit, because food is there, a need for comfort due to an emotional upset, or simply because the food tastes good.

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Liana Grobbelaar
email: Liana@cdecentre.co.za
If patients are prescribed nutritional and exercise therapies, there is no specific need for snacking. This may be decided on balance of individual preference versus metabolic treatment targets.

**Hypoglycaemic Agents**

**Insulin Regimens**

The different insulin action profiles below serve as guide as to whether a snack is needed or not.

- **Multiple daily injections (MDI) with insulin analogues**
  With an MDI, basal-bolus regimen using a **quick acting insulin analogue** (Apidra, NovoRapid or Humalog), combined with a **long acting analogue** (Lantus or Levemir), no snacks are needed.

  If a patient has a preference to snack, then additional insulin will be needed at the time of the snack. The carbohydrate content of the snack will determine the required dose. If a non-carbohydrate snack is chosen, (e.g. nuts, cheese, cold meat, egg, biltong, vegetables), no additional insulin is required.

- **MDI with regular and NPH insulins**
  **Short acting regular insulin** (Actrapid / Humulin R / Biosulin R) given before each meal covers the meal about to be eaten and may cover a small 10-20 g carb snack (e.g. a fruit or yoghurt, depending on the size and type) in between meals.

  **Intermediate acting insulins** (Protaphane / Humulin N / Biosulin N), used in a basal role, tend to peak in a non-physiological manner in the early hours of the morning. A bedtime snack may be needed to prevent overnight hypoglycaemia.

<table>
<thead>
<tr>
<th>Drug (Year of First Release)</th>
<th>Examples</th>
<th>Risk of Hypoglycaemia</th>
<th>Indication for a snack</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulphonylureas (1946)</td>
<td>• gliclazide; • glimepiride; • glibenclamide</td>
<td>↑</td>
<td>Oral drugs that have a high risk of hypoglycaemia may need careful monitoring.</td>
</tr>
<tr>
<td>Biguanide (1957)</td>
<td>• metformin</td>
<td>↓</td>
<td>If patients continually need to snack, this may be a sign that the dose should be adjusted.</td>
</tr>
<tr>
<td>Thiazolidinediones (1997)</td>
<td>• pioglitazone</td>
<td>↓</td>
<td>Snacks should not routinely be recommended unless mild, but frequent hypoglycaemia occurs.</td>
</tr>
<tr>
<td>DPP-4 inhibitors (2006)</td>
<td>• sitagliptin; • vildagliptin; • saxagliptin</td>
<td>↓</td>
<td>If this occurs, a dose reduction may also be considered.</td>
</tr>
<tr>
<td>Glucagon-like-peptide mimetics (2005)</td>
<td>• exenatide; • liraglutide</td>
<td>↓</td>
<td>Snacks should not routinely be recommended unless mild, but frequent hypoglycaemia occurs.</td>
</tr>
<tr>
<td>Insulin (1921)</td>
<td>• rapid acting; • short acting; • intermediate acting; • long acting; • biphasic premixed: - human - analogue</td>
<td>↓ or ↑</td>
<td>Snack recommendations will be dependent on the type of insulin regimen. This will be discussed below.</td>
</tr>
</tbody>
</table>

*↑ = high risk, ↓ = low risk
• **Twice daily pre-mixed insulin with a biphasic action**

Regular pre-mixed insulin (Actraphane / Humulin 30/70 / Biosulin 30/70 / Insuman Biphasic) may need mid-morning and bedtime snacks due to non-physiological high levels of insulin in between meals.

Twice daily pre-mixed analogue insulins (Humalog Mix 25 / Humalog Mix 50 / NovoMix 30) may or may not require between meal snacks. The usual advice for regular insulin pre-mixes is that they would require a snack mid-morning and bedtime. However because these mixes contain rapid acting insulin analogues, there may be less overlap at these time points. The need for snacking will depend on individual blood glucose responses.

• **Continuous Subcutaneous Insulin Infusion (CSII) – Insulin Pump Therapy**

With CSII providing a pattern of insulin secretion that is more physiological, there should be no need to snack, provided the basal insulin replacement rates are correct. However if snacking is a preference, one advantage of CSSI is that it removes the need for continual injections (One of the reasons for poor control on MDI, where snacks are not covered). Patients must however ensure that they bolus sufficiently for the snack by entering the correct carbohydrate value into the pump. Another advantage is that the pump will take into account the active insulin still available, thereby preventing ‘stacking’ of insulin. The ease of snacking and simply bolusing, however, may lead to an over consumption of energy, which could result in weight gain.

