Dietary advice for people with diabetes: the role of carbohydrate in dietary treatment and an assessment of video education

Pamela A. Dyson (2010)

Dietary advice for people with diabetes: the role of carbohydrate in dietary treatment and an assessment of video education

Pamela Ann Dyson

A thesis submitted in partial fulfilment of the requirements of Oxford Brookes University for the degree of Doctor of Philosophy

Volume I

July 2010

Abstract

This thesis investigated novel approaches to the delivery of lifestyle education for people with diabetes. The principles of dietary advice for diabetes recommend a high carbohydrate intake, yet carbohydrate foods raise blood glucose levels significantly. Study 1 was designed as a randomised controlled trial to evaluate the effect of a low carbohydrate (LC) and low fat (LF) diet on glycaemic control and body weight in 26 subjects. Weight loss was greater in the LC group (-6.9kg v -2.1kg, p=0.003). Glycaemic control improved in both groups with a reduction in A1c in both LC and LF groups (-0.3% v -0.2%, p=0.582). There were no significant changes in cardiovascular risk assessed by lipid levels and blood pressure.

Study 2 was designed as a randomised controlled trial to assess a novel education programme delivered by video for 42 people newly diagnosed with Type 2 diabetes. At six months follow-up, there was a significant increase in knowledge in the video intervention group (p=<0.0001). There were reductions in A1c (-0.7% v -0.6%, p=0.843), total cholesterol (-0.5mmol/l v -0.2mmol/l, p=0.347) and LDL cholesterol (-0.5mmo/l v 0.2mmol/l, p=0.1), and physical activity increased in the intervention group. There were no changes in the control group, but these differences failed to reach between group significance.

Study 3 was an intervention study examining structured education in 51 people with Type 1 diabetes. At one year's follow-up, there was a significant improvement in A1c levels (-0.3%, p=0.03) with no increase in body weight or hypoglycaemia. Diabetes related distress improved significantly at six months follow-up and this was maintained at one year (p=0.019).

These studies indicate that both education and modification of carbohydrate intake have a positive effect on outcomes in people with diabetes. People with Type 2 diabetes show increased knowledge after video education, and can achieve significant weight loss by adopting a low carbohydrate diet. People with long-standing Type 1 diabetes can significantly improve glycaemic control and quality of life by adopting a strategy of carbohydrate counting and insulin adjustment.

Table of Contents

List of figures

List of Tables

List of Abbreviations

ADA	**American Diabetes Association**
AE	**Adverse Event**
ADDQOL	**Assessment of Diabetes Dependent Quality of Life**
ADKnowl	**Assessment of Diabetes Knowledge**
BDA	**British Dietetic Association**
BITES	**Brief Intervention in Type 1 diabetes, Education for Self-efficacy**
BMI	**Body Mass Index**
CPENH	**California Pan-Ethnic Health Network**
CRF	**Case Record form**
CV	**Co-efficient of Variation**
DAFNE	**Dose Adjustment for Normal Eating**
DAWN	**Diabetes Attitudes, Wishes and Needs**
DCCT	**Diabetes Control and Complications Trial**
DEN	**Diabetes Education Network**
DES	**Diabetes Empowerment Scale**
DESMOND	**Diabetes Education and Self-management for Ongoing and Newly Diagnosed**
DHP	**Diabetes Health Profile**
DM	**Diabetes Mellitus**
DOH	**Department of Health**
DPP	**Diabetes Prevention Programme**
DPP-4	**Dipeptyl Peptidase 4**
DPS	**Diabetes Prevention Study**
DQOL	**Diabetes Quality of Life**
DVD	**Digital Video Disc**
ECG	**Electrocardiogram**
EQ-5D	**European Quality of Life – 5 Dimensions**
EURODIAB	**European Diabetes Study**
FBC	**Full Blood Count**
FFA	**Free Fatty Acids**

FIT	**Flexible Insulin Therapy**
GI	**Glycaemic Index**
GIP	**Glucose-dependent Insulintropic Peptide**
GL	**Glycaemic Load**
GLP-1	**Glucagon-like Peptide**
GMS	**General Medical Services**
GP	**General Practice/Practitioner**
HbA1c	**Glycosylated haemoglobin**
HDL	**High Density Lipoprotein**
HFS	**Hypoglycaemia Fear Scale**
IDF	**International Diabetes Federation**
IGT	**Impaired Glucose Tolerance**
Look-AHEAD	**Action for HEAlth in Diabetes**
LDL	**Low Density Lipoprotein**
NEP	**Newcastle Empowerment Programme**
NHS	**National Health Service**
NICE	**National Institute for Clinical Excellence**
NIH	**National Institute for Health**
LFT	**Liver Function Test**
OCDEM	**Oxford Centre for Diabetes, Endocrinology and Metabolism**
OxREC	**Oxford Clinical Research Ethics Committee**
PAID	**Problem Areas In Diabetes**
QA	**Quality Assurance**
QOF	**Quality and Outcomes Framework**
REACCT	**Re-education and Carbohydrate Counting Training**
RECLAIM	**Royal Infirmary of Edinburgh Carbohydrate Learning and Insulin Management**
SAE	**Serious Adverse Event**
SND	**Source Data Notes**
4-T	**Treating To Target in Type 2 Diabetes**
U&E	**Urea and Electrolytes**
UK	**United Kingdom**
UKPDS	**United Kingdom Prospective Diabetes Study**

US	**United States**
W-BQ 12	**12-item Well-Being Questionnaire**
WHO	**World Health Organisation**
WTE	**Whole Time Equivalent**

PAGE MISSING IN ORIGINAL

Acknowledgements

I would like to thank my two supervisors, Professor Jeya Henry and Professor David Matthews for their unstinting support, guidance, encouragement and good humour over the last five years.

I would also like to express my thanks to Sue Beatty, the research nurse who helped with administration of the studies, Irene Stratton for statistical support and the entire clinical research team in OCDEM for their useful comments and guidance. I am also indebted to my family for their support, particularly my husband Richard and sons Adam and Joe.

Lastly, a big thank you to all the subjects who willingly volunteered for my studies and without whom none of this would have been possible.

Dietary advice for people with diabetes: the role of carbohydrate in dietary treatment and an assessment of video education

Chapter 1

Introduction and background

1.0. Background

Diabetes Mellitus (DM) is a chronic condition characterised by high circulating blood glucose levels. It is caused by complete or relative lack of the hormone insulin. Insulin is normally produced by the pancreas; complete failure to produce insulin leads to Type 1 diabetes and insufficient insulin production leads to Type 2 diabetes. Type 1 diabetes usually presents in children and young adults, although it can be diagnosed ay any age. Type 1 diabetes is an autoimmune condition and accounts for approximately 10-20% of diabetes in the developed world. It is treated by a combination of insulin replacement by insulin injection, diet and exercise.

Type 2 diabetes is most frequently diagnosed in middle-aged or elderly people and is due to a combination of impaired insulin secretion and resistance to the action of insulin. Type 2 diabetes is associated with lifestyle factors and is more common in societies with high levels of obesity and low levels of physical activity. Approximately 80% of people with Type 2 diabetes are overweight or obese. Type 2 diabetes affects approximately 80 - 90% of those diagnosed with diabetes in the developed world and is treated by a combination of diet, exercise, oral medications and, increasingly, insulin.

1.1. Physiology of diabetes

Diabetes is a condition in which the body is unable to utilise the glucose (sugar) in the blood for energy due to complete or relative lack of insulin, insulin resistance (a condition where the body does not respond adequately to insulin) or a combination of the two. Insulin is produced and secreted in the β-cells of the pancreas and Type 1 diabetes is characterised by complete β-cell failure leading to absolute insulin deficiency. Type 2 diabetes is characterised by progressive β-cell failure and is usually associated with peripheral insulin resistance. Complete or relative lack of insulin results in raised blood glucose levels and the diagnosis of diabetes is made when the fasting blood glucose exceeds 7.0 mmol/l on two occasions (World Health Organisation 2000)

Glucose is the body's primary energy source and is provided by foods containing carbohydrate. Carbohydrate is found in starchy foods such as bread, potatoes, rice and pasta, sugary foods such as sweets, chocolate and cakes and occurs as natural sugars in milk and fruit. Excess glucose is stored in the liver and muscle as glycogen and is released into the blood stream as blood glucose levels fall.

Insulin is the hormone responsible for regulating blood glucose levels and is secreted by the β-cells of the pancreas. It is automatically released as blood glucose levels rise following carbohydrate intake. Insulin facilitates entry of glucose into muscle and adipose tissue cells to be utilised as energy. Lack of insulin leads directly to increased blood glucose levels and the symptoms of diabetes – polyuria, polydipsia, fatigue, weight loss and increased susceptibility to infections.

1.2. Long-term complications of diabetes

Exposure to high blood glucose levels in the short-term will result in the above symptoms, but one of the most important features diabetes is the association of sustained hyperglycaemia with long-term tissue damage. Diabetes is a disease with high rates of morbidity and mortality (Zimmet, Alberti et al. 2001). Diabetes is associated with macrovascular complications (cardiovascular disease, stroke, peripheral vascular disease) leading to heart attacks, haemiplegia and amputations, and with microvascular complications (neuropathy, retinopathy, nephropathy) causing foot ulcers, blindness and kidney failure (EURODIAB IDDM Complications Study Group 1994; Turner and Holman 1995). There is evidence that the risk of complications for people with Type 2 diabetes has been significantly reduced over the past thirty years and this reflects improvements in care (Nathan, Zinman et al. 2009). However, even those receiving intensive treatment still show relatively high levels of complications, with over one fifth developing proliferative retinopathy and 9% developing nephropathy and cardiovascular disease. Traditionally, Type 2 diabetes has been regarded as a disease of the elderly and of the affluent, but although diabetes remains the most common cause for adult blindness in the developed world, the morbidity of diabetes is no longer concentrated in the elderly and it affects those aged from 40 upwards. The age of diagnosis of Type 2 diabetes is now falling, and 8 to 45% of all diabetes reported among children and adolescents in the United States is now Type 2. The few data on follow-up of children suggest a high prevalence of microvascular and macrovascular complications among young adults who developed Type 2 diabetes during childhood (Fagot-Campagna, Pettitt et al. 2000). Diabetes-related mortality and morbidity now affects the working-age population and there are economic repercussions in terms of productivity and health-care costs, with a recent report

estimating that between 7-12% of NHS funding is spent on the treatment of diabetes and its complications and this amounted to £2.8 billion in 2007 (NICE 2008).

1.3. Global prevalence of diabetes

The global prevalence of diabetes has been estimated by the International Diabetes Federation (IDF) at 285 million in 2010 (6.4% of world population) and is projected to increase to 439 million (7.7%) by 2030, of whom over 75% will live in the developing world. This increase in prevalence is largely due to Type 2 diabetes and is associated with lifestyle factors. The predicted global increase in diabetes is shown below in Table 1.1.

Table 1.1. Predicted increase in diabetes prevalence 2010-2030

All diabetes	2010	2030
Total world population (billions)	7.0	8.4
Adult population (billions)	4.3	5.6
Number of people (20-79 years) with diabetes (millions)	285	439
World diabetes prevalence (%)	6.4	7.7

Source: *Diabetes Atlas* 4th edition. ©International Diabetes Federation, 2009

The rising incidence of diabetes is not confined to developing countries, and the IDF reports a wide geographic spread. Table 1.2 below shows the rising pandemic, split by world region. It illustrates that the diabetes epidemic, although well established in the developed world, will be much more prominent as an increasing problem in the developing world. For example, it is predicted that the number of people with diabetes will double in Africa and the Middle East and North Africa between 2010 and 2030.

Table 1.2. Regional estimates for diabetes (20-79 age group), 2010 and 2030

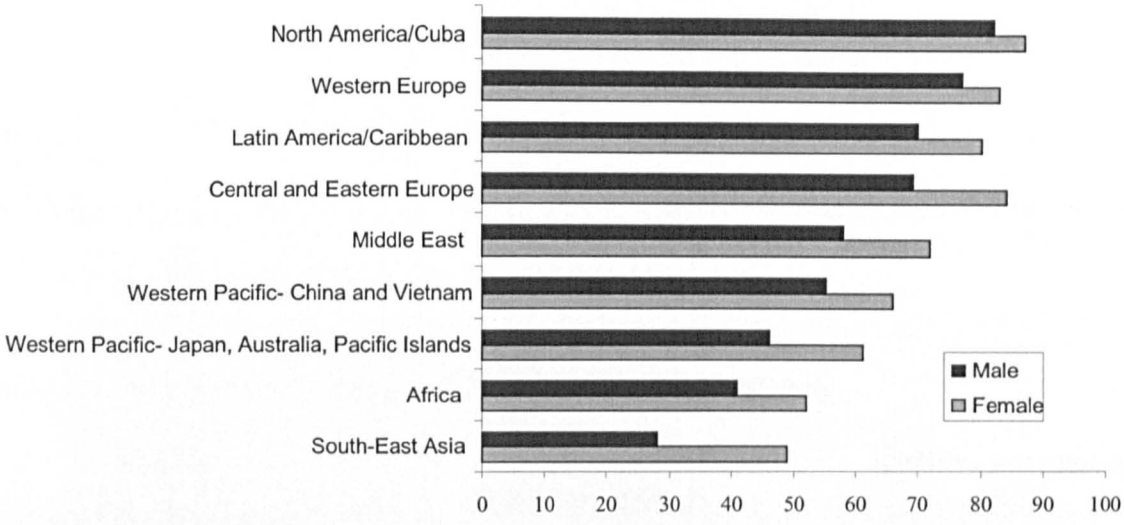
Region	2010			2030		
	Population (20-79) (millions)	No of people with DM (millions)	Diabetes prevalence (%)	Population (20-79) (millions)	No of people with DM (millions)	Diabetes prevalence (%)
NAC	320	37.4	10.2	390	53.2	12.1
MENA	344	26.6	9.3	533	51.7	10.8
SEA	838	58.7	7.6	1200	101.0	9.1
EUR	646	55.2	6.9	659	66.2	8.1
SACA	287	18.0	6.6	382	29.6	7.8
WP	1531	76.7	4.7	1772	112.8	5.7
AFR	379	12.1	3.8	653	23.9	4.7
Total	4345	284.6	6.4	5589	438.4	7.7

Source: *Diabetes Atlas* 4th edition. ©International Diabetes Federation, 2009

Key: NAC North America and Caribbean, MENA Middle East and North Africa, SEA South East Asia, EUR Europe, SACA South and Central America, WP Western Pacific, AFR Africa

There is a positive association between the developing diabetes epidemic and obesity as shown below (Figure 1.1). However it must be recognised that not all those who are obese become diabetic, nor is diabetes solely related to weight gain. Some individuals may be grossly obese for years without developing hyperglycaemia. There is also a large genetic component in type 2 diabetes, with strong familial traits and high concordance in twins. Nevertheless the diabetes epidemic has been strongly associated with the obesity epidemic, and there are specific racial groups (especially the Pima Indians) where the combination of inherited characteristics and obesity has been associated with diabetes in up to 50% of the population (Knowler, Pettitt et al. 1981).

Figure 1.1. Proportion of Type 2 diabetes (%) attributable to weight gain by region (30+ years)



Source: *Diabetes Atlas* second edition. ©International Diabetes Federation, 2003

1.4. Prevalence of type 2 diabetes in the United Kingdom

In the UK, the number of people diagnosed with diabetes is estimated to have increased from 1.4 million in 1996 to 1.8 million in 2004, equivalent to 3% of the population (DOH 2005). Diabetes registers have been introduced in General Practice over the past few years, and a random sample of practices reported a diabetes prevalence of 3.3% in 2006 (Millett, Car et al. 2007). This study also reported higher diabetes prevalence in smaller, more deprived practices (3.8%) compared with larger, more affluent areas (2.8%). More recent statistics from Diabetes UK, based upon the Quality and Outcomes Framework (QOF) introduced into general practice in the UK in 2004, suggest that the overall prevalence of diabetes in 2008 was 3.86% (Table 1.3).

These statistics do not differentiate between Type 1 and Type 2 diabetes, but as it is generally assumed that approximately 80-90% of diabetes in the developed world is accounted for by Type 2 diabetes, then it can be estimated that the prevalence of Type 2 diabetes in the UK is 3% (1.9 million people). An epidemiological approach applying prevalence rates for different populations to the 2001 census data has calculated that the prevalence of total diabetes in the UK is 4.41%, equating to 2,168,000 people, of which 2,002,000 (92.3%) have Type 2 diabetes (Forouhi, Merrick et al. 2006). These reported increases in diabetes are largely explained by an increase in the incidence of Type 2 diabetes.