**Exercise and snacking**

Carbohydrate supplementation and snacking may be one of the measures to prevent hypoglycaemia during moderate intensity exercise, such as running or cycling. It is important to try to preserve euglycaemia during exercise. However, it is not always possible to plan for exercise, and thereby make the necessary insulin adjustments prospectively. Thus carbohydrate intake (pre, during and post exercise) allows individuals with diabetes greater flexibility to manage the balance between exercise and glycaemia. Rigid carbohydrate supplementation without regard for pre-exercise blood glucose, previous metabolic response to exercise and individual insulin therapy is no longer appropriate. Such an approach may negate the beneficial glycaemic lowering effects of exercise in type 1 diabetes specifically and is why certain studies do not show improvements in HbA1c.

One would also need to consider whether exercise is part of a weight loss plan, as carbohydrate supplementation may blight weight loss goals. In this situation, one would strive as far as possible for insulin dose adjustment prior to exercise. This will involve an increase in the frequency of testing blood glucose levels, to ascertain specific adjustments.

Some evidence suggests that a low glycaemic index snack pre-exercise may improve aerobic performance in people with diabetes. The resulting sustained release of glucose can be useful in exercise that is more prolonged.

Recommendations all depend on the duration, intensity and type of exercise and it is best for patients to discuss this with their diabetes team for individual advice.

**Weight loss and snacking**

Obesity is a concerning risk factor associated with type 2 diabetes and weight loss is a fundamental part of the treatment. Most weight-loss diets advocate eating frequently to increase metabolism, reduce hunger and food cravings, improve glucose control, and reduce body weight. This offers an enticing dietary strategy to weight loss and / or maintenance of a healthy body weight. The limited number of publications surrounding this topic makes it difficult to know the exact impact of snacking on weight and appetite control.

A small, randomized, controlled trial (2008) asked the question ‘Should snacks be recommended in obesity treatment?’ 140 patients were randomized to two treatment arms; three meals per day or three meals and three snacks per day. Only 93 patients completed the study after a one-year period. The results showed no significant difference in weight loss in the groups having snacks and those without snacks.

In a cross sectional, descriptive study looking at the frequency of snacking in relation to energy intake and food choices in obese men and women compared to a reference population, the findings concluded that obese subjects snacked more frequently irrespective of physical activity. Snacks were positively related to energy intake. The contribution to energy intake was because snack choices were mainly from sweet and fatty food groups. The take-home message was that snacking must be carefully considered in obesity management.

A brief review of controlled-feeding studies (2011) mainly targeted appetite, hormonal and food intake responses...
potentially altered with eating frequency. They concluded that increased eating frequency, defined as eating more than three meals per day, was associated with minimal to no improvement in appetite control. However, they did find that reduced eating frequency, defined as less than three meals per day, appears to affect appetite control negatively. With these results, we can possibly assume that at least three meals a day can be justified but claims that increased eating frequency reduces hunger and improve glucose control are unsubstantiated.

Recommendations for weight loss should focus on the overall calorie intake of an individual instead of meal frequency.

**Appropriate options for snacking**

No concrete definition for a snack exists, and thus assessing your patient’s perspective of a snack is crucial.

A proposed definition for a snack is: ‘A snack is composed of solid food(s), including those typically eaten with a utensil (with or without a beverage) that occurs between habitual meal occasions for the individual, is not a substitute for a meal and provides substantially fewer calories than would be consumed in a typical meal’.

It is important to choose sensible, healthy snacks. We encourage patients to choose snacks with a high nutritional value to utilise an extra opportunity to consume vital nutrients. To prevent weight gain, it is suggested that a snack should contain not much more than 500 kJ or 120 kcal.

In conclusion, snacking should not be mandatory in the context of diabetes, but should be purposeful according to identified need. Consideration of individual treatment parameters is necessary before making snacking recommendations.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Carbohydrate Value</th>
<th>Energy value (kcal)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cherry tomatoes / baby carrots / celery sticks with low fat cream cheese (50 g)</td>
<td>negligible</td>
<td>125</td>
</tr>
<tr>
<td>Olives (25 g), about 8-10</td>
<td>negligible</td>
<td>30</td>
</tr>
<tr>
<td>Small handful of nuts or seeds (20 g)</td>
<td>negligible</td>
<td>138</td>
</tr>
<tr>
<td>Avocado or guacamole dip (45 g) with crudites</td>
<td>negligible</td>
<td>58</td>
</tr>
<tr>
<td>Biltong snapsticks (50 g)</td>
<td>negligible</td>
<td>103</td>
</tr>
<tr>
<td>Cheese wedge (One) or low fat cheese (25 g)</td>
<td>negligible</td>
<td>48 or 68</td>
</tr>
<tr>
<td>¼ cup of fresh raspberries (160 g)</td>
<td>&lt; 10 grams</td>
<td>40</td>
</tr>
<tr>
<td>2 Provita® crackers topped with cottage cheese (50 g)</td>
<td>&lt; 10 grams</td>
<td>99</td>
</tr>
<tr>
<td>Slim slab</td>
<td>&lt; 10 grams</td>
<td>67</td>
</tr>
<tr>
<td>Small diet yoghurt (check label)</td>
<td>&lt; 10 grams</td>
<td>50</td>
</tr>
<tr>
<td>Bag of Flings®</td>
<td>&lt;10 grams</td>
<td>58</td>
</tr>
<tr>
<td>Slice of Albany Ultima KJ Controlled Bread®</td>
<td>10.5 grams</td>
<td>67</td>
</tr>
<tr>
<td>Small apple or orange</td>
<td>10-20 grams</td>
<td>62</td>
</tr>
<tr>
<td>Cup of vegetable soup</td>
<td>10-20 grams</td>
<td>77</td>
</tr>
<tr>
<td>2 rice cakes with 1 tbsp peanut butter</td>
<td>10-20 grams</td>
<td>152</td>
</tr>
<tr>
<td>1 slice of toast with margarine &amp; Bovril</td>
<td>10-20 grams</td>
<td>130</td>
</tr>
<tr>
<td>4 Pro-Vita crispbreads with ricotta cheese (50 g)</td>
<td>10-20 grams</td>
<td>168</td>
</tr>
<tr>
<td>Glass of milk (2 %)</td>
<td>10-20 grams</td>
<td>130</td>
</tr>
<tr>
<td>Popcorn (2 cups), air popped</td>
<td>10-20 grams</td>
<td>62</td>
</tr>
<tr>
<td>Sugar free hot chocolate, made with 200 ml skim milk</td>
<td>10-20 grams</td>
<td>94</td>
</tr>
<tr>
<td>Bran muffin</td>
<td>20-30 grams</td>
<td>170</td>
</tr>
<tr>
<td>Cereal bar</td>
<td>20-30 grams</td>
<td>82</td>
</tr>
<tr>
<td>4 Ryvitas with cottage cheese (75 g)</td>
<td>20-30 grams</td>
<td>215</td>
</tr>
<tr>
<td>Yoghurt (125 g) and a banana</td>
<td>20-30 grams</td>
<td>179</td>
</tr>
<tr>
<td>Baked beans (80 g) on toast</td>
<td>20-30 grams</td>
<td>152</td>
</tr>
<tr>
<td>Sandwich, egg mayonnaise</td>
<td>20-30 grams</td>
<td>300</td>
</tr>
</tbody>
</table>
Seen at the 16th CDE Postgraduate Forum in Diabetes Management

8-10 August 2014, Birchwood Hotel & OR Tambo Conference Centre, Boksburg, Gauteng
Seen at the 16th CDE Postgraduate Forum in Diabetes Management

8-10 August 2014,
Birchwood Hotel & OR Tambo Conference Centre, Boksburg, Gauteng
It is interesting how each year the annual CDE Forum takes a different course. The organisers use their creative skills and try to keep it interesting for all who attend. Past, varied programmes have included small breakaway sessions, overseas speakers, themed lectures and workshops. Delegates have also experienced innovative and interesting sessions like language and cultural differences in patients and participatory exercise classes. One of the 2014 Forum international speakers, Emeritus Professor David Owens from the Institute of Molecular & Experimental Medicine at the Cardiff University School of Medicine in Wales, stated that in his opinion, the CDE Forum was the best meeting on diabetes in the world! This year we again experienced some superb lectures from esteemed speakers, but three mini-symposia on Nutrition, Hypoglycaemia and Education kept the meeting format interesting. Of special interest were several lectures that paid attention to cognitive function.

Diabetes Update Symposium
The 9th Diabetes Update Symposium, a now traditional Novo Nordisk / ACCU-CHEK sponsored satellite meeting prior to the CDE Forum, took place on the Friday of the Forum weekend. Acknowledged expert on hypoglycaemia, Prof Brian Frier, Consultant Physician in Internal Medicine and Diabetes at the Royal Infirmary of Edinburgh and Honorary Professor of Diabetes at the University of Edinburgh in the UK, presented an informative view of hypoglycaemia. He reminded the audience that hypoglycaemia is not limited to type 1 diabetes. Those with type 2 diabetes also experience hypoglycaemia, but it is often not recognised as such. Importantly, for the elderly, hypoglycaemia can prove fatal. He concluded that the
elderly should test or be tested more often and their poor memory, resistance to treatment and complacency should be taken into account in identifying possible hypoglycaemic events.