Table 1.3. Prevalence of diagnosed diabetes in the UK in 2008

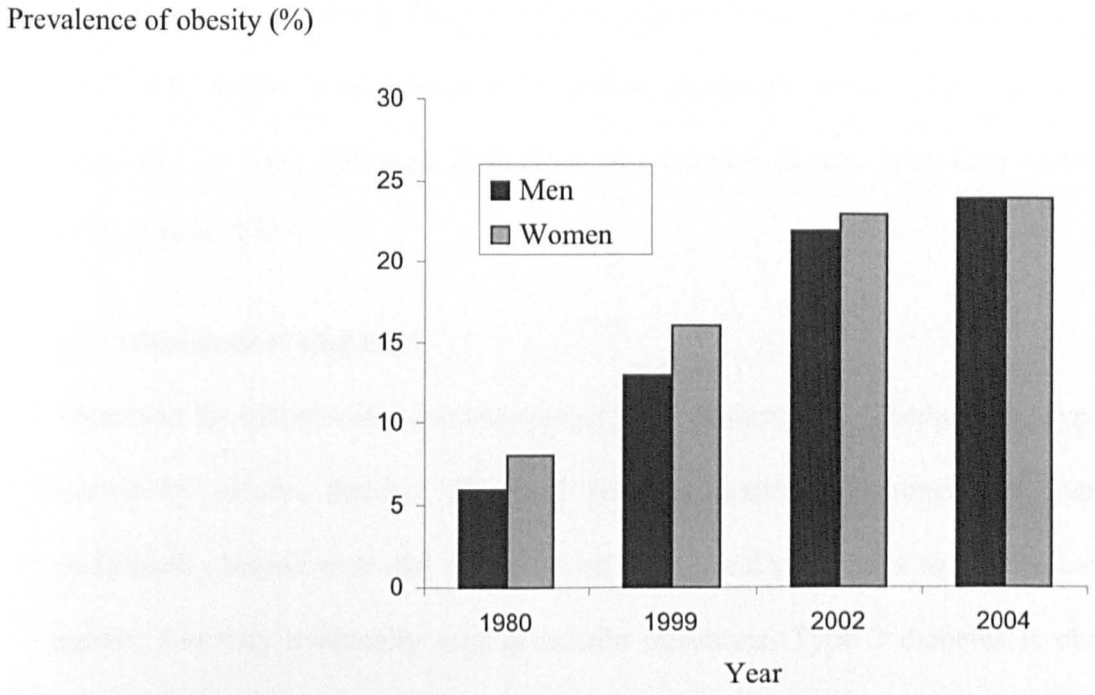
Country	Number of people with diagnosed diabetes	Diabetes prevalence (%)
England	2,088,335	3.9
Northern Ireland	60,822	3.3
Scotland	200,669	3.7
Wales	138,988	4.4
Total	2,488,814	3.86

Source: Department of Health, *Quality and Outcomes Framework (QOF) 2008*

The numbers of people with Type 2 diabetes in the UK are increasing rapidly due to several factors. Firstly, Type 2 diabetes is more common in elderly populations as shown by a mean age of diagnosis of 53 in the United Kingdom Prospective Diabetes Study (UKPDS 1998), and the British population is an ageing population. Although Type 2 diabetes is traditionally diagnosed in people over the age of 40, there is a worrying trend in increasing diagnosis in young children and adolescents. Type 2 diabetes has been diagnosed in over 100 children in the UK and is usually associated with obesity, although some authorities believe that this may be an under-diagnosis and as many as 1,400 children may have Type 2 diabetes in the UK (Lobstein and Leach 2004). Secondly, obesity is a strong risk factor for the development of

Type 2 diabetes and obesity is increasing rapidly in the UK, which has the fastest growing rate of obesity in the developed world (Rennie and Jebb 2005). In 2004, 22% of the adult population in the UK were obese (BMI >30) and 65% overweight or obese (BMI>25) (DOH 2005). Obesity prevalence in the UK is shown in Figure 1.2.

Figure 1.2. Prevalence of obesity in the UK (2004)



Source: Department of Health. *Health Survey for England, 2004*

The risk of developing Type 2 diabetes increases tenfold in people with a BMI of more than 30. There is also a strong independent risk of Type 2 diabetes associated with reduced physical activity (Bassuk and Manson 2005; Hu, Jousilahti et al. 2005).

Ethnicity is a strong predictor of diabetes; in the UK, people from ethnic minority groups have a higher crude prevalence of diabetes than White Europeans. The prevalence of diabetes in ethnic minority groups in the UK is shown in Table 1.4.

Table 1.4. Prevalence of diabetes amongst ethnic groups in the UK

Ethnic Group	Prevalence (%)
White European	4.29
Black African/Caribbean	5.69
South Asian	6.63
Other – Chinese, Japanese, Middle Eastern	2.13

Source: (Forouhi, Merrick et al. 2006)

A small study in primary care in London suggests that the higher prevalence of diabetes in UK South Asians is accompanied by poorer glycaemic control and this may be related to inequality in care, although biological and cultural factors may also contribute (Soljak, Majeed et al. 2007)

1.5. Treatment of diabetes

Treatment for diabetes is a combination of lifestyle factors and medication. Type 1 diabetes is treated by insulin therapy, diet and physical activity. Treatment of Type 2 diabetes traditionally begins with diet and exercise advice and progresses to medication, usually oral agents, and may eventually require insulin injections. Type 2 diabetes is characterised by progressive beta-cell failure and requires intensity of treatment over time. Although diet and lifestyle changes remain the first-line treatment for Type 2 diabetes, most people (80-90%) will require some form of medication to achieve long-term glycaemic control and reduce the chance of complications (Turner, Cull et al. 1999). For both Type 1 and Type 2 diabetes medication is most effective used in conjunction with dietary control and regular physical activity.

1.6. Lifestyle factors

Nutritional therapy is an integral part of effective management of diabetes and has a vital role in helping people with diabetes achieve and maintain optimal glycaemic control and reduce

the risk of long-term tissue damage (Bantle, Wylie-Rosett et al. 2008). There is evidence that intensive treatment of Type 1 diabetes, especially diet, improves outcomes (Delahanty and Halford 1993) and that dietary change, weight loss and increased physical activity are of benefit to people with Type 2 diabetes, although there are few well-designed studies investigating the most effective approach (Nield, Moore et al. 2007). The aims of dietary treatment are complementary to the aims of medical treatment and are summarised below.

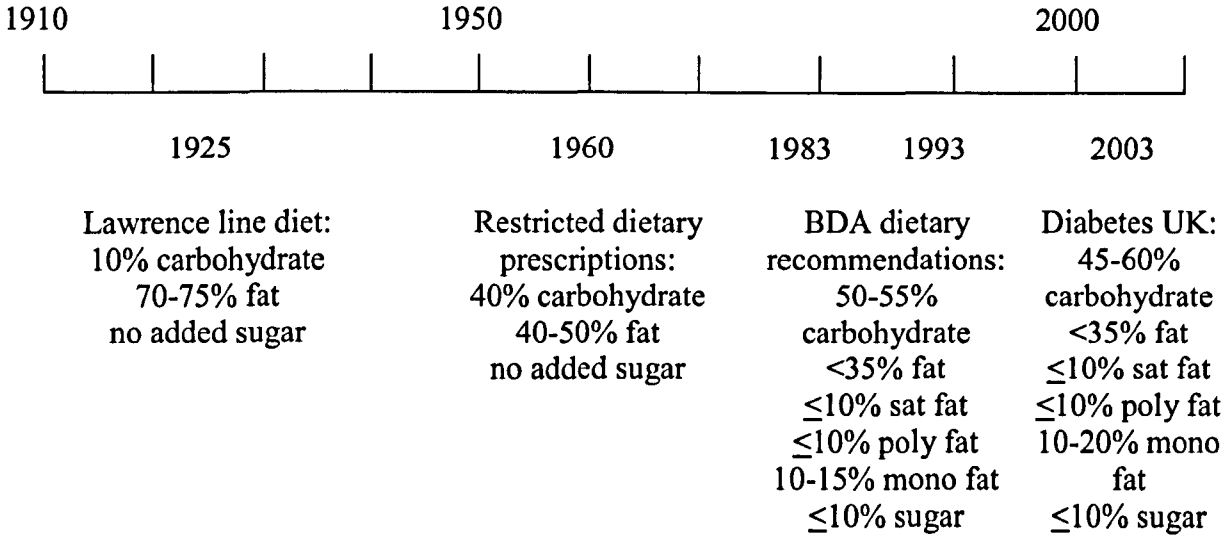
1.7. Goals of dietary treatment

- To maintain or improve health through the use of appropriate and healthy food choices
- To achieve and maintain optimal metabolic and physiological outcomes, including
    - reduction of risk for microvascular disease by achieving near normal glycaemia without undue risk of hypoglycaemia
    - reduction of risk for macrovascular disease, including management of body weight, dyslipidaemia and hypertension
    - To optimise outcomes in diabetic nephropathy and in any concominant disorder such as coeliac disease or cystic fibrosis

Source: *Manual of Dietetic Practice, 2001*

Many countries have published nutritional recommendations for diabetes (Ha and Lean 1998; Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK 2003; Bantle, Wylie-Rosett et al. 2008). Nutritional recommendations have been subject to change over the years as evidence emerged for effective dietary treatment of diabetes, and the main changes over the years are shown in Fig 1.3

Fig 1.3. Timeline of changes in nutritional recommendations for diabetes



In the UK it is common practice that the most recently published recommendations from Diabetes UK should form the basis for lifestyle advice offered to people with diabetes. The main nutritional recommendations are summarised in Table 1.5

Table 1.5. Nutritional recommendations for people with diabetes

Nutrient	Recommendation
Protein	Not <1g per kg body weight
Total fat	<35% total energy intake
Saturated fat	<10% total energy intake
n-6 polyunsaturated fat	<10% total energy intake
n-3 polyunsaturated fat	Eat oily fish 1-2 times/week
Monounsaturated fat	10-20% total energy intake) 60-70% total
Carbohydrate	40-50-% total energy intake) energy intake
Added sugar	Up to 10% total energy intake
Salt	<6g NaCl daily
Vitamins and anti-oxidants	Encourage natural sources
Fructose and sorbitol	No advantage over sucrose
Artificial sweeteners	Useful for the overweight
Herbal supplements	No evidence of benefit
Diabetic foods	None

Source: *Diabetes UK, 2003*

These recommendations from Diabetes UK show a change in emphasis from the last published nutritional recommendations (Nutrition sub-committee of the British Diabetic Association's Professional Advisory Committee 1992) and the main changes are summarised below:

1. A greater emphasis on the benefits of regular physical activity and weight management
2. More flexibility in the proportion of monounsaturated fat and carbohydrate in dietary intake
3. Sucrose no longer restricted to a specific amount
4. A recommendation to choose foods that have a low glycaemic index

1.8. Components of effective dietary interventions for diabetes

There is general agreement amongst the published dietary recommendations about the components of an effective diet for treating diabetes, although only the American Diabetes

Association attempts to grade the evidence-base for dietary recommendations (Bantle, Wylie-Rosett et al. 2008). There is also recognition that the majority of recommendations included in the latest review by Diabetes UK are concensus-based, rather than evidence-based (Lean 2003) and that as more evidence emerges, there may well be changes to current advice. At present dietary advice conforms to conventional healthy eating advice and includes the following recommendations:

Carbohydrate:

- Select foods of low glycaemic index
- Include at least five portions of fruit and vegetables daily
- Include moderate amounts of sucrose

Protein:

- Adopt a moderate protein intake

Fat:

- Reduce total and saturated fat

1.9. Recommendations for carbohydrate intake

1.9.1. Glycaemic index

There is still much controversy over the role of glycaemic index (GI) in the dietary management of diabetes. The evidence to date investigating the effect of low GI diets in people with diabetes remains controversial, with some authorities recommending this strategy as first-line treatment (Brand-Miller, Hayne et al. 2003), others recommending that low GI diets may have useful role (Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK 2003) and yet others who state that there is insufficient evidence

at present and that the amount rather than the type of carbohydrate is a better predictor of blood glucose levels (Sheard, Clark et al. 2004).

The benefits of low GI diets include improved glycaemia control, reduced cardiovascular risk and reductions in body weight (Brand-Miller, Hayne et al. 2003). Epidemiological evidence from the EUORDIAB study in 2810 people with Type 1 diabetes has shown that there is a significant association between the GI of habitual diets and A1c, with significantly lower A1c reported in those with low GI diets (Buyken, Toeller et al. 2001). There have been few randomised intervention studies investigating the effects of low GI diets in people with both Type 1 and Type 2 diabetes, but a meta-analysis of 14 trials published in 2003 has shown that low GI interventions lowered A1c by 0.43% compared with subjects adopting a high GI diet (Brand-Miller, Hayne et al. 2003). However, the authors point out that these studies were generally of short duration (only two lasted more than twelve weeks) and included small numbers. A more recent Cochrane review supports this meta-analysis and states that low GI or low glycaemic load (GL) diets reduce A1c by a weighted mean of 0.5%. However, the relevance of low GI diets in routine clinical practice has been questioned in two more recent, larger, randomised trials, both of which were conducted over longer periods of time in people with Type 2 diabetes (Ma, Olendzki et al. 2008; Wolever, Gibbs et al. 2008) and which failed to show any benefit in glycaemia when compared with other dietary strategies. One study compared low GI with high GI and low carbohydrate diets in 162 people over one year and reported no change between the three groups in glycaemic control (Wolever, Gibbs et al. 2008). The second study compared a low GI approach with standard American Diabetes Association (ADA) medical nutrition therapy and reported significant reductions in A1c and lipid levels in both groups, but no differences between the two groups (Ma, Olendzki et al.

2008). There was, however, a reduction in the use of glucose-lowering medication in the low GI group compared with the ADA group.

Studies of the effects of low GI diets in people with diabetes have tended to concentrate on glycaemic control, although the benefits of low GI diets in people without diabetes include increased weight loss, improvements in cardiovascular risk (Thomas, Elliott et al. 2007) and improved physical endurance (Wu and Williams 2006). This evidence that low GI diets are more effective for weight loss in people without diabetes has not been replicated for people with diabetes and there is no evidence that low GI diets are more effective for weight loss than other dietary interventions in people with diabetes. The two more (Ma, Olendzki et al. 2008; Wolever, Gibbs et al. 2008) recent studies conducted in people with Type 2 diabetes reported no significant weight change, despite the fact that obese subjects were recruited to both studies.

There is some evidence that low GI diets reduces cardiovascular risk in people with diabetes with evidence for reductions in both total and LDL cholesterol (Rizkalla, Taghrid et al. 2004), reductions in C-reactive protein (Wolever, Gibbs et al. 2008), and epidemiological evidence that low GI diets are associated with higher HDL cholesterol levels in people with Type 1 diabetes (Buyken, Toeller et al. 2001).

One of the main criticisms for low GI diets as a strategy for blood glucose control is that there are other dietary approaches that are more effective for glycaemic control. The amount of carbohydrate, rather than the type, is the best predictor of postprandial glycaemic response (Sheard, Clark et al. 2004). There is evidence that dietary approaches addressing the amount of carbohydrate (carbohydrate counting) and insulin adjustment can significantly reduce A1c

in people with Type 1 diabetes (DAFNE study group 2002). In addition, total energy intake has a significant effect on glycaemia. The United Kingdom Prospective Diabetes Study (UKPDS) showed that a decrease in energy intake significantly reduced A1c in people with Type 2 diabetes (UKPDS 1990).

It has also been suggested that low GI diets are difficult to understand and implement and that this may be counter-productive for those adopting this dietary strategy. It is challenging for those well-versed in healthy eating to discover that wholemeal bread and baked potatoes are high GI foods and should be reduced if adopting a low GI diet. However, a recent study suggests that these criticisms are not substantiated in practice and that implementing a low GI diet is no more challenging than adopting a more conventional approach (Ma, Olendzki et al. 2008)

In conclusion, there is evidence that reductions in glycaemic load, which may be achieved by adopting a low GI diet, improve blood glucose control and cardiovascular risk in people with diabetes and this strategy is included as a recommendation in most published guidelines.

1.9.2. Fruit and vegetables

Dietary recommendations about fruit and vegetable intake refer to the general health effects of eating fruit and vegetables and are not directly related to glycaemic control. Intakes of fruit and vegetables show positive associations with a reduction in chronic disease including cardiovascular disease and some cancers in people with diabetes (Nothlings, Schulze et al. 2008). In people without diabetes, epidemiological studies have shown that fruit and vegetable intakes are inversely associated with some cancers (Lunet, Valbuena et al. 2007), coronary heart disease (He, Nowson et al. 2007) and stroke (He, Nowson et al. 2006). The

role of fruit and vegetables in the prevention and treatment of diabetes is unclear. Studies have shown that there is no evidence that fruit and vegetables intake have a protective role in the development of diabetes (Liu, Serdula et al. 2004; Hamer and Chida 2007), although there is some evidence for green leafy vegetables (Bazzano, Li et al. 2008). Fruit contains a natural sugar, fructose, and this raises blood glucose levels after eating and although there is no evidence that fruit intake has any effect upon diabetes, there is limited evidence for fruit juice consumption (Bazzano, Li et al. 2008).

1.9.3. Sucrose

There is less importance attached to the role of sucrose (table sugar) in the diet of people with diabetes as it has long been known that sugar does not increase blood glucose levels more than starchy foods containing the same amount of carbohydrate. There is still widespread belief amongst people with diabetes that 'simple sugars' like sucrose are digested and absorbed quickly and should be avoided and that 'complex carbohydrates' like bread and potatoes are digested more slowly and should form the basis of the diet. There is now robust research supporting the evidence that the total amount of carbohydrate eaten will determine post-prandial blood glucose levels regardless of whether it is derived from sucrose or starch (Sheard, Clark et al. 2004).

Traditionally table sugar (sucrose) has been restricted for people with diabetes and the original nutritional recommendations from the British Diabetic Association (now known as Diabetes UK) in 1992 stated that no more than 25g sucrose should be consumed daily. Research supports the recommendation that sucrose can provide up to 10% total energy intake (Slama, Haardt et al. 1984) without compromising glycaemic control. For the average adult consuming 2000 kcal/day this would translate as approximately 50g of sucrose each day.

There has been some recent research questioning sucrose intake for people with diabetes, particularly concerning the role of the high fructose corn syrup used in the manufacture of soft drinks in the development of obesity and diabetes, with some authorities identifying a causal link from epidemiological evidence and recommending a reduction in the intake of soft drinks (Schulze, Manson et al. 2004; Malik, Schulze et al. 2006). A recent review and meta-analysis supports reduction in soft drink intake (Vartanian, Schwartz et al. 2007) although in the absence of any randomised controlled trials in people with diabetes, it is difficult to draw firm conclusions.

1.10. Recommendations for protein intake

The role of protein in the management of diabetes remains uncertain, and the effect of protein on blood glucose levels, renal function and body weight remain unclear. A study in people with Type 2 diabetes has demonstrated that dietary protein appears to promote insulin secretion and reduces post-prandial blood glucose levels (Gannon, Nuttall et al. 2003)

There are further questions about protein's effects on renal function and the development of nephropathy in people with diabetes. There is little evidence of a causal relationship between protein intake and the development of renal disease and the majority of nutritional recommendations state that usual protein intakes (15-20% of total energy intake) are advised for people with diabetes (Bantle, Wylie-Rosett et al. 2008). For those with established renal disease, a Cochrane review has shown that limiting protein intake reduces the rate of progression of renal disease, but that this is not statistically significant (Robertson, Waugh et al. 2007).