Johannesburg Paediatric Endocrinologist, Dr. David Segal, looked at glycaemic variability, which may be associated with increased cardiovascular risk with intensive treatment regimens. He noted the availability of insulin analogues, which could help to reduce variability, but patient behaviours were just as important. To reduce glycaemic variability, patients should be more conscientious about taking each insulin dose, reducing their dietary carbohydrate intake and performing a combination of aerobic and non-aerobic exercises.

Cape Town Endocrinologist, Dr Joel Dave spoke about current therapies and their ability to meet the needs of type 2 diabetes, that is, to reduce complications and stop beta cell failure. He concluded that beta cell failure still occurs over time in spite of current therapies. With oral agents, the risk of hypoglycaemia is least with gliclazide. Most therapies result in weight gain, but metformin and dipeptidyl peptidase-4 (DPP-4) inhibitors are more weight neutral, glucagon-like peptide-1 (GLP-1) analogues are beneficial and insulin detemir may lead to a little weight loss for those who have a body mass index (BMI) of 25 kg/m² or more. Low carbohydrate diets proved effective in increasing beta cell function and preventing the onset of diabetes in those who had ‘pre-diabetes’ (intermediate hyperglycaemia).

In a second session at the Diabetes Update Symposium, Prof Frier discussed the ability of two new therapies to meet the needs of type 2 diabetes. Sodium-glucose co-transporter-2 (SGLT2) inhibitors act on the proximal tubule in the kidneys, usually responsible for 90% of glucose reabsorption from urinary filtrate, to decrease blood glucose by increasing renal glucose excretion. He felt that this new class held some promise. The highly selective SGLT2 inhibitor, dapagliflozin, showed benefits in glycaemic control, reduced dose of insulin and in weight reduction. There was no risk of hypoglycaemia and blood pressure was lower. The most common adverse effect was an increased risk of urinary tract infections.

Clinical Pharmacologist, Professor Jacques Snyman, Business Development Director at Agility Global Health Solutions, gave the final lecture of the day with a discussion on what comprised ethical relationships between health care providers, health care funders and patients. He highlighted how money as a motivator in practice can give rise to unethical behaviours, especially in research projects. Providers, industry and patients have equal responsibility to keep informed, do research and be ethical in their behaviour. He concluded that there is a delicate balance between ethical behaviour and greed with a high risk of legal repercussions if this balance was not achieved.

Product Launches
It makes sense that a large weekend event like the CDE Forum, with the entire spectrum of the wider diabetes team present, offers the perfect platform for new product launches. Two diagnostic companies made good use of the opportunity to launch their products at satellite events prior to the formal Forum academic sessions:

SMBG – The next step in the journey... Hosted by Abbott Diabetes Care
Concurrent to the National CDE AGM, which discussed the challenges and possible solutions in the managed care of diabetes, others were taken on a historical journey with Diabetes Specialist Nurse Michael Brown, as a prelude to the launch of a new Abbott blood glucose meter.

Michael reminded us of the delicate balance that exists between insulin and other diabetes therapies and life (including food, exercise, stress, time, money and other health problems and their medications) in attempts to attain and maintain healthy blood glucose levels. Starting with an overview of the problem of diabetes, patient expectations from their blood glucose meters, and insights into proxy and direct, and qualitative and quantitative measures of glycaemic monitoring, Michael began the journey by looking at the characteristics of the smallest and most responsive glycaemic sensor prototype, the human beta cell. With this in mind, he took us back as far as 1552 BC to the first written record of diabetes and polyuria by Hesy-Ra. Observation that the urine of people with diabetes attracted ants and bees provided the first crude qualitative test for the presence of diabetes. Over the next two and half millennia, physicians gradually developed greater insights into this condition. From the crude medieval proxy measure of ‘taste-testing’ urine, the discovery and production of insulin from 1922 spurned a search into monitoring the outcomes of therapy. We briefly relived the last 92 years of the journey wherein some of the greatest advances were made - from the first test strips for urine glucose to the small, modern, second-generation blood glucose meters.

The scientific and historical background was set and the Abbott Diabetes Care (ADC) Team launched the FreeStyle Optium Neo blood glucose and ketone monitoring system. With a beautifully clear ‘Paperwhite’ screen and a design focus on improving daily diabetes management for people with diabetes, the Neo features visual glucose trend indicators, monitoring of blood ketone levels and insulin logging. Additionally, it has a programmable insulin-dosing feature, for use in consultation with the diabetes team. Meter data can be downloaded onto a computer where glycaemic trends can be viewed and analyzed.
FreeStyle

Optium Neo

The FreeStyle Optium Neo Blood Glucose Monitoring System from Abbott Diabetes Care

The Abbott Diabetes Care team at the CDE Forum August 2014
Accu-Chek/Novo Nordisk hosted ‘Are you ready to connect?’ Diabetes Evening

This evening launch was great fun and introduced by Talk Radio 702’s morning ‘trafficologist’ and technology and gadget guru for Cape Talk and 702, Aki Anastasiou. Aki immediately ‘connected’ with the theme of the evening “Better Connected for Better Decisions” and the audience, by presenting a thought-provoking history of communications technology. He gave insights into the future of this ever-changing space and shared how the field of health care could benefit by embracing new, smarter devices and means of connectivity and data sharing and storage. Forum weekend attendees were the first to experience the innovative Accu-Chek® Connect diabetes management system, which enables wireless connection of a blood glucose monitor to a patient’s Smartphone ‘app’, and then, into the ‘cloud’. This means that anytime, anywhere, both patient and their authorised health care provider can share, review and learn from the collected blood glucose data, without the meter even needing to be at the consultation. Additionally, a mum can see how her child is doing whilst away at school, or even a loved one can receive updates of their partner during the workday. The app is the first to feature the ‘bolus advisor’ from Accu-Chek, meaning that not only are blood glucose results shared, but insulin advice can be given and captured as well.

Indeed, this new system from Accu-Chek shows how healthcare and technology are merging, so that we can make better decisions by being better connected. Dr David Segal reviewed the new system for the delegates, and Cape Town Endocrinologist, Dr Landi Lombard, showed some results already seen among patients who have experienced ‘connected living’.

These launches marked further steps in an on-going evolution towards greater patient independence in the self-management of diabetes.

The CDE Forum Academic Sessions

Emeritus Professor at Cardiff University, Prof David Owens began the Forum with an informative and comprehensive comparison of seven different glucagon-like peptide-1 (GLP-1) analogues, current and future, under the headings below, highlighting the pros and cons of each.

Prof Brynne Ascott-Evans, Head of the Division of Endocrinology and Diabetes, Stellenbosch University and Tygerberg Hospital, looked at incretins as treatment for beta cell disease. He addressed three important questions:

a) do the incretins offer beta cell protection?
b) do incretins influence cardio-vascular outcomes?
c) do incretins come with a higher risk of pancreatitis or pancreatic cancer?

We have in-vitro and in-vivo evidence of beta-cell protection from animal models, but only in-vitro evidence of preserved human islet morphology. In theory, cardio-vascular outcomes should be improved, but we have no prospective powered study data yet. The risk of pancreatitis or pancreatic cancer is inconclusive but it would seem the benefits far outweigh the risks.

The Annual ‘Ascending Star’ Lecture

Recent Cardiff University Graduate, Dr Neville Wellington, had the honour of delivering the ‘Ascending Star’ Lecture entitled “Glucose, Oxidative Stress and Low Carbohydrate Lifestyles”. This annual lecture opportunity, paired with a Novo Nordisk Travel Award, recognises the doctor or educator who achieved the top marks in either the Cardiff University or University of South Wales Postgraduate Diploma in Diabetes in the previous year. Neville began by pointing out how both carbohydrates and insulin have the potential to make us fat and how obesity is a response to dietary stimulus. He showed how excess dietary carbohydrate and resultant hyperglycaemia caused damage to the endothelial cells via inflammation and the build up of plaque in the arteries. Even transient hyperglycaemia increases the inflammation of aortic endothelial cells for up to 6 days after blood glucose normalises. He discussed how a low carbohydrate diet improved all aspects of the ‘metabolic syndrome’ effectively and how those with an intake of 50 g of carbohydrate per day improved dramatically. He felt that anyone with diabetes should have low carbohydrate, low fat meal plans and include proteins, greens, avocados and olive oil in their menu.
Mini-symposium on Nutritional Approaches for Diabetes

Three Registered Dieticians looked at how dietary interventions can improve diabetes control.

Mrs Ria Catsicas noted that the Look AHEAD (Action for Health in Diabetes) study showed that weight loss and fitness reduced cardiovascular disease (CVD) events for up to 4 years in older patients with type 2 diabetes. It also increased their mobility, reduced sleep apnoea and reduced the amount of medication needed. She highlighted the importance of eating consistently, small quantities, the right kinds of foods, drinking water as opposed to carbohydrate drinks and doing regular exercise. She suggested behaviour modification techniques such as goal setting, using food diaries, and contact with the patient 2-4 times a month for reinforcing feedback and motivation.