The effects of adopting a high protein (providing >20% total energy intake), weight reducing diet for people with Type 2 diabetes have been investigated in some small studies. These short-term studies suggest that high protein diets can improve blood glucose levels and promote weight loss (Gannon, Nuttall et al. 2003; Gannon and Nuttall 2004), but as these diets are also low in carbohydrate it is difficult to evaluate the different effects of altering the macronutrient content of the diet. The American Diabetes Association continues to recommend that high protein diets should not be promoted for weight loss as the long-term effects of high protein intakes on renal function are unknown.

1.11. Recommendations for fat intake

All authorities recommend a reduction in both total and saturated fat for people with diabetes, and this advice is associated with the risk of cardiovascular disease. The risk of cardiovascular events in people with diabetes is 2-4 times that of the non-diabetic population and the majority of dietary recommendations for people with diabetes are the same as those with pre-existing cardiovascular disease as they appear to have similar risk (Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK 2003; Bantle, Wylie-Rosett et al. 2008).

1.12. The effectiveness of lifestyle modifications in treating Type 2 diabetes

1.12.1. Dietary modification

Dietary modification has been shown to be effective in both Type 1 and Type 2 diabetes, although the emphasis varies between the two. People with Type 1 diabetes are encouraged to assess carbohydrate intake at each meal and snack, and to inject insulin to match the amount of carbohydrate eaten. This strategy has been shown to improve glycaemic control in two large randomised controlled trials (DCCT study group 1993; DAFNE study group 2002).

Dietary intervention, including weight loss, at diagnosis of Type 2 diabetes has been shown to be effective in the short-term. The UKPDS reported that in 5,000 people newly diagnosed with Type 2 diabetes, dietary modification reduced fasting plasma glucose from 11.5 to 8.3 mmol/l over three months and this corresponded to a drop in A1c (a longer term measure of levels of glycaemia) from 9.1 to 7.0% (Manley, Stratton et al. 2000) This reduction in blood glucose level was associated with a reduction in weight of 3kg. However, the UKPDS went on to show that after 10 years of treatment in the group randomised to conventional treatment (diet alone unless clinical indication that more intensive treatment was required) only 8% were able to achieve the target value of fasting plasma glucose <7.8 mmol/l (Turner, Cull et al. 1999). This result reflects the nature of Type 2 diabetes, characterised by beta cell failure over time, as well as the failure of dietary strategies alone to control plasma glucose levels effectively. There remains some uncertainty about the effectiveness of dietary modification over the long-term and a recent Cochrane review (Nield, Moore et al. 2007) concluded that there are no high quality data in the efficacy of the dietary treatment of Type 2 diabetes. This review states that these conclusions reflect a lack of high quality studies that examine a range of interventions rather than that dietary interventions are ineffective in the treatment of Type 2 diabetes. At present, it is still not clear which dietary strategies are most effective in the treatment of Type 2 diabetes, but the lack of effectiveness of therapy underpins the idea that there may well be a more effective way of delivering lifestyle advice which can have positive outcomes.

A recently published study from the United States, the Look-AHEAD (Action for HEAlth in Diabetes) study (Pi-Sunyer, Blackburn et al. 2007) set out to test the efficacy of lifestyle interventions in people with established Type 2 diabetes and showed that a structured

education programme can improve biomedical outcomes significantly over one year. This was a large, multi-centred randomised controlled trial with over 5,000 patients taking part. The patients were randomised to either intensive diabetes education and support or usual care. All medical diabetes care remained the responsibility of the patients' diabetes physician, regardless of study allocation. At the end of 1 year, those who received the lifestyle intervention showed a significant reduction in HbA1c of 0.8% (from 7.2% to 6.6%) compared with no change in the control group receiving usual care. This improvement in glycaemic control was associated with a reduction in weight of 8kg, with no change in weight in the control group. There were also significant improvements in cardiovascular risk factors with lower blood pressure, increases in physical fitness and improvements in lipid profile in the intervention group. The Look-AHEAD study utilised experience from the Diabetes Intervention Programme (Knowler, Barrett-Connor et al. 2002) that had identified lifestyle strategies that resulted in a 60% reduction in progression to type 2 diabetes in individuals with impaired glucose tolerance (IGT) and who were at high risk of diabetes. The strategies employed in look-AHEAD study included dietary and physical activity strategies, and a key component was tailoring the lifestyle programme to the individual.

1.12.2. Components of effective dietary intervention

Evidence suggests that the most effective component of dietary intervention for people with Type 1 diabetes is strategies to address carbohydrate assessment and insulin adjustment. There is little evidence for the relative importance of other macronutrients (protein and fat) in the diet of people with Type 1 diabetes.

For people with Type 2 diabetes, the limited evidence available suggests that strategies employed to maximise the effectiveness of dietary treatment include energy restriction

resulting in weight loss and increased physical activity. The relative importance of and their role in glycaemic control and weight loss has yet to be fully investigated. There has been very little work done on assessing the relative importance of the separate contributions of diet and physical activity to blood glucose control and management of Type 2 diabetes.

Effective dietary strategies employed in Look-AHEAD emphasised energy restriction and this was achieved by utilising three approaches comprising individual portion-controlled meal plans, provision of liquid replacement meals and frozen prepared meals and a reduction in fat intake aiming to provide <30% energy as total fat and <10% as saturated fat. In addition, recent evidence has supported the role of moderate protein intakes in weight loss (Westerterp-Plantenga, Nieuwenhuizen et al. 2009) and it was recommended that a minimum of 15% of total energy intake was derived from protein.

1.12.3. Physical activity

Epidemiological studies have demonstrated that physically active individuals are less likely to develop Type 2 diabetes (Manson, Rimm et al. 1991; Manson, Nathan et al. 1992) and that sedentary behaviour is positively associated with Type 2 diabetes (Hu, Leitzmann et al. 2001; Hu, Li et al. 2003). It has also been established that physical activity can help prevent the development of Type 2 diabetes when used in combination with diet and weight loss (Tuomilehto, Lindstrom et al. 2001; Knowler, Barrett-Connor et al. 2002). The role of physical activity in the treatment of diabetes is less certain, with contradictory evidence suggesting that there may be little benefit in terms of glycaemic control for those with Type 1 diabetes, although there is a reduction in cardiovascular risk (Kavookjian, Elswick et al. 2007). Evidence of benefit for those with Type 2 diabetes suggests that glycaemic control improves in the physically active with a weighted mean reduction of 0.66% in A1c (Boule,

Haddad et al. 2001). In addition, cardiovascular risks are reduced, although there is no evidence of weight loss with increased physical activity (Boule, Kenny et al. 2003).

Physical activity strategies employed in the successful Look-AHEAD study included home-based exercise programme and a gradual progression to 175 minutes of moderate activity each week

1.13. Medical Treatment of Diabetes

People diagnosed with Type 1 diabetes are treated by a combination of insulin replacement via injection or pump therapy and diet. Diet and lifestyle are traditionally seen as the first-line treatment for Type 2 diabetes, but the majority of people (80-90%) will require some form of medication to minimise the risk of long-term tissue damage or complications.

1.13.1. Oral medications

There are five different types of oral medication available to treat Type 2 diabetes and their generic name, brand names, action, dosage and side effects are summarised in Table 1.6. Metformin (the only available biguanide) is usually given as the first-line agent and is the most commonly prescribed drug for diabetes. It has been used in the UK since the 1970s. It is derived from guanidine, an active ingredient found in French lilac and its main effect is to increase insulin sensitivity, it does not cause weight gain and there is limited evidence that it may be associated with a reduced risk of cardiovascular events (NICE 2008). Around 10% of patients do not respond to metformin and approximately 5-10% patients a year on metformin will need another agent in addition. If maximum tolerated doses of metformin fail to achieve target glucose levels, a second agent is commonly added to metformin and this is either a sulphoylurea agent, a thiazolidinedione or a metiglinide.

Table 1.6. Oral hypoglycaemic agents for treatment of diabetes

Drug – generic name	Brand name	Action	Dosage	Side-effects
Biguanides:				
• Metformin	Glucophage	Increases insulin sensitivity Decreases liver glucose production	500-2500mg daily with food	Nausea, indigestion, diarrhoea
• Extended release metformin	Glucophage SR		500-2500mg once daily	
Sulphonylureas:				
• Gliclazide	Diamicron	Stimulate insulin production by the pancreas	40-160mg twice daily	Hypoglycaemia, weight gain
• Extended release gliclazide	Diamicron MR		30-120mg once daily	
• Glibenclamide	Daonil, Euglcon		5-15mg twice daily	
• Glipizide	Glibenese, Minodiab		2.5-20mg twice daily	
• Glimeperide	Amaryl		1-4mg once daily	
Thiazolidinediones:				
• Rosiglitazone	Avandia	Enhance the action of insulin	4-8mg once daily	Weight gain, fluid retention
• Pioglitazone	Actos		15-45mg once daily	
Metiglinides:				
• Repaglanide	Prandin	Stimulate insulin secretion post-prandially	2-4mg with meals	Hypoglycaemia, weight gain
• Nateglinide	Starlix		60-180mg with meals	
Alpha-glucosidase inhibitor				
• Acarbose	Glucobay	Reduces speed of absorption of carbohydrate from the gut	50-200mg with meals	Wind, diarrhoea, bloating

Sulphonylureas were discovered by chance during the Second World War when it was observed that soldiers treated with sulphonamide antibiotics experienced a reduction in blood glucose levels. These drugs act directly on the pancreas and stimulate insulin production. Thiazoladinediones (also known as glitazones) have been used only relatively recently since 1997. They are taken once daily, starting with a low dose, and take up to three months to have their maximum effect. Their action is to reduce insulin resistance and they produce a lowering of blood glucose levels comparable to metformin or sulphonylurea. Glitazones tend to be given as second or third-line therapy, partly because of the 10-12 weeks it takes for them to have effect. Generally a person is offered a combination of metformin and a sulphonylurea before a glitazone is added in. Glitazones are also often prescribed if a person cannot tolerate metformin.

Meglitinides are short-acting agents that are used to control post-prandial rises in glucose. They are taken before main meals and stimulate insulin production for that meal. Essentially, they may be viewed as a short-acting sulphonylurea. The fifth class of oral agent is acarbose, an alpha-glucosidase inhibitor, which relies upon reducing the rate of carbohydrate digestion and controlling post-prandial blood glucose rises. This agent has unpleasant side-effects of wind, abdominal pain, bloating and diarrhoea. As its effect is moderate compared to the other agents available, it is rarely prescribed.

1.13.2. Insulin

Insulin therapy is essential for those with Type 1 diabetes and is a necessary adjunct to therapy in people with Type 2 diabetes who are failing to achieve target blood glucose levels although they are taking maximum doses of oral hypoglycaemic agents. There are many different insulins available and selection of the appropriate type, dose and regimen depends

upon the lifestyle of each individual. There are many different forms of insulin available in the UK but until the 1980s insulin was only available by extraction and purification from the pancreas of cattle and pigs. This animal insulin is still in use but has been largely replaced by human insulin that is engineered genetically in laboratories. Newer analogue insulin has been introduced over the past five years in an attempt to replicate the action of naturally produced insulin in the body. Table 1.7 shows the main types of insulin in use in the UK today.

Table 1.7. Insulin treatment for diabetes

Type of insulin	Examples of insulin	Onset of action	Peak action	Length of action
Rapid-acting analogue	NovoRapid (Aspart), Humalog (Lispro), Apidra (Glulisine)	5-10 minutes	90 minutes	3 hours
Short-acting	Actrapid, Humulin S, Insuman Rapid, Hypurin Bovine Neutral, Hypurin Porcine Neutral	30 minutes	2 hours	4-8 hours
Medium and long-acting	Insulatard, Humulin I, Insuman Basal, Hypurin Bovine Isophane, Hypurin Porcine Isophane	90 minutes	4 - 12 hours	12 – 24 hours
Long-acting analogue	Lantus (Glargine), Levemir (Detemir)	-	-	18 -24 hours
Mixed insulin	Mixtard 10, 20, 30 and 40, Humulin M3, Insuman Comb 15,25 and 50, Hypurin Porcine 30/70 Mix	30 minutes	2 – 8 hours	16 – 20 hours
Analogue mixed insulin	Humalog Mix 25 and 50, NovoMix 30	5-10 minutes	90 minutes	16 – 20 hours

1.13.3. Incretin agonists and inhibitors

Two new agents for treatment of type 2 diabetes have recently been introduced and they are known as incretin agonists and DPP-4 inhibitors. Incretins are gut hormones that are secreted in response to ingestion of nutrients and the most widely researched are human glucagon-like peptide (GLP-1) and glucose-dependent insulinotropic peptide (GIP). Incretins have four main physiological actions including:

1. Stimulation of glucose-dependant insulin secretion from the pancreas
2. Reduction in glucagon secretion
3. Inhibition of gastric emptying, thus promoting satiety
4. Weight reduction

Two incretin agents are available for treatment of type 2 diabetes (exenatide and liraglutide) and these are both synthetic hormones that act like GLP-1. They are injected therapies and are often introduced as an alternative to insulin therapy when people with Type 2 diabetes are unable to achieve glycaemic control on oral agents alone. One advantage of these therapies is that they do not induce hypoglycaemia as their action is glucose-dependent. There are two documented side-effects; nausea associated with their effects upon gastric emptying and pancreatitis.

Incretins are rapidly degraded in the body by the action of an enzyme known as dipeptidyl peptidase 4 (DPP-4). The introduction of DPP-4 inhibitors, which act to increase concentrations of endogonous, naturally occurring incretin hormones by preventing rapid degredation by DPP-4, is another strategy that has been developed to help control blood glucose levels in people with diabetes. Two oral DPP-4 inhibitors have been developed to date (sitagliptin and vildagliptin) and are used as an add-on therapy to metformin or

glitazones when there is inadequate glycaemic control. DPP-4 inhibitors have a weaker action than the injected incretin agonists and do not promote weight loss. The most frequently documented side-effect is that of nausea.

Table 1.8 shows the relative effectiveness of all the medical treatments available for Type 2 diabetes and their effects upon A1c and body weight.

Table 1.8. Effects of medication for diabetes on A1c and weight

Medication	Change in A1c (%)	Change in body weight (kg)
Oral medication:		
Metformin	-1.14[1]	0.3[1]
Sulphonylureas	-1.52[1]	3.8[1]
Thiazolodinediones:		
Rosiglitazone	-1.16[1]	3.1[1]
Pioglitazone	-0.97[1]	3.0[1]
Metiglinides:		
Repaglinide	-1.32[1]	NR[1]
Nateglinide	-0.54[1]	NR[1]
Alpha-glucosidase inhibitors	-0.77[1]	-0.1[1]
Insulin	-0.8 to -1.4[2]	1.9 to 5.7[2]
Incretins:		
GLP-1 agonists	-0.97[3]	-2.37[3]
DPP-4 inhibitors	-0.74[3]	0.5[3]

1 (Bolen, Feldman et al. 2007)
2 (Holman, Thorne et al. 2007)
3 (Amori, Lau et al. 2007)
NR: not reported

1.14. Effectiveness of medical treatment of diabetes

1.14.1. Oral agents

The most robust evidence for the relative effectiveness of the two most commonly used oral agents was supplied by the UKPDS in 1998 (UKPDS 1998). This study evaluated long-term follow-up of people with Type 2 diabetes who were randomly allocated to sulphoylureas or metformin and showed a significant reduction in A1c over ten years. There were no differences between the two groups for any outcomes, with the exception of body weight (a mean gain of 3 kg in the sulphonylurea group vs no change in the metformin group), hypoglyacaemia and a reduction in cardiovascular events in the metformin group (UKPDS 1998).

1.14.2. Insulin therapy

Insulin is essential treatment for Type 1 diabetes, and evidence suggests that good glycaemic control is associated with a reduction in long-term tissue damage (DCCT study group 1993). In the DCCT study, those in the intervention group achieved better glycaemic control than those in the control group by increasing the number of injections to at least four each day (one injection of long-acting insulin and three injections of short-acting insulin with meals; the basal prandial regimen) or by using an insulin pump.

There is very little evidence for the most effective method of insulin therapy in treating Type 2 diabetes. Insulin was included as a treatment option in the UKPDS and showed no greater benefit in terms of glycaemic control compared with groups allocated oral therapy (UKPDS 1998). Three insulin regimens are commonly used when initiating therapy in people with Type 2 diabetes. Most commonly, one injection of long-acting insulin (usually at night)is introduced together with maintenance of oral therapy. Alternatively, two injections (usually

before breakfast and before the evening meal) of biphasic (mixed) insulin may be initiated. Any sulphonylurea agent would usually be discontinued although metformin therapy is usually maintained. The third and most complex regimen is the introduction of a basal bolus (prandial) regimen of one injection of medium or long-acting insulin at night, and three injections before main meals of rapid or short-acting insulin. Any sulphonylurea agent would usually be discontinued. A recent trial, the 4-T (Treating To Target in Type 2 diabetes) compared three insulin regimens; one injection of long-acting insulin, two injections of biphasic insulin and three injections of rapid-acting insulin before meals and showed that all three regimens significantly reduced A1c from baseline over one year. The regimen of one injection per day showed less of a reduction in A1c compared to the other groups, but there was also less hypoglycaemia and weight gain in this group (Holman, Thorne et al. 2007).

1.15. Treating Type 2 diabetes in Primary Care

Traditionally, diabetes education has been largely provided by secondary care with the majority of diabetes specialist dietitians and nurses based within diabetes centres in hospitals, but the increasing prevalence of Type 2 diabetes and the recent introduction in 2003 of the General Medical Services (GMS) contracts in primary care have resulted in the growing responsibility for first-line treatment of Type 2 diabetes being assumed by primary care (Fitzsimons, Wilton et al. 2002). This movement from secondary to primary care has been supported by the introduction of the Quality and Outcomes Framework (QOF) as part of the new GMS contracts in 2004.

1.16. The Quality and Outcomes Framework

The QOF is the annual reward and incentive programme detailing General Practice (GP) results. It was introduced in 2004 and participation is purely voluntary, although in 2006 over

90% of general practices within the UK took part in the scheme (Khunti, Gadsby et al. 2007). QOF awards GP surgeries achievement points, which translate into financial incentives, for managing many chronic disease e.g. cardiovascular disease, asthma and diabetes, for how well the practice is organised, for the patient's experience and for the amount of additional services offered such as child and maternity services. In April 2006, a revised QOF was introduced and was divided into four domains including clinical, organisational, patient experience and additional services.