Mr Hamish van Wyk critically evaluated several studies and guidelines on recommended ‘diets’ for people with both type 1 and type 2 diabetes. On a practical level, he recommended that nutritional interventions for diabetes should be patient specific, with low carbohydrates - as low as the patient will go.

Ms Michelle Daniels presented an evidence-based case for carbohydrate (CHO) counting as an effective strategy to improve glycaemic control and promote better quality of life in both type 1 and type 2 diabetes. Group education programmes like DAFNE (Dose Adjustment for Normal Eating) and DINE (Diabetes Insulin and Normal Eating) enable patients to ‘Carb-Count’ successfully. She used case studies to illustrate how to do it. However, over and above CHO counting, she explored the need for protein and fat counting, since these give rise to post-prandial glucose increases too and shared how to do this. High fat content in a meal will be absorbed more slowly than CHO, so insulin doses would need to be programmed in 2 parts – a normal bolus for CHO at mealtime and a later bolus 3-8 hours later depending on the quantity of fat/protein units.

Mini-symposium on Hypoglycaemia

Prof Frier presented a very detailed and comprehensive lecture on the pathophysiology of hypoglycaemia. He made a number of key points. Because, in the resting state, the brain uses 80% of available glucose to function, hypoglycaemia has a profound impact on cognitive performance, particularly in reducing speed of response. In the long term, frequent hypoglycaemia can reduce the young child’s intelligence quotient (IQ) and fluid intelligence. There is not too much change in young adults. In the elderly, crystallised intelligence remains intact, but they are at more risk of accelerated dementia. If the elderly have a lower IQ at the start, they are more likely to have more hypoglycaemia events, which have serious implications for the brain. ECG abnormalities and arrhythmias, particularly at night, occur more frequently from hypoglycaemia than hyperglycaemia. Loss of awareness of hypoglycaemia occurs in 25% of adults and 20% of children. This group has a 3-6 times greater risk of severe hypoglycaemia. The elderly have a higher risk of morbidity since the diagnosis of hypoglycaemia may be missed or misinterpreted, especially in the context of co-morbidities.

Boksburg Specialist Physician, Dr Angela Murphy, looked at clinical implications of chronic recurring hypoglycaemia for children, the elderly and during pregnancy. Mortality is rare but there is a risk of being found ‘dead in bed’ in patients who have underlying susceptibility through autonomic dysfunction. Seizures affect fronto-cerebral function of the brain and have an effect on concentration, memory, speed and fine-motor coordination, especially in children. The effect on vision is blurring, double vision and a significant reduction in retinal and contrast sensitivity. Many changes occur in the heart during hypoglycaemia. Children experience enuresis. Hypoglycaemia has a significant impact on quality of life - it affects mood so that patients feel a loss of control, patients have to monitor before and during exercise and driving; 25% of patients reported missing time and being less productive during working hours and patients with diabetes are often excluded from being firemen or commercial pilots.

Dr Joel Dave looked more closely at cognitive functioning in type 2 diabetes. Since about 64% of patients have HbA1c levels above target, he posed 4 questions, the first being “Is there more cognitive impairment in this population?” Executive function impairment was found in 52% of patients in a study done in Cape Town. Those in midlife showed a cognitive decline, with a decreased amount of grey and white matter especially in the frontal and temporal lobes. There appeared to be an association between retinopathy and dementia. Diabetes increases the risk of cognitive dysfunction and increases the rate of cognitive decline. “Why this is true?” was the second question. He presented plausible pathophysiological mechanisms for diabetes causing cognitive decline. The answer to the third question “Is it preventable?” was that no robust evidence proves that good glycaemic control can improve cognition. The answer to the final question “Is cognitive decline associated with poor diabetes control” was yes, it is. In clinical practice, we should screen patients with type 2 diabetes for cognitive impairment at baseline and for cognitive decline at least annually using validated methods such as the General practitioner assessment of cognition (GCOG), Mini-Cog and the Memory Impairment Screen (MIS). These tools are available on the internet. We should also remember to screen for vitamin B12 deficiency in patients on metformin.
LifeScan, manufacturers of the OneTouch® brand of Blood Glucose Monitoring Systems, is aware of some of the challenges that people with diabetes and their families face on a daily basis, especially around maintaining motivation levels and sticking to their diabetes management plan.