The total allocation of points is 1,000, and the majority of points (655) is allocated to the clinical domain. The clinical domain consists of 80 indicators across 19 clinical areas and diabetes is allocated the highest number of indicators (18) compared to the next highest (cardiovascular disease with 10 indicators). Importantly, the indicators for diabetes include outcomes together with maintaining current records. For example, practices are awarded points for recording A1c over the past 15 months, and more points are allocated if a certain percentage of the population have A1c results below the two cut-off points of 10% and 7.4%.

1.17. Provision of dietetic support in primary care

'Practical dietary assessment and education play an essential part in the management of the diabetic condition.' So begins a report published by the British Diabetic Association (BDA), now known as Diabetes UK, in 1987 (Nutrition sub-committee of the British Diabetic Association 1987). This report, which investigated the provision of dietetic advice for people with diabetes and took place during 1984, first highlighted the inequality in the service provision in the UK. It reported that 20% of all hospital diabetes clinics were being run without the presence of a dietitian. In nearly 30% of all health districts, general practitioners

had no access to dietitians and freely admitted that they rarely discussed their patient's diet, whether a dietitian was available or not.

This report showed that there was great variability in provision of dietitians involved in diabetes education in different parts of the UK, ranging from over 3 dietetic whole-time equivalents (WTE) per one hundred thousand catchment population in North West Thames to just over 1 WTE in the West Midlands. Furthermore, four districts (South West Herts, Hereford, Bath and Oxford) agreed to log the actual amount of time spent conducting one-to-one interviews with people with diabetes over a 2-week period in February 1985. This showed that there were further inequalities in time spent with people with diabetes, ranging from 20.2 hours per one hundred thousand catchment population in South West Herts to 5.2 hours in Oxford. This is the first documented evidence of the under-provision of dietetic time to diabetes in Oxford. This report went on to recommend the minimum service that should be offered in diabetes care and stated that there should be a minimum of 15 hours of direct dietietic time per 100 000 population per week. Only 17% of the areas surveyed in this study met this standard. A further recommendation was made that there should be 1.5 WTE dietitians for every 250 000 population. It also recommended that with the increasing trend to treat people with diabetes in the community that there should be 'early action' to address the lack of care in the community.

Publication of this report was followed by a second survey in 1996 and which was published in 2000 (Nelson, Lean et al. 2000). The marked difference in regional provision of services was still noted, with ranges of 2.0 to 27.6 hours per 100 000 thousand population per week, and a median of 10.7 h/100000/week and only 37% of the health areas (compared with 17% in 1987) reported providing the minimum levels of care recommended in the 1987 report.

Publication of this report was followed by a document recommending core staffing levels for an average district specialist diabetes care team and included recommendations for dietetic services (Diabetes UK 2000). The main recommendations for dietetic services included the following:

- 1.5 state-registered dietitians with a special interest in diabetes per 250 000 population
- 15 hours direct contact time per 100 000 population per week
- dietetic consultation within 4 weeks of diagnosis
- non-crisis review annually

Again, provision of services in Oxford did not compare well to the rest of the country and it was decided to audit the dietetic service to people with diabetes during 1999-2000 within Oxfordshire and compare this to the published recommendations. Over 2,500 people with diabetes were seen by dietitians in Oxfordshire during this period, the majority being seen in primary care. The population of Oxfordshire at this time was approximately 600 000 and the Table 1.9 shows the comparison between service provision with the recommendations. The service in Oxfordshire is fragmented and divided between primary and secondary care. Secondary care is provided to out-patients by a specialist diabetes dietitian at the Oxford Centre for Diabetes, Metabolism and Endocrinology (OCDEM) at the Churchill Hospital and by general dietitians at the Horton Hospital in Banbury. All diabetic in-patients at all hospitals within Oxfordshire are seen by general dietitians or by those with specialities other than diabetes e.g. renal, gastroenterology. In addition children with diabetes are seen by a part-time paediatric dietitian who is based at the John Radcliffe hospital. Provision in primary care is provided by a team of community dietitians who run clinics in various parts of the county.

Table 1.9. Comparison between care recommendations from Diabetes UK and service provision in Oxfordshire

Recommendation	Oxfordshire
1.5 WTE per 250 000 population	1.5 WTE per 600 000 population
90 hours direct contact time per week	40 hours direct contact time per week
Consultation within 4 weeks of diagnosis	Only applicable to newly diagnosed type 1 patients and paediatric services
Annual review	Available at Horton hospital, Banbury and to paediatric patients

A further report published in 2002 (Winocour, Mearing et al. 2002) confirmed that little had changed to improve dietetic services to people with diabetes, with only 3% able to provide the recommended 22 hours per week and annual reviews available in only 15%.

The conclusion from this body of evidence is that provision of dietetic advice to people with diabetes within Oxfordshire has been under-resourced and fails to meet national targets. Issues around the funding of dietetic services mean that it is unlikely that there will be any increase in diabetes dietitians over the next few years and it may be more efficient to utilise the skills of the specialist secondary care team to provide support to primary care in the delivery of lifestyle education to people with diabetes.

1.18. Delivering diabetes education in primary care

The emphasis of most published reports is on the provision of dietetic care from hospital based diabetes centres, whereas the majority of care for those with Type 2 diabetes now takes place in primary care. General practitioners (GPs) are increasingly responsible for provision of care for 75% of their patients with diabetes (Pierce, Agarwal et al. 2000) and both the increasing incidence and prevalence of diabetes and the new GMS contracts for primary care

suggest that the burden of care will fall increasingly on the primary care team. A recent report highlighted the inadequate provision of dietetic services in primary care and made the statement that 'there is an urgent need to develop effective techniques for lifestyle alterations that can be used in the primary care setting' (Winocour, Mearing et al. 2002). Diabetes care can be complex and demanding for both patients and primary healthcare workers alike (Agarwal, Pierce et al. 2002). However, GPs are keen to provide diabetes care and their patients prefer the primary health care setting. A King's Fund report examining patients' perspectives on the way ahead for self-management for long-term conditions identified that patients feel that an on-going relationship with their primary health-care team is fundamental to enhancing self-management (Corben and Rosen 2005). There is little evidence assessing the relative effectiveness of diabetes education delivered in primary rather than secondary care, although two large studies demonstrating positive outcomes of education have taken place in community settings (Davis, Heller et al 2008, Deakin, Cade et al 2006) and a recent review reports that group education is especially effective in community gathering places (Loveman, Frampton et al 2008).

The challenge is to provide education to patients that can be delivered by the primary health care team, that is cost-effective and that is shown to have a positive effect upon the health of people with diabetes. GPs are aware of their duty of care to provide nutrition information to people with diabetes, a recent study in Australia showing that 86% agreed that nutrition advice was necessary for people with diabetes and 79% provided some nutrition counselling (Nicholas, Pond et al. 2005). A recently published survey of primary care in the Trent region showed that just under one third (31.3%) of patients with Type 2 diabetes in primary care are being managed by diet alone and it is important that these patients receive the best possible

lifestyle advice (Hippisley-Cox and Pringle 2004). There does seem to be a relationship between doctors' nutrition counselling skills and the percentage of patients receiving dietary advice and this will exert an effect upon patients (Agarwal, Pierce et al. 2002). Overall, there seems to be some recognition amongst GPs that nutrition counselling is important for people with diabetes, but that GPs do not generally have the necessary resources or skills to effect this. Family physicians are keen to implement lifestyle interventions for Type 2 diabetes, but are hampered by barriers and use of ineffective strategies. It is likely, under present economic restraints within the health service in the UK that there will never be a sufficient number of dietitians to deliver dietary advice to people with Type 2 diabetes in the primary care setting and that other methods of delivering this information should be designed and evaluated.

1.19. Delivering diabetes education in secondary care

As GPs have assumed more responsibility for care of people with Type 2 diabetes, there has been recognition that the delivery of education for those with Type 1 diabetes is more specialist and should include a structured approach (Diabetes UK 2005). In the UK, many centres have adopted structured group education for people with Type 1 diabetes and run education programmes that address carbohydrate counting and insulin adjustment. A randomised controlled trial has demonstrated that this approach improves glycaemic control and quality of life, but this has not been shown clearly in clinical practice, despite many centres adopting structured group education for people with Type 1 diabetes.

1.20. Summary

Although there is some evidence for the effectiveness of lifestyle advice in people with diabetes, there remains some uncertainty about both the content and the delivery of education programmes. One of the most contentious issues concerns the type and amount of

carbohydrate that should be recommended to people with diabetes. Recent recommendations state that there should be more flexibility in the amount of carbohydrate in the diet of people with diabetes, and that this may improve glycaemic control and weight loss. In addition, new approaches to delivering lifestyle education may improve outcomes for people with diabetes. Traditionally, consultations take place on an individual basis, but the combination of a shortage of dietitians and the increasing prevalence of Type 2 diabetes has meant that many people receive little, if any, advice at diagnosis. People with Type 1 diabetes have long been subject to rigid dietary prescriptions and may benefit from more flexibility. Novel, innovative methods to deliver lifestyle advice are urgently needed and should be subject to rigorous evaluation before application to clinical practice.

1.21. Hypothesis

The over-arching hypothesis for this thesis is that the introduction of a more flexible approach to carbohydrate intake and utilising more innovative methods of delivering lifestyle education to people with diabetes will improve knowledge, glycaemic control and cardiovascular risk.

Study 1 is designed to investigate the role of carbohydrate in weight loss and glycaemic control in people with Type 2 diabetes. If reducing carbohydrate intake is related to weight loss, then overweight people with Type 2 diabetes who adopt a low carbohydrate diet will show significant weight reduction compared to those who adopt a relatively high carbohydrate diet.

Study 2 is designed to examine the effects of a novel method if delivering education by means of video for people newly diagnosed with Type 2 diabetes. If video education is effective for

the treatment of Type 2 diabetes, then exposing people to video education will increase knowledge of diabetes and may improve glycaemic control and cardiovascular risk.

Study 3 is designed to explore an innovative approach to education in people with Type 1 diabetes; that of carbohydrate counting and insulin adjustment taught in a group setting. If matching insulin to the amount of carbohydrate eaten is effective in improving glycaemic control and quality of life in people with Type 2 diabetes, then introducing this system will show improvements in these parameters.

1.22. Aims

The aims of these three studies are:

- To design and assess the impact of a low carbohydrate diet on body weight, A1c, ketone and lipid levels in people with and without Type 2 diabetes
- To develop a novel video-based lifestyle education programme for people newly diagnosed with Type 2 diabetes and to evaluate changes in knowledge, glycaemic control and quality of life
- To evaluate the effect of an education programme for carbohydrate counting and insulin adjustment on glycaemic control and quality of life in people with Type 1 diabetes.

Chapter 2

Materials and methods

2.0. Introduction

This chapter is divided into five main sections exploring the methodology and materials for assessment of the following:

1. Study protocol and designs for:
   - Study 1: an investigation of low carbohydrate diets in the treatment of Type 2 diabetes
   - Study 2: an assessment of video lifestyle education for people newly diagnosed with Type 2 diabetes
   - Study 3: an assessment of carbohydrate counting and insulin adjustment for people with Type 1 diabetes
2. Statistical analyses
3. Dietary intake, quality of life and diabetes knowledge
4. Anthropometric measurements
5. Blood chemistry

2.1. Study protocols and design

2.1.1. Ethical approval and compliance with Good Clinical Practice (GCP)

A study protocol was written and submitted to the Oxford Clinical Research Ethics Committee (OxREC) for Study 1 (low carbohydrate diet study) and Study 2 (video education). Both studies were approved by the ethical committee, the low carbohydrate study was allocated the reference OxREC 04/Q1606/39, and the video education study reference OxREC C03.097. All subjects taking part in Studies 1 and 2 received subject information sheets and provided informed consent before undertaking the trial. Copies of the letters

confirming ethical approval can be found in Appendix 1 of Volume 2. Copies of the subject information sheets and consent forms can be found in Appendices 2 and 3 respectively. Study 3, which was designed to investigate the effect of carbohydrate counting and insulin adjustment in people with Type 1 diabetes was submitted to the Oxford Clinical Research Ethics Committee and received verbal Chairman's approval. The Chairman stated that this study was assessed to be part of clinical audit and, as such, was not considered to be subject to formal submission to the ethics committee.

In compliance with recommended data collection and storage, Case Report Forms (CRF) and Source Data Notes (SDN) were established for all three studies. These forms were completed by members of the research team. Data were stored on computer using Data Protection Act 1999 guidelines and using pre-assigned randomisation numbers. Patients were asked, on the consent form, for permission to access their medical records.

Safety monitoring included recording adverse events (AE). An adverse event (AE) is any unintended or unfavourable sign, symptom or disease occurring during the course of the study, whether or not believed to be related to the intervention. This includes any worsening of a medical condition that was present at time of entry into the study and any clinically significant change in laboratory values. A serious adverse event (SAE) is an adverse event that falls in any of the following categories:

Results in death

Is life-threatening

Requires admission to hospital as an inpatient

Is an important medical event which requires intervention to prevent one of the above.

All adverse events were reported in subject's clinical and trial files and were reported to the study co-ordinator who was responsible for reporting serious adverse events to the ethics committee. During the low carbohydrate study, the video education study and the assessment of carbohydrate counting and insulin adjustment study there were no serious adverse events.

2.1.2. Low carbohydrate diet study (Study 1)

2.1.2.1. Study design

This study was designed as a randomised, parallel group study to assess the safety and efficacy of a low carbohydrate diet for people with established Type 2 diabetes. Randomisation was undertaken by means of sealed envelopes equivalent to the number of subjects and filled fifty-fifty with an indicator of either a low carbohydrate diet or healthy eating advice. Two separate sets of envelopes were prepared for both diabetic and non-diabetic subjects. An independent observer witnessed randomisation. 24 subjects were invited to take part in the study. They were randomly allocated as follows:

12 subjects with Type 2 diabetes randomly allocated to:
6 subjects followed a low carbohydrate diet for 12 weeks
6 subjects followed a standard healthy eating weight reduction programme

12 subjects without diabetes randomly allocated to:
6 subjects followed a low carbohydrate diet for 12 weeks
6 subjects followed a standard healthy eating weight reduction programme

2.1.2.2. Intervention

Groups were randomised by allocation. Both groups followed the same protocol. The intervention group was asked to adhere to a low carbohydrate diet (less than 40g/carbohydrate per day) for 12 weeks. The control group was asked to adopt a standard low fat weight

reduction regime for 12 weeks. All subjects received an individual dietetic consultation and full written information together with formal dietary guidelines. The diets were explained to all subjects by a qualified dietitian at an individual randomisation visit and written information was provided.

2.1.2.3. Low carbohydrate diet

The low carbohydrate diet used in this study was formulated to provide <50g/carbohydrate per day. This quantity of carbohydrate was selected as there is evidence that ketosis readily occurs below this level (VanItallie and Nufert 2003). The most well-known low carbohydrate diet, the Atkins diet, recommends that intakes of carbohydrate should be ≤20g/day for the first two weeks of the regimen and then should be gradually increased. This severe carbohydrate restriction involves avoiding all foods containing sugar and starch, all fruit, all milk and most vegetables and many people find they have side-effects of headache, fatigue, constipation and halitosis on this regimen. Fruit and vegetables are considered essential for health, and the exclusion of milk products may lead to low calcium intakes over the long-term. As a result, it was decided that the diet used in this study should be designed to provide 40g/carbohydrate per day and that all carbohydrate should be supplied from milk, fruit and vegetables as shown in Table 2.1.

Food portion lists of fruit and vegetables were provided to all subjects and they were advised to select from these lists the following amounts; all subjects were advised to consume either 200ml (⅓pt) skimmed or semi-skimmed milk or 125g pot natural or diet yogurt and this provided calcium and 10g carbohydrate. In addition, subjects were advised to select either 2 portions of fruit (20g carbohydrate) and 2 portions of vegetables (10g carbohydrate), or 1 portion of fruit (10g carbohydrate) and 4 portions of vegetables (20g carbohydrate).

Table 2.1. Carbohydrate content of foods for low carbohydrate diet

Food	Amount	Amount of carbohydrate per portion (g)	Total carbohydrate (g)
Milk (skimmed or semi-skimmed)	200ml	10	10
Natural or diet yogurt	125g pot	10	10
Fruit	1 portion	10	10
Vegetables	1 portion	5	5

In addition subjects were provided with a list of all starchy and sugary foods and advised to avoid these foods completely. Information was supplied about foods containing protein and carbohydrate and all subjects were advised that they could choose freely from these lists but that they may lose weight more quickly if they reduced energy intake by selecting lower fat alternatives. Concerns have been expressed about saturated fat intakes on low carbohydrate diets, and as a result subjects were advised to include lean meats, poultry, fish and game, low-fat dairy products, avoid large amounts of saturated fat and use mono-unsaturated fat. A copy of the dietary information given to the subjects can be found in Appendix 4 of Volume 2.

2.1.2.4. Low fat diet

The subjects in the healthy eating group were given information in accordance with the dietary guidelines of Diabetes UK and were issued with a leaflet produced by Diabetes UK which incorporates a ten-step approach to eating well. The ten steps are:

1. Eating three meals daily and avoiding missing meals
2. Including starchy carbohydrate at each meal
3. Reducing total and saturated fat intake
4. Increasing fruit and vegetable intake

5. Including more legumes (beans and lentils)
6. Including oily fish at least twice weekly
7. Limiting sugar and sugary foods
8. Reducing salt and salty foods
9. Drinking alcohol in moderation
10. Avoiding diabetic foods and drinks

Subjects were given specific advice about reducing energy intake to 500kcal/day less than their calculated energy requirements and counselled to aim for slow weight loss at the rate of 1-2lb (0.5-1.0kg) per week rather than reducing intake to achieve rapid weight loss.

2.1.2.5. Physical activity

All subjects, regardless of dietary allocation, were encouraged to increase physical activity and advised to exercise at moderate intensity for 30 minutes at least 5 and preferably 7 days per week.

2.1.2.6. Monitoring

Subjects were required to monitor blood glucose and ketone concentrations four times per day (before breakfast, before lunch, before the evening meal and before bed) during the twelve weeks of the intervention. The subjects used a hand-held combined glucose and ketone meter (Optium, Medisense) which relied upon a simple finger-prick test and which has shown to be an accurate reflection of both blood glucose and ketone levels (Wallace, Meston et al. 2001). During the study, all subjects were asked to complete a three-day food diary, glucose and ketone concentration diary and quality of life and hunger questionnaires. This information was collected at baseline and at monthly intervals throughout the study. Intervention for both groups included a weekly telephone call to monitor blood ketone and blood glucose

concentrations and to offer any support required by the patient. There were clinic visits at four weekly intervals for monitoring and data collection.

2.1.2.7. Outcomes

The primary outcome was change in body weight and body mass index (BMI). Secondary biomedical outcomes included A1c, blood ketone concentrations, lipid concentrations and blood pressure. In addition, measures were made of dietary intake, quality of life and hunger. Routine safety measurements included urea and electrolytes (U&E), liver function tests (LFT), full blood count (FBC) and recording adverse events.

2.1.2.8. Trial Procedures

Subjects were eligible for the study if they fulfilled the following inclusion criteria; aged over 18 years of age, BMI >25 kg/m^2, without Type 2 diabetes or with Type 2 diabetes treated by diet alone or metformin monotherapy. Exclusion criteria included individuals with Type 1 or Type 2 diabetes treated by insulin, sulphonylurea or thiazolidinedione therapy, pregnancy or women of childbearing age without adequate contraception, breastfeeding women, major psychiatric disease including eating disorders, history of alcohol or drug abuse, creatinine level >150mmol/l, abnormal liver function tests (>1.5 x upper limit of reference interval) or any known malignancy. All subjects attended a screening visit prior to the start of the study, a randomisation visit and then 2 monthly visits with a final visit at the end of week 12. In addition, all subjects were offered weekly telephone calls for support and to monitor blood glucose and blood ketone concentrations. All visits required the subject to be fasting, and to ensure this, all subjects were advised to have nothing to eat or drink from midnight the night before attending clinic visits. Subjects were seen a total of five occasions during the study.

At the screening visit, signed consent was taken from each subject and baseline data were collected; these included each subject's age, gender, duration of diabetes, prescribed antidiabetic therapy and other medication. A physical examination recorded height, weight, waist and hip measurement, bioimpedance to asses total body fat and blood pressure. In addition an ECG was performed as a safety measure. Blood samples were collected to assess fasting blood glucose and ketone concentrations, A1c and lipid levels. Quality of life and hunger were assessed using validated questionnaires and each subject was requested to complete a three day food diary.

Once the screening visit had assessed the subject's eligibility for the study and prior to the randomisation, subjects were asked to self-monitor ketone and glucose concentrations for one week while maintaining their usual diet. Readings were taken at staggered times, including before breakfast, mid-morning, before the mid-day meal, mid-afternoon, before the evening meal and at bedtime to provide a picture of usual ketone concentrations for each subject.

Once the subjects had been randomised to either a low carbohydrate or a low fat diet, they were asked to self-monitor and record glucose and ketone concentrations four times a day during the first week of the diet. If ketone concentrations above 2.9mmol/l were recorded, the subjects were advised to contact the research team was. After the first week of the diet, ketone and glucose concentrations were measured 4 times daily for 1 day each week.

On completion of the study at week 12, data were collected for comparison with baseline and included weight, waist and hip measurement, bioimpedance to asses total body fat and blood pressure. Blood samples were collected to assess fasting blood glucose and ketone

concentrations, A1c and lipid levels. Quality of life and hunger were assessed using validated questionnaires and each subject was requested to complete a three day food diary.

Professor David Matthews (DRM) acted as Principal Investigator (PI), Sue Beatty (SB) as research nurse and Pamela Dyson (PAD) as dietitian and lead investigator for this study. All three researchers contributed to the concept and design of the study. PAD wrote the protocol, prepared the paperwork, including the diet booklets, and gained ethical approval for the study. SB and PAD recruited subjects to the study and conducted the clinical visits, with SB taking blood samples, and both researchers collecting and recording anthropometric data. PAD explained and reinforced dietary advice at each clinic visit. SB and PAD collected and entered all clinical data and PAD undertook analysis of the results and drafted a paper for publication. All authors critically reviewed the manuscript and approved the final version submitted for publication in a peer-reviewed journal; Diabetic Medicine.

2.1.3. Video education study (Study 2)

2.1.3.1. Study design

The aims of this study were to design and produce video-based lifestyle education for people newly diagnosed with Type 2 diabetes and to evaluate the effect of video education on diabetes knowledge, biochemical and anthropometric outcomes and quality of life. This study was designed as a randomised parallel group study. Randomisation took place using sealed envelopes filled 50:50 with either immediate or delayed randomisation. Randomisation was observed by an independent monitor. One group was offered the intervention programme immediately, and the other group acted as a delayed intervention control group. All control subjects were offered the programme at the end of the six month study.

2.1.3.2. Intervention - design and production

The video-based education intervention consisted of three 20-minute videos covering three topics; diet and Type 2 diabetes, weight management and physical activity. Subjects randomised to the intervention group were encouraged to watch all three videos in their own time and given evaluation forms to assess each video. These evaluation forms included both visual Lickert scales and free-text boxes. Subjects were given stamped addressed envelopes to return the videos after watching, if this did not occur within three months of randomisation, subjects were telephoned and asked to return the videos.

There is still much discussion about the components of dietary education that should be delivered to people newly diagnosed with Type 2 diabetes. The content of lifestyle education programmes for Type 2 diabetes usually include information about the effect of different foods upon blood glucose levels, on risk factors for cardiovascular disease, weight management and physical activity (Deakin, Cade et al. 2006). It was decided to elicit the views and opinions of people with diabetes and to incorporate these concepts into the videos for the healthy eating programme. A simple questionnaire was designed based upon the topics that are usually included in lifestyle education programmes. A random sample of patients with Type 2 diabetes who attended the Oxford Centre for Diabetes, Endocrinology and Metabolism (OCDEM) during the months January – March 2005 was asked to complete the questionnaire in order to formulate the topics to be included in the video. A sample of the questionnaire is shown in Appendix 5 of Volume 2. The results from these questionnaires were analysed and these data were used as the basis for the videos. A proposal was developed from the questionnaires to generate three 15-20 minute programmes available on video and DVD covering the following modules:

Food choices

Physical activity

Weight management

A full curriculum for each topic was written and is shown below:

2.1.3.2.1 Food choices

Section	Topic	Action plan
1	Introduction	Provide an overview of the effects of food on blood glucose and the principles of a healthy diet – reduced fat, reduced sugar and increased dietary fibre. Discuss the effects of changing eating habits on quality of life. Emphasise the practical application of the theory of nutritional recommendations.
2	Carbohydrates and blood glucose	Provide information about the carbohydrate foods which affect blood glucose levels. Divide the carbohydrate foods into four main groups – starchy foods, sugary foods, fruit and fruit juices and milk and yogurt. Summarise the foods which most affect blood glucose levels.
3	Cardiovascular risk and fat	Advise reduction in animal (saturated) fat. Recommend substitution of fish (omega-3 oils) for meat and poultry. Recommend substitution of monounsaturated and polyunsaturated fats for saturated fat.
4	Healthy eating	Introduce the concept of a healthy diet. Recommend at least 5 portions of fruit and vegetables every day. Advise moderate intake of starchy carbohydrate foods and issue reminder of effect on blood glucose levels.

		Advocate low fat protein foods. Suggest small amounts of sugary and fatty foods. Address issues of culture and different eating patterns.
5	Case studies	Provide case studies of people with diabetes who have successfully adapted their eating patterns.
6	Summary	Provide a summary of a healthy diet for diabetes.

2.1.2.3.2. Physical activity

Section	Topic	Action plan
1	Introduction	Offer an overview of the importance of physical activity in terms of the balance between food, activity and medication. Mention positive effects on blood glucose levels, reducing cardiovascular risk, weight management and well-being.
2	Type of activity	Discuss suitable activity. Explore different factors including enjoyment, any physical limitations, compatibility with current lifestyle and starting with gentle exercise.
3	How to increase exercise	Discuss gradual increase of exercise, starting with increased daily physical activity.
4	Safety issues	Provide guidelines for exercising safely including checking with a doctor before starting moderate or strenuous exercise, wearing suitable footwear and never exercising when unwell. Offer suggestions for suitable treatment for hypoglycaemia.
5	Case study	Provide case study illustrating how to overcome barriers to

		exercise, fitting it into daily life and the benefits of increasing physical activity.

2.1.3.2.3. Weight management

Section	Topic	Action plan
1	Introduction	Provide overview of the relationship between diabetes and weight gain. Discuss cause and effect.
2	Case study	Provide illustration of successful weight management for people with diabetes.
3	Weight management strategies	Offer suggestions for different strategies for weight loss including: • Healthy eating and reduction in quantity eaten • Calorie controlled diet • Slimming clubs
4	Setting targets	Discuss optimum and realistic weight loss.
5	Contact details	Provide contact details for slimming clubs

2.1.3.2.4. Video production

The videos were filmed, edited and produced by Joose TV, a web-casting company with experience in the field of diabetes and health education. The written curriculum was used as the basis for each video and it was recommended that the best approach for health education was to use a mix of comments and advice from experts and a case study approach to illustrate the practical application. For each video, a diabetes dietitian and GPs who have a specialist

interest in diabetes were interviewed and encouraged to discuss food choices, weight management and physical activity. Relevant quotes were then taken from each of these interviews and used for each specific topic. Some gaps were identified, where the expert had not fully explained a subject and a script was then written to be used as a voice-over. An example of this can be found in the 'Food choices' video where a voice-over explaining the effects of carbohydrate foods on blood glucose was inserted. A copy of the voice-over script can be found in Appendix 6 of Volume 2.

The opinion and advice from experts was supported by the use of case studies. Relevant case studies were identified for each topic and interviewed at home, in a non-threatening environment, to elicit their experiences of living with diabetes. Personal experiences from each case study were intercut with quotes from the health professional who had advised each subject and this formed a story line to illustrate the practical application of the theory already outlined by the health expert.

Joose advised that filming each interview with the health professionals and people with diabetes would last between 2-3 hours. It was also necessary to produce some background shots of different foods groups, people exercising and people eating. A filming schedule was drawn up over a 4-week period to complete the filming. All footage was then encoded in the studio at Joose TV and produced approximately 30 hours of footage. The film editor at Joose made the first cut based upon the agreed curriculum for each video and the final cuts were made in conjunction with the specialist dietitian to produce the final 3 videos. The editing process took 2 weeks to complete.

There were some tensions between the production team and the dietitian involved in the filming process. The main area of difference was the focus for the videos. The Joose team, and especially the director, were interested in entertainment, drama and engaging the emotions of the audience, and often encouraged the case studies to put more expression and drama into their stories. The dietitian felt that the main objective should be on supplying accurate and practical advice for people with diabetes and that the individuals in the case studies should be allowed to tell their own stories in their own way. For example, the weight management case study features Muriel who has lost a significant amount of weight, but in the video makes the simple statement 'So, I lost the seven stone.' The director felt that this statement did not betray enough delight in her achievement and attempted to encourage her to say the sentence in various ways to increase the drama. Muriel found this process quite uncomfortable and was unable to make the statement sound natural and eventually the dietitian asked the director to stop and move on. The original statement remains in the final cut.

Once the three videos had been designed and produced, a pilot study was undertaken to evaluate the content and presentation. A random sample of 10 people with type 2 diabetes was selected from routine diabetes clinic, and these subjects were asked to watch each of the three videos and complete a pre-designed form for feedback. A copy of the form is shown in Appendix 7. Once evaluation was complete, the videos received their final edit, taking into account the comments received, and were then copied for use in the video education study.

2.1.3.3. Outcomes

Primary outcomes included changes in levels of diabetes knowledge and understanding, changes in dietary intake and biomedical outcomes including A1c and lipid levels. Secondary

outcomes consisted of weight loss, bioimpedence to assess levels of body fat, quality of life and changes in physical activity, assessed by use of a pedometer.

2.1.3.4. Trial procedures

Subjects were referred to this study from Primary Care through either their General Practitioner (GP) or Practice Nurse once their eligibility had been assessed and confirmed. The inclusion criteria were for subjects aged over 18 years of age, who had had Type 2 diabetes diagnosed within the previous six months. Exclusion criteria included individuals with Type 1 diabetes, pregnancy or women of childbearing age without adequate contraception, breastfeeding women, major psychiatric disease including eating disorders, history of alcohol or drug abuse, creatinine level >150mmol/l, abnormal liver function tests (>1.5 x upper limit of reference interval) or any known malignancy. None of the subjects had received formal education from a State Registered Dietitian at entry to the study. All eligible subjects received information about the study by post and were invited to join the study. They then attended for a baseline clinic visit and a six month follow-up visit. At the baseline visit, signed consent was taken and baseline data were collected. These data included each subject's age, gender, duration of diabetes, prescribed antidiabetic therapy and other medication. A physical examination included collection of data on height, weight, waist and hip measurement, bioimpedance to asses total body fat and blood pressure. Blood samples were collected to measure A1c and lipid levels. Quality of life and diabetes knowledge were assessed using a validated questionnaire. After the baseline visit, all subjects were given a stamped addressed envelope to return a 3-day food diary and record of physical activity using a pedometer at baseline. Once collection of baseline data was complete, they were randomised to either the intervention or the control group. Those subjects allocated the video intervention

were sent either a DVD or video set of the three education programmes. All subjects were sent a 3-day food diary and record of pedometer readings two weeks before the end of the study and asked to complete these and return them at their six-month follow-up visit.

The final visit took place at 6 months post-intervention and data were collected for comparison to baseline including weight, waist and hip measurement, bioimpedance to asses total body fat and blood pressure. Blood samples were collected to A1c and lipid levels. Quality of life and diabetes knowledge were assessed using validated questionnaires and the completed three day food diary and record of physical activity were collected.

Pamela Dyson (PAD) acted as lead investigator and assumed overall responsibility for the day to day running of this study with Professor David Matthews (DRM) acting as Principal Investigator (PI) and Sue Beatty (SB) employed as research nurse. All three researchers contributed to the concept and design of the study. PAD wrote the protocol, prepared the paperwork, organised and supervised the production of the videos and gained ethical approval for the study. SB conducted the clinical visits, taking blood samples and collecting and recording anthropometric data. SB and PAD collected and entered all clinical data and PAD undertook analysis of the results and drafted a paper for publication. All authors critically reviewed the manuscript and approved the final version submitted for publication in a peer-reviewed journal; Journal of Human Nutrition and Dietetics.

2.1.4. Carbohydrate counting and insulin adjustment (Study 3)

2.1.4.1. Study design

This study was designed as an intervention trial investigating the effects of structured education on glycaemic control and quality of life in people with Type 1 diabetes.

2.1.4.2. Intervention

A structured education programme known as InSight was designed and written by a diabetes specialist nurse and a diabetes specialist dietitian. The Insight programme was designed to facilitate skills for matching insulin to carbohydrate intake based upon reflection from self-monitoring diaries and to support self-management of hypoglycaemia, hyperglycaemia, illness and exercise. InSight utilised the adult learning model comprising of experiment and enquiry and relates to empowerment principles (Funnell and Anderson 2004; Meetoo and Gopaul 2004) . It is based upon the theory of experiential learning, interaction of participants, partnership and focuses on developing new skills for self care. There is recognition of the shift from the clinician's traditional approaches, recommending treatments from the medical model and perspective, to an approach of integration of individualised goals (Rollnick, Mason et al. 1999). Skills are developed through feedback and reflection. Participants are taught to match insulin to carbohydrate over a four week period relating the new information gained to actual life situations, in every day life. The role of the health care professional is to provide support to people on the programme to enable them to develop realistic short and long term management goals and to help them acquire the knowledge and skills necessary to achieve those goals.

Carbohydrate counting was facilitated by different techniques, aiming to present the information using different strategies to maximise the acquisition of skills in the course participants. The strategies taught included weighing and calculation from food tables, reading and interpreting nutritional labels and visual estimation. Foods that are commonly eaten in different portion sizes and that are difficult to quantify included breakfast cereals, rice, pasta and mashed potato. The participants were provided with scales for the duration of

the course and pocket-sized copies of food tables and encouraged to weigh their individual portions of food and calculate the carbohydrate content. Nutritional information from food tables was also used and participants were taught to interpret the data provided on the packet. In addition, participants were provided with small booklets, compiled by the consultant dietitian and which included the carbohydrate content of commonly eaten foods, for example sliced bread, biscuits and pots of yogurt. Photographs of foods with calculated carbohydrate content were also utilised to support carbohydrate calculations.

Mealtime insulin doses were calculated from the total daily dose of insulin taken by each individual using the formula:

$$\frac{\text{Total daily dose of insulin}}{50} = \text{amount of insulin (in units) for every 10g carbohydrate eaten}$$

For example, someone with a total daily dose of 100 units of insulin, would inject 2 units of insulin for every 10g carbohydrate eaten. A large pasta meal containing 100g carbohydrate would require 20 units insulin and a lower carbohydrate meal, for example, steak and salad with a small baked potato would contain approximately 50g carbohydrate and would require 10 units of insulin.

The programme took place over four weeks, including 15 hours education in total. Weeks 1, 3 and 4 comprised three hours each of education and week 2 consisted of one six-hour session .The full programme is shown in Table 2.2. In addition to carbohydrate counting and insulin adjustment, the course included diabetes physiology, managing hypoglycaemia and hyperglycaemia and addressed specific lifestyle issues such as alcohol, exercise and stress.

Table 2.2. InSight course programme

Week 1 (3 hours)	Week 2 (6 hours)	Week 3 (3 hours)	Week 4 (3 hours)
Introductions – getting to know one another	Reflection of past week utilising self-monitoring diaries		
Sharing ideas and understanding about diabetes	Carbohydrate counting – theory and practice	Hypoglycaemia	Diabetic ketoacidosis
Exploring the physiology of diabetes	Insulin ratios and correction doses	Managing physical activity	Managing illness
		Effects of alcohol	Any other topics identified by course participants
Self-monitoring using daily diaries and data collection			Using all the information together

The programme was co-facilitated by two diabetes professionals from nursing and dietetic disciplines, with a physician providing clinical support when patients identified specific medical issues. Professional input to the study included delivery of the programme, follow-up at six months and one year and audit and evaluation of the results. In total, each course was calculated to require 44 hours of professional time.