OneTouch® is excited to launch to the South African public, a brand new on-line diabetes education portal, called Lamasat – meaning Simple Touches. The Lamasat Programme is designed to empower people with diabetes and their family members with the right knowledge and the correct tools to guide them through their journey with diabetes.

Lamasat simplifies diabetes management principles into healthy lifestyle actions, which we call the 4 C’s. If you have diabetes, you should do these simple things every day:

• Check your blood glucose levels
• Control your blood glucose highs and lows
• Consume healthy food
• Care for your health and well-being

Best of all, membership to the Lamasat Programme is free! The Programme has 5 stages, each providing bite-sized portions of information and practical tools, to apply what you learn into your daily life.

The Lamasat Programme allows members to:

Know More – through reading and the provision of enduring educational material

Do Things – with steps and actions that will help build healthy diabetes lifestyle habits

Feel in Control – through attending events and interactions that motivate, inspire and build confidence.

It’s quick and easy to register for the free membership to the Lamasat Programme:

1. On the Internet @ www.4cprogramme.com
2. By phone: Contact the toll-free* OneTouch® Customer Care line on 0800 600 345 (weekdays 9 am-5 pm)
3. Or E-mail us @ cclifescan@its.jnj.com and ask for more info about the Lamasat Programme

*Toll-free via a Telkom landline
Facilitated Case Discussion
An expert Physician and Endocrinologist panel consisting of Drs Adri Kok, Greg Hough, Duma Khutsoane and Prof Pankaj Joshi closed the day with a facilitated discussion of a clinical case study on a patient with type 2 diabetes. They reviewed current and potential medication regimen of this patient. He had notable cognitive dysfunction and poor memory. The question asked was, “Could the cognitive dysfunction be improved?” Evidence from the ACCORD Ming study suggested that the answer was no. When he presented with chest pain and further complications, including peripheral neuropathy and retinopathy followed by acute pancreatitis, it was noted that with his many episodes of hypoglycaemia there was an increase in complications. They again recommended that a baseline of cognitive function should be taken early on and this should be re-evaluated regularly.

The 16th CDE Forum Awards Dinner
As usual, this Annual Dinner provided a pause in the formal academic proceedings. A number of South African Graduates of Cardiff University and the University of South Wales Postgraduate Diplomas and MSc Degrees in Diabetes, attended a graduation ceremony to receive their Qualifications. In addition, the following CDE Providers were honoured for Clinical Excellence:

- Sr Laurie van der Merwe from Empangeni received the Servier ‘Diabetes in the Community’ Award for the ‘CDE Centre with the Most Community Involvement’.
- Dr Gerhard Stemmet from Bloemfontein received the Sanofi-Aventis Award for the Centre providing ‘Exceptional service to patients in a previously disadvantaged community’.
- Dr Gerhard Herbst from Standerton received the Sanofi-Aventis Award for the Centre with the ‘Most Improved Glycaemic Control’.
- Dr Hans Snyman from Brits received the Sanofi-Aventis Award for ‘Good Clinical Practice’.
- Sr Razana Allie from Johannesburg received the LifeScan Annual Travel Award for the ‘Diabetes Educator showing the best personal and professional development & growth during the past year’.
- Dr Neville Wellington from Kenilworth in Cape Town received a Novo Nordisk Travel Award, paired with the opportunity to present the Annual ‘Ascending Star’ Lecture at the 2014 CDE Forum.
- Sr Sheridan Williamson from Port Elizabeth received the Novo Nordisk Award for the ‘Top Diabetes Educator’.
- Dr Cornelis de Hoog from Krugersdorp received the Novo Nordisk Award for the ‘Best CDE Centre’.

Well done to all the winners!
Ms Angie Naidoo from Sanofi Aventis presented the Sanofi-Aventis Award for the Centre with the “Most Improved Glycaemic Control” to Dr Gerhard Herbst.

and the Sanofi Award for “Good Clinical Practice” to Dr Hans Snyman from Brits

Ms Anna Alexeeva from LifeScan presenting the LifeScan Travel Award for the Diabetes Educator who has shown the best personal and professional development and growth during the past year to Sr Razana Allie

Mr David Broomfield from Novo Nordisk presenting the Novo Nordisk “Ascending Star” Award for the doctor who achieved the top marks in the Cardiff Diabetes Diploma last year to Dr Neville Wellington

Sr Sheridan Williamson receives the Novo Nordisk Award for the ‘Top Diabetes Educator’ from Mr David Broomfield of Novo Nordisk.