2.1.4.3. Outcomes

The primary outcome was change in glycaemic control measured by A1c, and secondary outcomes included changes in body weight, lipid levels, quality of life and hypoglycaemia measured by validated questionnaires.

2.1.4.4. Study procedure

People with Type 1 diabetes were recruited from the Oxford Centre for Diabetes, Endocrinology and Metabolism (OCDEM) by direct referral from a physician, nurse or dietitian during attendance at a routine clinic visit. All subjects expressing an interest in taking part in the InSight sessions were sent details of the course and invited to contact the facilitators to confirm attendance. Inclusion criteria were subjects aged over 18 years of age with Type 1 diabetes treated by a basal prandial insulin regimen using analogue insulin. Exclusion criteria included individuals with Type 2 diabetes, pregnancy or women of childbearing age without adequate contraception, breastfeeding women, major psychiatric disease including eating disorders and any history of alcohol or drug abuse. All subjects who volunteered to take part in the study were included in the education sessions.

Subjects were invited to the next available group and attended the four-week course. Before each course began, baseline data were collected from all subjects using computerised medical records available in OCDEM. Data collected included age, gender, duration of diabetes, prescribed antidiabetic therapy and other medication, height, weight, A1c and lipid levels. Quality of life and frequency and awareness of hypoglycaemia were assessed by validated questionnaires.

Six months after the education course, subjects were invited to return for a follow-up visit and data were collected for comparison with baseline including weight, A1c and lipid levels, quality of life and frequency and awareness of hypoglycaemia.

Pamela Dyson (PAD) and Janet Sumner (JS, Lead Diabetes Specialist Nurse) acted as co-investigators and assumed overall responsibility for the day to day running of this study with the support of the Clinical Director of OCDEM, Dr Jonathan Levy. PAD and JS contributed to the concept and design of the education programme and PAD wrote the curriculum, prepared the paperwork, and gained Chair's approval for the audit from the local ethics board. PAD and JS jointly facilitated the education programme. Clinic nurses performed routine blood sample collection and recorded anthropometric data. PAD collected and entered all clinical data and undertook analysis of the results.

2.2. Statistical analysis and power calculations

2.2.1. Power calculations for the low carbohydrate study

The power calculation for the low carbohydrate study (Study 1) was based upon weight change from baseline using a two group paired t-test of equal means at a 5% statistical significance. Weight changes from previous studies showed a treatment effect of 3.0 SD. Power calculations indicated that group sizes of 9 would give >90% power at the 0.05 level. The sample size was calculated as 10 in each group and we aimed to recruit 12 to each arm to allow for possible drop out.

2.2.2. Power calculations for the video education study

The power calculation for the video education study (Study 2) was based upon changes in A1c from baseline to follow-up at 6 months. Based on a paired two group t-test of equal means at a 5% statistical significance, the trial had a statistical power of 95% to detect a mean change of 0.5% A1c in the intervention group with an approximate standard deviation of 0.1. Power calculations indicated that group sizes of 27 would give >90% power at the 0.05 level. The sample size was calculated as 30 in each group and we aimed to recruit 40 to each arm to allow for possible drop out, as attrition rates are traditionally high in dietary intervention studies (Dansinger, Tatsioni et al. 2007).

2.2.3. Power calculations for the carbohydrate counting study

The number of subjects taking part in this study was decided on a pragmatic basis taking into account achievable numbers of groups and the optimal number of participants per group. It was calculated that 50 participants could be recruited within the study period.

2.2.4. Data analysis

Date were analysed as absolute change from baseline in subjects who completed the final assessment at three months for the low carbohydrate study (Study 1), six months for the video education study (Study 2) and one year for the carbohydrate counting study (Study 3). Statistical analysis was carried out on an intention to treat basis. In the low carbohydrate study, analysis was performed by means of last observation carried forward at one or two months and this applies to one subject allocated to the low carbohydrate group and one subject allocated healthy eating advice. There were no drop-outs after randomisation in the low carbohydrate group, and this contrasts with four subjects in the low fat group who refused

follow-up after randomisation. In the case of the four drop-outs, missing outcomes were not replaced. In the video education study, there were no drop-outs in the video intervention group and three in the control group; missing outcomes were not replaced for these three subjects who failed to attend the six-month visit. In the carbohydrate counting study, three subjects were lost to follow-up and missing outcomes were not replaced.

All data were analysed using parametric statistics where appropriate and in the low carbohydrate study, triglyceride values were log-transformed before analysis. Normally distributed continuous data variables were compared between groups using independent sample *t*-tests.

The ADKnowl questionnaire used to assess diabetes knowledge in the healthy living video intervention study was analysed as recommended by Clare Bradley (ADKnowl guidelines, Appendix 3). Briefly, the items are scored on a true/false/don't know basis and entered onto an Excel spreadsheet, with a further column to record no response. The percentage of correct answers to each question in both the intervention and the control group was calculated and the absolute changes from baseline could then be analysed and comparisons made between the two groups. As these are non-parametric data, analysis was performed using the Mann-Whitney statistical test.

Data from the PAID and hypoglycaemia questionnaires were analysed using the Mann-Whitney statistical test.

2.3. Dietary intake, physical activity, quality of life and diabetes knowledge

2.3.1. Dietary intake

An objective measure of the impact of dietary education is change in dietary intake. There has been much discussion about the efficacy of measuring dietary intake by self-reported food diaries, with the overwhelming evidence supporting the supposition that most people under-report dietary intake (Kipnis, Midthune et al. 2002; Day, Wong et al. 2004) and that there is a strong inverse correlation between self-reported energy intake and obesity (Prentice, Black et al. 1996). The 7-day food diary has long been regarded as the gold standard for measuring dietary intake (James, Bingham et al. 1981) and is widely used in large epidemiological studies (Bingham 1997). A recent survey of the dietary intake of people with diabetes has shown that 3-day food records are as accurate as 7-day records (Toeller, Buyken et al. 1997) and for this reason have been utiltised in both the low carbohydrate ketogenic diet study (Study 1) and the video education study (Study 2). A copy of the 3-day dietary record booklet can be found in Appendix 8. Nutritional data from the food diaries were analysed by a computer programme, Dietplan5 for Windows (Forestfield Software Ltd 2002).

2.3.2. Physical activity

Changes in physical activity can be determined using objective and subjective measures. Objective measures investigate changes in physical fitness either by measuring the amount of oxygen used or by monitoring heart rate during an exercise test. Indirect measures of changes in physical activity may include self-reported activity diaries or use of a pedometer. Changes in physical activity in the video education study (Study 2) were assessed by means of pedometer as this method is cheap, non-invasive and has been well validated (Freedson and Miller 2000). In addition, the use of pedometers has been shown to be effective in increasing physical activity and improving health (Bravata, Smith-Spangler et al 2007).

2.3.3. Hunger and appetite

One hypothesis for the efficacy of low carbohydrate, ketogenic diets is that ketone production is associated with a reduction in hunger. This hypothesis was tested in Study 1 by measuring hunger and appetite using a validated scale (Roth 1993). This is based upon a Lickert visual analog item and was completed by the participants in the low carbohydrate study before a main meal to indicate the level of hunger. The item was scored from 1-10 using a ten-point scale ranging from 'starving hungry, feeling irritable and dizzy' to 'stuffed to the point of feeling ill'. Lower scores denoted higher levels of hunger and higher scores satiety. The scale is shown in Table 2.3.

Table 2.3. Roth hunger scale

Response	Score
Stuffed to the point of feeling ill	10
Very uncomfortably full, need to loosen belt	9
Uncomfortably full, feel stuffed	8
Very full, have overeaten	7
Comfortably full, feel satisfied	6
Comfortable, neither hungry nor full	5
Beginning to feel peckish	4
Hungry, feel ready to eat	3
Very hungry, unable to concentrate	2
Starving hungry, feel irritable and dizzy	1

2.3.4. Quality of life: the WHO-5 Well-Being Index

General quality of life in both the low carbohydrate (Study 1) and video education studies (Study 2) was measured by the widely-used and well-validated questionnaire, the WHO-5 Well-being index (World Health Organisation 1998). The WHO-5 Well-being index is not diabetes-specific and consists of five simple statements relating to general quality of life over the previous two weeks. Each statement offers a choice of six responses ranging from 'all of

the time' to 'none of the time'. Each answer is allocated a number from 0-5 as shown in Table 2.4.

Table 2.4. WHO-5 Well-Being index: scoring system

Response	Score
All of the time	5
Most of the time	4
More than half of the time	3
Less that half of the time	2
Some of the time	1
At no time	0

Higher numbers denote better well-being. Raw scores are calculated by summing the figures of each response with 0 representing the worst possible and 25 the best possible quality of life. WHO-5 Well-being scores are commonly reported as a percentage and this is derived by multiplying the raw score by 4. A raw score of <13 (52%), or any answers that score either 1 or 0 indicate poor well-being and may be a symptom of depression. A change of 10% in the percentage scores is indicative of a significant change in quality of life. A copy of the WHO-5 Well-being form can be found in Appendix 9.

2.3.5. General Health: the EQ-5D

General health was measured in the video education study (Study 2) by means of the EQ-5D (Brookes, Rabin et al. 2003) which assesses overall health status by means of five dimensions related to mobility, self-care, pain, depression and anxiety and ability to perform usual activities. Each item has three responses these are scored according to the problems that the item presents to the individual, ranging from 'no problem' through 'some problem' to 'extreme problem'. In addition, the EQ-5D VAS was completed by the participants. The EQ-5D VAS is a standard vertical 20 cm visual analogue scale (similar to a thermometer) for scoring a participant's rating for their current health-related quality of life state. The scale is

numbered 0-100 and a score for an individual's health state can be read off the scale and recorded. Value sets for evaluation of the EQ-5D are available, or the data can be reported as a percentage of the population under study reporting any problem (whether slight or extreme) for each of the dimensions. A copy of the EQ-5D form can be found in Appendix 10.

2.3.6. Diabetes related distress: the PAID and hypoglycaemia

The PAID questionnaire (Polonsky, Anderson et al. 1995) was used to assess diabetes related distress in the study investigating the effect of carbohydrate counting and insulin adjustment (Study 3). PAID consists of twenty questions addressing potential problem areas relating to diabetes and each question is scored from 0-4. PAID scores are totalled and expressed as a percentage, with 100% representing maximum diabetes-related distress and 0% minimum distress. Scores over 40% indicate a significant problem. Hypoglycaemia is a major concern for people with Type 1 diabetes and incidence and severity was measured by a validated questionnaire (Clarke, Cox et al. 1995). Copies of the PAID questionnaire and the hypoglycaemia questionnaire can be found in Appendix 11.

2.3.7. Diabetes Knowledge: the ADKnowl questionnaire

Study 2 was designed as an education programme and it was therefore necessary to measure any change in diabetes knowledge. Changes in diabetes knowledge were assessed by a questionnaire (the ADKnowl questionnaire) provided by agreement with Professor Clare Bradley (© Bradley, 1993 latest revision 2001). The ADKnowl is a validated questionnaire for measuring diabetes knowledge (Speight and Bradley 2001; Bradley and Speight 2002). The full ADKnowl questionnaire includes 25 items questions sub-divided into 2-9 sections with a total of 123 questions covering the topics which include general knowledge about

diabetes, medication (tablets and insulin) for treating diabetes, hypoglycaemia, food, alcohol and physical activity, tissue damage and complications of diabetes, foot care and A1c levels.

As the ADKnowl is designed to be analysed item-by-item, users can remove items from the ADKnowl that may not be relevant to the study in question without affecting the overall validity of the questionnaire. Items that are retained cannot be edited, reworded or changed in any way without affecting validity and opportunities to compare with other datasets. Many of the original items of the ADKnowl were not necessary for measuring changes in knowledge of lifestyle factors in the healthy living study and some question were deleted leaving only the relevant questions. The sections covering insulin and tablets are designed to be used only by those taking insulin or sulphonylurea tablets and these were deleted. In addition, the sections covering foot care, hypoglycaemia and A1c were also deleted. The final questionnaire included the following sections:

Section 1: General knowledge of diabetes (5 questions)
Section 2: Effects of physical activity (4 questions)
Section 3: Food and blood glucose levels (7 questions)
Section 4: Food knowledge (9 questions)
Section 5: Alcohol (3 questions)
Section 6: Tissue damage and complications of diabetes (3 questions)
Section 7: Regular examinations (5 questions)

The edited ADKnowl questionnaire used in the healthy living study included seven sections with a total of 36 questions. The questionnaire is designed to be analysed item by item, and it is not recommended that the responses are summed into a composite score, or that a composite knowledge score is related to biomedical indices e.g. A1c. Data from the ADKnowl were analysed item-by-item by presenting the data as percentage of correct reponses to each item by at baseline and at six months in each group. The changes within each

group were compared over the six months of the study and non-parametric analysis was applied to assess significance. A copy of the edited ADknowl questionnaire can be found in Appendix 12.

2.4. Anthropometric measurements

2.4.1. Body weight

A change in total body weight was the primary outcome for measuring the efficacy of interventions that were designed to reduce energy intake or increase physical activity in overweight individuals in both the low carbohydrate and video education studies. Body weight was measured in kilograms by SECA electronic, quality assured scales.

2.4.2. Body Mass Index

A recorded change in total body weight reflects absolute change without allowance for height differences and for this reason a measure of relative weight change was utilised. The most commonly used measurement is that of body mass index (BMI) (World Health Organisation 1995). BMI was calculated using the specified formula wt(kg)/ht(m^2). This calculation requires a measurement of height and this was assessed by means of a wall-mounted appliance (Holtain Ltd, Crymych, Devon).

2.4.3. Waist/hip circumference

Waist and hip circumference were measured a using standard operating procedure (SOP) in the clinical research unit at OCDEM (World Health Organisation 1995). There is growing evidence that the risk of chronic disease is correlated more significantly with abdominal adipose tissue than with peripheral adipose fat and that this is reflected by the waist:hip ratio (Janssen, Katzmarzyk et al. 2002). Waist:hip ratio was calculated by measuring the circumference of the waist and the hips and using the formula waist(cm)/hips(cm).

2.4.4. Body fat

Percentage body fat was measured by body composition analysis. This measurement utilises bioelectrical impedance analysis (BIA) using the Bodystat ^{R} 1500 which had been shown to have an accuracy of within 5% compared to the gold standard of densitometry using underwater weighing (Fuller, Fowler et al. 1994). Body fat was assessed to determine the components of weight lost during the studies and to explore the assumption that the weight loss associated with low carbohydrate diets is associated with fluid and lean tissue loss (Kennedy, Chokkalingam et al. 2005).

2.4.5. Blood pressure

Blood pressure was measured by Omron HEM 757 electronic blood pressure meter. Blood pressure is a surrogate marker for cardiovascular risk and has been shown to fall in individuals who lose weight and increase physical activity (Williams, Poulter et al. 2004).

2.5. Blood chemistry

All blood chemistry was performed by the central biochemistry laboratory at the John Radcliffe Hospital, Oxford and all analysers undergo rigorous quality assurance.

2.5.1. A1c

Glycated haemoglobin (A1c) is the internationally agreed standard for the measurement of longer-term glycaemic control (Barth, Marshall et al. 2008) and was used to asesss glycaemic control in two land-mark studies of diabetes, the Diabetes Control and Complications Trial (DCCT study group 1993) and the United Kingdom Prospective Diabetes Study (UKPDS 1998). Levels of A1c in people without diabetes range from 4.9-6.1%. A1c analysis in these studies was performed using a HA 8160 (A Menarini Diagnostics, Wokingham, Berkshire)

utilising dedicated high pressure liquid chromatography (HPLC) involving reverse phase partition and ion exchange chromatography. Quality assurance performed during the period of the three studies showed the following values of A1c: low 5.6% @ CV of 15.1%; high 9.0% @ CV of 9.4%

2.5.2. Lipid levels

Many people with Type 2 diabetes show symptoms of the metabolic syndrome including dyslipidaemia, characterised by high total cholesterol, high levels of low density lipoprotein cholesterol (LDL), reduced levels of high density lipoprotein cholesterol (HDL) and elevated triglyceride levels (Zimmet, Magliano et al. 2005). Total cholesterol, HDL cholesterol and triglycerides were analysed by multi-stage enzymatic assay by the Seimens Advia 2400 analyser. Quality assurance performed during the period of the three studies showed the following values: total cholesterol; low 2.9 mM @ 3.62%; high 5.8mM @ 6.7%, HDL cholesterol; low 0.89mM @ 3.7%; high 2.6mM @ 7.6%, triglycerides; low 1.06mM @ 7.9%; high 2.92mM @ 7.7%. LDL cholesterol was calculated from levels of total cholesterol, HDL cholesterol and triglycerides levels using the Friedewald equation (Tremblay, Drouin et al. 2004).

2.5.3. Blood ketone levels

Low carbohydrate diets have been shown to increase blood ketone levels (VanItallie and Nufert 2003)and these levels were assessed in the low carbohydrate diet study. Blood ketone levels were measured by a hand-held combined glucose and ketone meter (Optium ™, Medisense) using a finger-prick test which has shown to be an accurate reflection of both blood glucose and ketone levels (Wallace, Meston et al. 2001).

2.6. Funding and conflict of interest

There was no conflict of interest in any of the three studies contributing to this thesis. Study 1, the low carbohydrate diet study was funded by a grant from Medisense UK, Abbott Laboratories. Study 2, the video education study, was partly funded by an unrestricted educational grant from the Sugar Bureau. Study 3, the carbohydrate counting and insulin adjustment study was partly funded by a research grant from Diabetes UK.

2.7. Summary

This chapter describes the materials and methods required for all three studies described, namely the low carbohydrate diet study for people with Type 2 diabetes (Study 1), the video education study for people newly diagnosed with type 2 diabetes (Study 2) and the carbohydrate counting and insulin adjustment study for people with Type 1 diabetes (Study 3). Details of the full protocol, ethical approval and all anthropometric, biochemical and psychosocial data collection and analyses are reported. In addition, a full description of the formulation of all interventions used in the studies is provided and the results of these studies are reported in Chapters 3, 5 and 6.

Chapter 3

Low carbohydrate diets and diabetes

3.0. Introduction

Historically, diabetes has been seen as a disorder of carbohydrate metabolism and the first recommendations for dietary treatment included reduction of carbohydrate as the mainstay for dietary treatment. This concept was first introduced in the UK by RD Lawrence in 1925 and he proposed that dietary intake, especially carbohydrates, should be restricted to help maintain blood glucose levels within the normal range, following the teaching of Frederick Allen in the US (Allen 1925). This type of prescriptive diet, concentrating on carbohydrate restriction was given to patients over the next 50 years and formed the foundation of dietary advice for people with diabetes (Moran 2004).

The first move towards more liberal diets came in 1982 with the publication by the British Diabetic Association (now Diabetes UK) of the first nutritional recommendations for people with diabetes (Nutrition sub-committee of the British Diabetic Association 1982). These recommendations promoted the 'healthy eating' approach for treating diabetes and suggested that the nutritional advice to those with diabetes should not differ greatly from the approach proposed for the general population. The biggest change in these recommendations was that of fat reduction and of an increase in so-called complex carbohydrate intake. Starchy foods were actively encouraged and people with diabetes were commonly advised to fill half their plate with bread, cereals, grains, potatoes, rice and pasta.

Some critics argued against this approach as it is well established that the main type of nutrient in food that affects blood glucose levels is carbohydrate (Sheard, Clark et al. 2004). Most foods contain a mixture of fat, protein and carbohydrate, but foods containing mainly

protein and fat have a minimal effect on blood glucose levels compared to carbohydrate-containing foods. Carbohydrates are found mainly in starchy and sugary foods. All carbohydrates are digested into glucose and appear in the bloodstream between 10 minutes and 2 hours or more after eating, and there has been increasing opposition to the idea that a high-carbohydrate diet is the most effective method of controlling blood glucose levels in people with diabetes. There is still uncertainty about the relative effects of the type and the amount of carbohydrate on postprandial blood glucose levels. There remains some disagreement amongst experts about the importance of the type of carbohydrate rather than the total amount and this has become polarised with some experts arguing for the type of carbohydrate, especially in terms of glycaemic index (Brand-Miller, Hayne et al. 2003) and some recommending approaches incorporating assessment of the amount of carbohydrate (Wheeler and Pi-Sunyer 2008).

3.1. Quality of carbohydrate

Much interest has been expressed in the concept of effects of different types of carbohydrate on blood glucose levels, body weight control and cardiovascular risk in people with diabetes and most research has investigated the effects of two main components of the diet, that of glycaemic index and whole grain foods and dietary fibre.

3.1.1. Glycaemic index and glycaemic load

The term glycaemic index (GI) was first coined in 1981 by researchers at the University of Toronto (Jenkins, Wolever et al. 1981) and was used to give an indication of the effect of different carbohydrate foods on blood glucose levels. The glycaemic index is a scale that ranks carbohydrate foods by how much they raise blood glucose levels compared to a reference food containing an equivalent amount of carbohydrate, usually glucose or white

bread. These first studies by Jenkins showed that high GI foods cause large fluctuations in glucose and insulin levels; conversely low GI foods cause smaller fluctuations and as a result should be of benefit to people with diabetes.

GI is measured practically in the laboratory using a standard procedure. Foods containing 50g of carbohydrate are fed to fasting volunteers and samples for measurement of blood glucose levels are taken every 15 minutes for 2-3 hours. The results of the blood glucose levels are plotted on a graph over time and compared to a reference food, usually pure glucose. The GI of each food is expressed as a number and the lower the number, the lower the GI of the food.

Both the amount (quantity) and type (quality) of carbohydrate will affect blood glucose levels and the concept of the glycaemic load (GL) was introduced in an attempt to rationalise these two components (Salmeron, Manson et al. 1997). The glycaemic load reflects the amount and the type of carbohydrate eaten. The GL can be calculated from the following equation:

$$\text{Glycaemic load} = \frac{\text{glycaemic index of a food}}{100} \times \text{net carbs}^*$$

* net carbs = total carbohydrate – dietary fibre

Low GI diets have been proposed for people with diabetes and claims have been made for beneficial effects on glycaemic control, body weight and cardiovascular risk (Thomas and Elliott 2009). There is also evidence for the effects of low GI diets in the prevention of Type 2 diabetes, with a recent systematic review showing that lower GI diets were associated with reduced fasting blood glucose and A1c (Livesey, Taylor et al. 2008) and another meta-analysis reporting that low GI or low GL diets are independently associated with a reduction in the risk of Type 2 diabetes and heart disease (Barclay, Petocz et al. 2008).

3.1.2. Whole grain foods and dietary fibre

Whole grain foods are rich in dietary fibre and have been associated with both prevention and treatment of diabetes. Evidence for the protective effect of whole grain foods is based upon epidemiology and a recent systematic review of this epidemiological evidence has shown that whole grain intake is inversely associated with risk of Type 2 diabetes (de Munter, Hu et al. 2007). However, a recent Cochrane review of evidence from prospective trails has concluded that the evidence is too weak for firm conclusions of whole grain foods in the prevention of diabetes (Priebe, van Binsbergen et al. 2008). There is more evidence for the role of dietary fibre in the treatment of diabetes, and high fibre intakes have been advocated for over twenty five years (Mann, Kinmonth et al. 1981) and the American Diabetes Association make the recommendation of at least 14g/dietary fibre for each 1000 kcal consumed (Wheeler and Pi-Sunyer 2008).

3.2. Quantity of carbohydrate

The effect of a known quantity of carbohydrate on blood glucose levels has been little studied, although it is well-recognised that carbohydrates raise blood glucose after eating. Studies done in people with Type 1 diabetes have shown that 10g of dietary glucose raises blood glucose by 2 mmol/l, and 20g glucose raises blood glucose by 5mmol/l within 30 minutes of ingestion (Wiethop and Cryer 1993). If this is extrapolated to an average meal containing 50g carbohydrate, this suggests that a theoretical rise in blood glucose of 10 -12.5 mmol/l would follow ingestion of this amount of carbohydrate. People with Type 2 diabetes who exhibit insulin insufficiency may show a postprandial rise in glucose above target levels following a high carbohydrate meal. Traditionally, dietary advice for people with diabetes first recommended carbohydrate restriction in an attempt to control glucose levels after eating.

More recent recommendations have relaxed this approach and recommended higher levels of carbohydrate, up to 55% of total energy intake, accompanied by a reduction in fat intake (Nutrition sub-committee of the British Diabetic Association's Professional Advisory Committee 1992), although there is now recognition by most authorities that for some individuals less carbohydrate may be beneficial, especially for the obese individual (Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK 2003; Bantle, Wylie-Rosett et al. 2008; Wheeler and Pi-Sunyer 2008).

Average carbohydrate intakes in the UK are between 200-300g/day. The National Diet and Nutrition Survey reports mean carbohydrate intakes of 203g/day for women and 275g/day for men (Henderson, Gregory et al. 2003) and dietary survey data from the INTERMAP study have reported means intakes of 307g/day of available carbohydrate for adults in the UK (Zhou, Stamler et al. 2003). There are no recommendations for a maximum amount of dietary carbohydrate for health, and only one authority, the American Diabetes Association, recommends a minimum amount of carbohydrate for health of 130g/day, based upon calculated average demand for glucose by the brain (Wheeler and Pi-Sunyer 2008). This suggests that there is a theoretical range of recommended daily carbohydrate intake of 130 – 300g/day, and there is no evidence for the optimum amount within this range for people with diabetes. There has been increasing interest in the positive effect of low carbohydrate diets on glycaemic control, weight loss and cardiovascular risk factors in people with Type 2 diabetes (Kennedy, Chokkalingam et al. 2005) and this has led to re-evaluation of their role in treatment and an acknowledgement by the American Diabetes Association that they may have a role in weight loss (Bantle, Wylie-Rosett et al. 2008). There remains much discussion between experts about the optimum amount of carbohydrate in the diet, and this is especially

true of people with type 2 diabetes who are overweight or obese (Mann and McAuley 2007). As a result of this uncertainty about the role of carbohydrate in the diet of people with type 2 diabetes, this chapter includes:

- A review of the evidence of the effects of low carbohydrate diet in people with type 2 diabetes
- The results of a low carbohydrate dietary intervention over three months in people with and without type 2 diabetes

3.3. A review of the safety and efficacy of low carbohydrate diets for people with Type 2 diabetes

3.3.1. Background

The health benefits of weight loss for overweight and obese people with Type 2 diabetes are now well established (Aucott, Poobalan et al. 2004) but controversies remain over the most effective dietary intervention to promote successful weight loss (Moore, Summerbell et al. 2004). The majority of people with diabetes who successfully lose weight will regain the lost weight over subsequent months or years (Wadden, Butryn et al. 2004). All national and international diabetes organisations promote weight loss by a combined strategy of increased physical activity and a reduction in energy intake achieved by reducing total fat, saturated fat and processed carbohydrate, but they continue to recommend relatively high total carbohydrate intakes of between 45-60% energy intake (Wolever, Barbeau et al. 1999; EASD 2000; Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK 2003; Bantle, Wylie-Rosett et al. 2008). There is limited evidence that high carbohydrate diets may stimulate appetite and increase energy intake in people with the metabolic syndrome and Type 2 diabetes (Boden, Sargrad et al. 2005) and claims have been made that low fat, high carbohydrate diets may exacerbate obesity and hyperglycaemia (Arora and McFarlane 2005).

This has led to some discussion about the optimal amount of carbohydrate in the diets of people with diabetes to induce weight loss and improve glycaemic control (Mann and McAuley 2007) and an investigation of the safety and efficacy of low carbohydrate diets in the treatment of Type 2 diabetes (Kennedy, Chokkalingam et al. 2005).

Low carbohydrate diets, of which Atkins is probably best known, have been popular for weight reduction since the 1960s. Reduction in carbohydrate intake and thus availability of glucose stimulates fat oxidation to supply energy and this process results in loss of body fat stores and ultimately weight loss. There is now substantial evidence from randomised controlled trials for the positive effect of low carbohydrate diets for weight loss in people without diabetes over the short-term (Foster, Wyatt et al. 2003; Samaha, Iqbal et al. 2003; Stern, Iqbal et al. 2004; Volek, Sharman et al. 2004; Yancy, Olsen et al. 2004), but there is limited evidence for the use of these diets over the longer term and in people with Type 2 diabetes. It has been proposed that the mechanism of action of low carbohydrate diets would benefit people with Type 2 diabetes but whether there would be any added effect over and above reduction in energy intake with associated weight loss is open to question.

The benefits of improvement in glycaemic control for people with Type 2 diabetes were established by the results of the United Kingdom Prospective Diabetes Study in 1998 (UKPDS 1998). Weight loss improves insulin sensitivity and lowers A1c in people with diabetes, but glycaemia *per se* is not the full picture for risk reduction in type 2 diabetes (Aucott, Poobalan et al. 2004). Other risk factors include high blood pressure, alterations in lipid levels and central obesity and are associated with insulin resistance. This cluster of risk factors is found in overweight and obese people with type 2 diabetes and is referred to as the metabolic syndrome (Alberti, Zimmet et al. 2006). The presence of metabolic syndrome

increases cardiovascular risk, which remains the leading cause of death for people with Type 2 diabetes. The evaluation of weight reducing diets, including low carbohydrate diets, should investigate effects on glycaemic control, measures of insulin resistance and cardiovascular risk reduction in people with Type 2 diabetes.

3.3.2. Definition of low and reduced carbohydrate diets

Low carbohydrate diets have been called a variety of names including ketogenic diets, high protein diets and high fat diets. Some high fat and high protein diets are not particularly low in carbohydrate and it has been proposed that the term low carbohydrate should include any diet providing ≤50g carbohydrate/day (Volek and Westman 2002; Adam-Perrot, Clifton et al. 2006) . Although there are differences between individuals in blood ketone levels with various amounts of dietary carbohydrate, it has been shown that ketosis readily occurs at carbohydrate intakes below this level of 50g/day (VanItallie and Nufert 2003).

For the purposes of this review, low carbohydrate refers to a diet providing ≤ 50g of carbohydrate per day, and reduced carbohydrate refers to any dietary intervention designed to lower usual carbohydrate intake.

3.3.3. Mechanism of action of reduced carbohydrate diets

Carbohydrate metabolism begins with digestion in the small intestine to monosaccharides, including glucose, which are then absorbed into the blood stream. Glucose is the preferred metabolic fuel in all tissues of the body, and levels in the bloodstream are regulated by the hormones insulin, glucagon and adrenaline. Consumption of excess energy by the individual (whether as protein, fat or carbohydrate) will result in fat deposition within adipose tissue by the process of lipogenesis. Reduced carbohydrate diets, which are designed to limit both

energy intake and available glucose, result in increased fat oxidation generating free fatty acids (FFA) to supply energy needs and lead ultimately to weight loss (Adam-Perrot, Clifton et al. 2006).

Fatty acids can be oxidised by the liver and muscles for energy production. In the liver, FFA are partially oxidised to form ketone bodies as the liver attempts to limit the rise in plasma FFA levels. Unlike muscle tissue, the liver has no need to completely oxidise FFA for its own energy needs and ketone bodies are produced as an overflow mechanism of FFA metabolism. These ketone bodies can be used as a fuel by tissues including the brain and muscles.

Ketone levels are raised in most fasting subjects where fat oxidation is providing energy, although levels do not rise to those seen in diabetic ketoacidosis, as the presence of insulin inhibits acceleration of ketone production. There has been much discussion over the role of ketones in the mechanism of weight loss in low carbohydrate diets; animal models suggest that circulating ketones have a direct effect on appetite by increasing satiety (Volek and Westman 2002). There is some contradictory evidence for the effects of mild dietary-induced ketosis, one study reported ketogenic low carbohydrate diets have a more favourable effect on glycaemia (Gumbiner, Wendel et al. 1996) and a more recent report stated that ketogenic diets have no metabolic advantage over non-ketogenic low carbohydrate diets (Johnston, Tjonn et al. 2006).

Restriction of carbohydrate intake is usually accompanied by a reduction in total energy intake leading to weight loss; there is now evidence from trials of low carbohydrate diets in people without diabetes that energy restriction accounts fully for all weight loss in individuals adopting these diets (Bravata, Sanders et al. 2003).

3.3.4. Characteristics of studies

Metabolic studies in people with Type 2 diabetes on reduced carbohydrate diets have shown that low carbohydrate diets have direct effects on glucose metabolism by reducing plasma glucose levels (Gannon and Nuttall 2004), increasing insulin sensitivity (Boden, Sargrad et al. 2005), and reducing postabsorptive glycogenolysis (Allick, Bisschop et al. 2004). At present, there are no published studies of randomised controlled trials including an intervention diet which provides ≤50g carbohydrate/day in people with Type 2 diabetes. An electronic search was performed using MEDLINE (1966 – March 2007), EMBASE (1988 – March 2007) and the Cochrane Central Register of Controlled Trials (1991 – March 2007) using the search terms low carbohydrate, Type 2 diabetes and weight loss. All studies relating to intervention trials of low carbohydrate diets in people with Type 2 diabetes were included. Six trials were identified and included in this review, see Fig 3.1 Of these six trials, only one was a randomised controlled trial, two were designed as cross-over trials and the remaining three trials were single-arm intervention studies. The follow-up period of these trials tended to be of short duration and ranged from 14 days to 22 months, and only two studies reported data beyond six months. There was little consistency in the amount of carbohydrate included in the intervention arms of these studies, the lowest intake is reported as <20g/day and the highest as 95g/day. In addition, the number of subjects in each study ranged from 10 -102 and, where age was reported, was confined to a population in late middle-age (average age 51 – 66 years). Table 3.1 summarises details of the clinical trials of hypocaloric, reduced carbohydrate diets in people with Type 2 diabetes that have been published to date.