Prof Larry Distiller receives the Novo Nordisk Award for the ‘Best CDE Centre’ from Mr David Broomfield of Novo Nordisk on behalf of the winner, Dr Cornelis de Hoog.
Beyond HbA1c: Other measures of glycaemic control

Durban Endocrinologist, Dr Hoosen Randeree, started the second day of the Forum by looking at different ways of measuring glycaemic control other than HbA1c, including fasting glucose, postprandial glucose, and glycaemic variability. He spoke of standard deviation of readings and of continuous glucose monitoring as being useful ways of illuminating glycaemic variability.

Mini-symposium on Education Techniques

Empangeni-based Diabetes Specialist Nurse, Sr Laurie van der Merwe, presented a case for using structured education programmes in diabetes. The outcome of programmes such as DAFNE (Dose Adjustment for Normal Eating) proved to be cost-effective with a 34 % savings on medications, the development of fewer complications and increasing lifespan by four or more years. HbA1c values were lowered with a 60 % reduction in hypoglycaemia. It was especially good for newly diagnosed patients. DESMOND (Diabetes Education and Self Management for Ongoing and Newly Diagnosed) increased patient knowledge and quality of life. Structured programmes should however be adapted to individual patients needs as well as to the South African context.

Johannesburg Diabetes Specialist Nurse, Sr. Hester Davel noted that the education ‘push’ to provide goals and solutions for the patients should be balanced with health coaching ‘pull’ approach, where patients are empowered to take responsibility for their diabetes and to solve their own problems once they have the knowledge to apply. The educator needs to be flexible and work with each patient according to their particular needs as defined by themselves.

Consultant Endocrinologist from the University Hospital of Wales in Cardiff, Dr Steve Davies, talked about social networking as an educational tool. Patients spend about 8 hours per annum with health practitioners. For the rest of the year, the patient needs to practice self-management. Social networking and other electronic and internet-based media including smart phones, messaging ‘apps’, Facebook, Twitter and YouTube can assist clinicians to reach more patients and in facilitating peer-to-peer support. Topics included could be current opinions, case blogs, ‘expert patient’ stories, and education. At present, only 7 % of doctors use social networking yet, potentially, it is an excellent medium of communication.

Gestational Diabetes- A quest for consensus

Johannesburg Paediatrician and Critical Care Specialist, Dr Despina Demopoulos, Cape Town Endocrinologist, Dr Magda Conradie and Durban-based Diabetes Specialist Nurse, Sr Fiona Prins formed the panel for this discussion, expertly facilitated by Dr Stan Landau, on the best care for a woman with gestational diabetes. The paediatrician wanted to know more about the mom’s control and the condition of the baby, the endocrinologist wanted a whole team of practitioners on board, including an obstetrician, a medical officer, an educator and a dietitian, and the diabetes educator wanted to prepare the mom for a successful outcome by helping her control her blood glucose levels. The discussion concluded with a call for the approach to gestational diabetes to be standardised, and recognition of the unique role of and insights provided by each member of the care team.

Longevity in type 1 diabetes

CDE Principal Physician and Managing Director, Prof. Larry Distiller, ended the Forum on a positive note as he reviewed the reasons why some people with type 1 diabetes live so long. Early glycaemic control was important although it became less so as the patient got older. Patients with higher HDL cholesterol levels, lower daily insulin requirements, no central obesity, lower blood pressure, and those who did not smoke, nor had microalbuminuria after 15-20 years could potentially live longer than others could. A family history of longevity also made a difference to the length of time the patient lived.

This 2014 Forum provided all delegates with new motivation, new ideas and new hope that our efforts make a difference to people with diabetes. To end with a phrase often used by Prof Distiller, “Now, let’s go and save some lives!”

Mr Lesley Philips talking about the newly formed South African Chapter of the Cardiff University Alumni Association
Previous Graduate of the Cardiff University Postgraduate Diploma in Diabetes, Dr Julien Trokis, paying tribute to the Graduands and highlighting the enormous effort needed to achieve this qualification.

Emeritus Professor at Cardiff University, Prof David Owens conferred Postgraduate Diplomas and MSc Degrees on four Graduands

Dr Hemant Makan (MSc)

Dr Rohit Dulabh
Dr Ruth Davis, Academic Course Director from the University of South Wales, assisted by Prof Larry Distiller, conferred the Postgraduate qualifications in Diabetes from the University of South Wales.

Dr Ruth Davis congratulating the MSc Degree Graduates.

Dr Mahomed Asvat

Dr Louis Botha

Jacqueline Lubbe
Dr Ruth Davis congratulating the Postgraduate Diploma Graduates

Linda Reid

Helen Cyster

Daniel Kabuzi

Petronella van Tonder
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