Fig 3.1. Quorum flowchart of reviewing process for articles investigating low carbohydrate diets and Type 2 diabetes

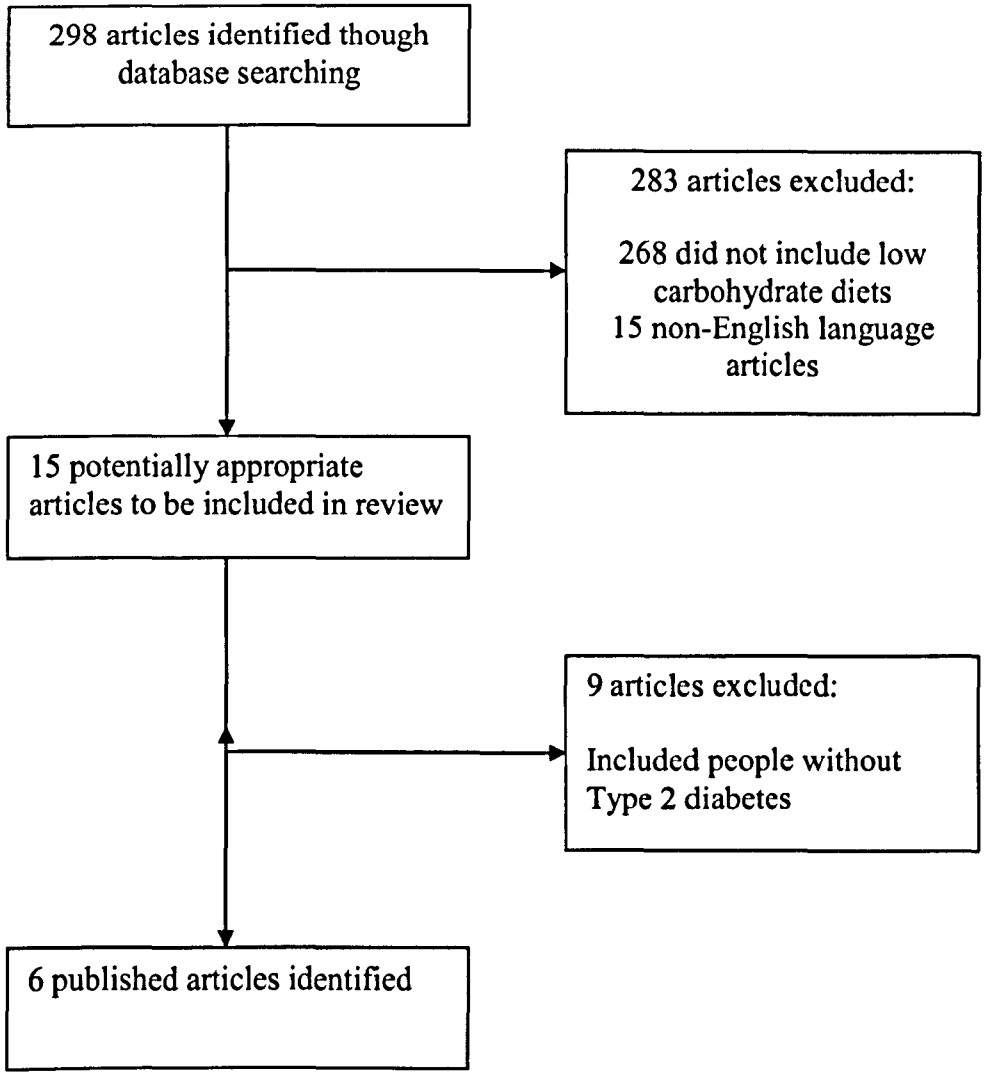


Table 3.1. Details of clinical trials of hypocaloric, low carbohydrate diets in people with Type 2 diabetes

Author	No of subjects	Age (years)	Male/Female	Type of study	Carbohydrate intake (g/day)	Duration of study
Gutierrez, 1998	28	66.4	8/20	Cross-over	25% total energy*	8 weeks
Robertson, 2002	88	N	42/46	Single arm intervention	≤40	12 months
Boden, 2005	10	51	3/7	Cross-over	21	14 days
Yancy, 2005	28	56	20/8	Single arm pilot intervention	≤20	4 months
Nielsen, 2005	31 (16 intervention/15 control)	N	N	Non-randomised intervention with comparison control group	75-95	6 months
Neilsen, 2006	28 (16 intervention/7 cross-over/5control)	N	N	Retrospective follow-up of above study	*	22 months
Daly, 2006	102 (51 intervention/51 controls)	58.7	49/53	Randomised controlled trial	70	3 months
Daly, 2006	206	N	N	6 month follow-up data from above study	*	6 months

* No details of absolute carbohydrate intake given
N No data reported

3.3.5. Positive effects of low carbohydrate diets

Low carbohydrate diets are postulated to have positive effects on body weight, glycaemic control, body composition, medication use and attrition rates in people with Type 2 diabetes. These factors are discussed more fully below.

3.3.6. Weight and glycaemic control

Table 3.2 shows changes in weight, BMI and A1c from baseline for each of the six studies in people with Type 2 diabetes. A short-term, 8-week cross-over study in 28 individuals of a diet providing 25% energy as carbohydrate showed significant reductions in weight and A1c, compared with a diet providing 55% energy from carbohydrate after cross-over for 12 weeks (Gutierrez, Akhavan et al. 1998). An intervention study of a low carbohydrate diet (<40g/day) in 88 subjects reported significant reductions in both A1c and body weight at one year (Robertson and Broom 2002). A short-term in-patient study (21g carbohydrate/day) over 14 days in 10 obese subjects reported greater weight loss and significant improvements in glycaemic control and insulin sensitivity (Boden, Sargrad et al. 2005). A more recently published intervention study reported improvements in A1c and body weight over 16 weeks with a 20g/day carbohydrate intake in 28 subjects (Yancy, Foy et al. 2005). A non-randomised study in 31 diabetic subjects compared a 20% carbohydrate diet (equivalent to 85-96g/day for men and 75-85g/day for women) to a low fat diet (25% energy as fat) over six months and reported significant reductions in weight and A1c (Nielsen, Jonsson et al. 2005). This benefit remained at 22 months retrospective follow-up. Interestingly, 7 of the original 15 controls had crossed over to the reduced carbohydrate diet after the six months intervention and these data are included in the follow-up report at 22 months (Nielsen, Westerlund et al. 2006). A recent and larger scale randomised trial of 102 patients with Type 2 diabetes

compared a prescribed 70g/day carbohydrate intake (actual reported intake 109.5g/day) with a low fat diet (<35% energy as fat) and showed a significant reduction in body weight and improvement in lipid profile, but no significant change in A1c over 3 months compared with a control (Daly, Paisey et al. 2006). Data for 206 subjects at 6 months showed maintenance of significant weight loss, but no change in differences in A1c between the two groups (Daly, Piper et al. 2006).

Other studies that have been published show that a reduction in carbohydrate intake does have positive effects in people with diabetes, but as these studies either included a small diabetic sub-group (54 of 132 subjects) and did not fully report changes in the sub-group (Stern, Iqbal et al. 2004) or used a relatively high carbohydrate intake (138g/day) (Sargrad, Homko et al. 2005), they have not been included in this review. Two studies also excluded from this review investigated the effect of a eucaloric low carbohydrate diet on glycaemic control in people with Type 2 diabetes and have shown positive metabolic effects of low carbohydrate diets in the absence of weight loss, including reductions in glucose and insulin levels, A1c and triglyceride levels (Allick, Bisschop et al. 2004; Gannon and Nuttall 2004).

It can be seen from Table 3.1 that only three of these studies reduced carbohydrate intakes to < 50g/day, and that two of these three studies were of short duration (less than six months). The lack of randomised controlled trails in this area and the varying amounts of carbohydrate prescribed in the trials to date make it difficult to reach significant conclusions about the role of carbohydrate in weight loss and glycaemic control for people with Type 2 diabetes.

Table 3.2. Changes from baseline in body weight, BMI, and A1c in studies of hypocaloric, low carbohydrate diets in people with type 2 diabetes

Author	Weight (kg)			BMI (kg/m^2)			A1c (%)		
	Baseline	Weight loss	P value	Baseline	Change	P value	Baseline	Change	P value
Gutierrez, 1998	76.2	1.2	N	28.5	-0.4	N	9.7	-1.7	<0.05
Robertson, 2002	109.6	7.2	N	38.6	-2.5	N	9.6	-0.8	N
Boden, 2005	114.8	2.0	0.042	40.3	-0.8	N	7.3	-0.5	0.006
Yancy, 2005	131.4	8.7	<0.001	42.2	-2.8	<0.001	7.5	-1.2	<0.001
Nielsen, 2005	100.6	11.4	<0.001	36.1	-4.1	<0.001	8.0	-1.4	<0.001
Nielsen, 2006	N	8.6	<0.001	N	-3.2	<0.001	N	-1.1	<0.001
Daly, 2006	102.0	3.6	0.001*	36.1	-1.3	N	9.1	-0.55	0.132*
Daly, 2006	N	3.8	<0.0005*	N	N	N	N	-0.48	NS

N No data reported
NS Not significant
* Significance assessed by comparison with changes from baseline in the control group

3.3.7. Body composition

All these published studies report significant weight loss with hypocaloric, reduced carbohydrate diets in people with Type 2 diabetes. A theoretical view is that the majority of weight loss seen with low carbohydrate diets is due to water losses associated with glycogen depletion and increased urinary ketone secretion (Denke 2001). However, this is not supported by evidence from two of the studies in diabetic subjects that investigated body composition and which showed that the majority of weight loss is explained by a reduction of body fat rather than fluid loss (Boden, Sargrad et al. 2005; Yancy, Foy et al. 2005)

Fat distribution, and specifically high levels of visceral fat, is a known risk factor for heart disease and diabetes and changes in intra-abdominal fat were investigated by two studies. Both report a positive effect of a reduction in carbohydrate intake; one reports a 5% decrease in waist circumference from 130 to 123 cm ($p=<0.001$) (Yancy, Foy et al. 2005) and another reports a loss of 4.4cm over 6 months ($p=<0.001$) (Daly, Piper et al. 2006).

3.3.8. Medication

The majority of studies in people with type 2 diabetes have included subjects taking a variety of glucose-lowering therapies, including metformin, thiazolodinediones, sulphonylurea and insulin. The studies that report changes in medication as a result of adopting a reduced carbohydrate diet have recorded either reduction or discontinuation of this medication (Gutierrez, Akhavan et al. 1998; Boden, Sargrad et al. 2005; Yancy, Foy et al. 2005; Daly, Paisey et al. 2006; Nielsen and Joensson 2006). For example, Daly (Daly, Paisey et al. 2006) reported that insulin doses were reduced in 85% of those adopting a low carbohydrate diet compared with 22% of the low fat group. Robertson (Robertson and Broom 2002) stated that

before adoption of a reduced carbohydrate diet, all sulphonylurea therapy was stopped and insulin doses reduced by 50%.

3.3.9. Attrition rates

Attrition rates in dietary intervention studies are traditionally high, commonly between 30-60% (Dansinger, Gleason et al. 2005). In these low carbohydrate studies in people with diabetes, attrition rates ranged from no drop-outs for the short-term studies over 2 and 8 weeks, to 10-25% attrition for those conducted over three months to two years. These rates are relatively low, but little is known about attrition rates for most reduced carbohydrate intervention studies over the longer term.

3.3.10. Potential adverse effects of reduced carbohydrate diets

Intuitively, it has been assumed that reduced carbohydrate diets, by definition, provide higher amounts of both fat (especially saturated fat) and protein in the diet. Potential adverse effects associated with this dietary change include dyslipidaemia as a result of a high fat intake, decline in renal function and increased calcium loss from bones as a result of increased protein intake, compromised nutritional intake as a result of low intakes of dietary fibre, fruit, vegetables and milk products and finally, adverse effects of ketosis on cognitive function. At present, there is little evidence for the safety of reduced carbohydrate diets in people with Type 2 diabetes over the long-term and this is discussed more fully below.

3.3.11. Cardiovascular risk

Concern has been expressed about the effect of high fat intakes on blood lipid levels and risk of cardiovascular disease. This view is largely refuted for people without diabetes by a meta-analysis of the effect of low carbohydrate diets that shows favourable changes in triglyceride

and HDL cholesterol, but higher LDL cholesterol (Nordmann, Nordmann et al. 2006). A recent review of aspects of low carbohydrate diets in people without diabetes reports significant reductions in both postprandial lipaemia and fasting triglyceride levels (Adam-Perrot, Clifton et al. 2006). Evidence from long-term studies in people with diabetes is lacking; from the studies reported in Table 3.2, only 4 authors report changes in lipid levels and these are summarised in Table 3.3. Two studies have reported a significant reduction in triglyceride levels, with no significant changes in total cholesterol, HDL cholesterol or LDL cholesterol (Yancy, Foy et al. 2005). Neilson (Nielsen and Joensson 2006; Nielsen and Joensson 2008) did not report levels of LDL cholesterol at either 6 or 22 months follow-up, but stated there was a significant rise in HDL cholesterol from baseline and no significant change in triglyceride and total cholesterol levels. Daly (Daly, Paisey et al. 2006) does not report absolute values for total, HDL and LDL cholesterol at 3 months, but states that there was significant reduction in the ratio of total:HDL cholesterol in the group allocated a reduced carbohydrate intake. Lipid levels have not yet been reported for this study at 6 months follow-up.

The main trend from these studies appears to be a reduction in triglyceride levels with no significant change in other lipid levels and little evidence for any increased cardiovascular risk in people with type 2 diabetes. However, these results must be interpreted with caution as these studies are short-term and most lack a control group.

3.3.12. Renal function

Nutritional recommendations for people with diabetes promote a moderate protein intake to reduce the risk of renal disease. Reduced carbohydrate diets are assumed to include larger quantities of dietary protein and this may have an impact on renal function. However, only

two of the studies in people with Type 2 diabetes have investigated renal function. One study over two weeks reported a small but significant rise in mean blood urea nitrogen, but no changes in serum creatinine and uric acid levels and no changes in urinary creatinine and albumin excretion (Boden, Sargrad et al. 2005). Yancy (Yancy, Foy et al. 2005) reported a reduction in serum creatinine and an increase in urea nitrogen over 16 weeks. Neither of these changes reached statistical significance. Counter-intuitively, a recent case-study reported that a low carbohydrate diet was successful in preventing end-stage renal failure in one individual (Nielsen, Westerlund et al. 2006). There are no data from long-term studies in people with or without diabetes to either support or refute claims that reduced carbohydrate diets impair renal function and further research is needed in this area (Bantle, Wylie-Rosett et al. 2008).

3.3.13. Calcium balance

There have been suggestions that reduced carbohydrate diets may have a negative impact in bone health and calcium metabolism as the acidosis associated with the presence of ketones, coupled with high protein intakes, promotes urinary calcium loss (Adam-Perrot, Clifton et al. 2006). There is very little evidence for the effects of reduced carbohydrate diets on calcium metabolism. One study in people without diabetes showed no effect of increased dietary protein over the short-term (Kerstetter, O'Brien et al. 2005) and only one study in people with diabetes investigated effects on serum calcium levels (Yancy, Foy et al. 2005). This study reported no change in serum calcium levels over 16 weeks in individuals adopting a low carbohydrate diet. There is no evidence for the effects of reduced carbohydrate diets on calcium balance and risk of osteoporosis in the long-term.

Table 3.3. Effect of low carbohydrate diets on lipid levels in people with Type 2 diabetes

Author	Total cholesterol (mmol/l)			HDL cholesterol (mmol/l)			LDL cholesterol (mmol/l)			Triglycerides (mmol/l)		
	Baseline	Change	P value	Baseline	Change	P value	Baseline	Change	P value	Baseline	Change	P value
Boden, 2005	4.68	-0.44	<0.02	1.16	-0.02	NS	2.61	-0.05	NS	1.84	-0.65	<0.001
Yancy, 2005	4.61	-0.07	NS	0.92	+0.07	NS	2.51	+0.26	NS	2.69	-1.12	0.001
Nielsen, 2005	5.6	+0.5	NS	1.1	+0.2	<0.001	3.9*	+0.3*	N	1.4	0	NS
Nielsen, 2006	N	-0.1	NS	N	+0.2	<0.001	N	-0.2*	N	N	0	NS
Daly, 2006	4.89	N	N	1.2	N	N	2.6*	N	N	2.5	-0.67	0.223

* Calculated from published data
N. No data reported
NS. Not significant

3.3.14. Dietary intake

A major criticism of reduced carbohydrate diets is that they may be nutritionally inadequate as they restrict consumption of foods generally associated with good health; fruit, vegetables, starchy foods and milk products (Adam-Perrot, Clifton et al. 2006). There is no evidence at present to support or refute these claims. Two studies in people with diabetes reported dietary intake and found that although there was a highly significant decrease in both energy and carbohydrate intake, that absolute intakes of both protein and fat did not change significantly (Yancy, Foy et al. 2005). A third study (Daly, Paisey et al. 2006) reported that there were significant increases in the amount of protein and fat in the diet of a reduced carbohydrate group compared with a control group receiving low fat advice, but this study was a comparison between groups after the intervention and did not include any data at baseline. However, although absolute protein and fat intakes were higher in the reduced carbohydrate group, these still remain lower than maximum recommended levels. In addition, the reduced carbohydrate group ate similar amounts of fruit and vegetables and significantly more oily fish than the low fat group. Calcium intakes were similar between the groups, but fibre intakes were significantly lower in the reduced carbohydrate group. The long-term nutritional effects of reduced carbohydrate diets in people with Type 2 diabetes are unknown.

3.3.15. Cognitive function and quality of life

There is very little published evidence for the effects of low carbohydrate ketogenic diets on either quality of life or cognitive function in people without diabetes and no published data for people with diabetes. Of the two small, randomised, controlled trials studies conducted on people without diabetes, one study including 21 obese women allocated to either ketogenic or nonketogenic very low energy liquid diets reports that hypocaloric, ketogenic diets have no effect on performance on attention tasks, but neurophysical tests requiring high order mental

processing and flexibility are adversely affected (Wing, Vazquez et al. 1995). A six-week randomised trial in 20 obese individuals used the Profile of Mood States (POMS) to measure six distinct mood states and found that a low carbohydrate ketogenic diet showed no difference in five of the six mood states measured, but that vigour-activity scores were significantly lower in those allocated a ketogenic diet (Johnston, Tjonn et al. 2006).

3.3.16. Conclusions

In summary, the available evidence shows that in all intervention studies of reduced carbohydrate diets in people with Type 2 diabetes, there were reductions in both body weight and A1c. No study published to date has shown a deleterious effect on glycaemic control or cardiovascular risk factors, but these findings should be interpreted with caution as there were no control groups in the majority of studies and that these studies are of short-term duration. At present, it appears that low carbohydrate diets may be useful over the short term to promote weight loss in people with Type 2 diabetes, but there is no evidence that these diets are more successful in the long-term than traditional approaches. More research is needed to investigate the long-term effects of these diets on weight loss, glycaemic control, lipid levels, calcium metabolism and nutritional adequacy in people with diabetes. In an attempt to identify the role of low carbohydrate diets in people with and without type 2 diabetes, we designed and evaluated a randomised controlled trial of low carbohydrate diets (<40g/day) over three months.

3.4. A randomised controlled trial of low carbohydrate and low fat, healthy eating diets in people with and without Type 2 diabetes

3.4.1. Introduction

There has been a resurgence of interest in the role of low carbohydrate diets and weight loss over the past few years. Evidence from randomised, controlled trials have shown low

carbohydrate diets are effective for weight loss over the short-term in people without diabetes (Brehm, Seeley et al. 2003; Foster, Wyatt et al. 2003; Yancy, Olsen et al. 2004), but the effect appears to be related to reduction in energy rather than carbohydrate intake (Bravata, Sanders et al. 2003) and concern has been expressed about the long-term effects of these diets (Astrup, Meinert Larsen et al. 2004). In addition, little is known about the long-term effects of these diets, studies have shown that weight loss is greater with low carbohydrate diets compared with low fat diets over six months (Samaha, Iqbal et al. 2003), but the difference disappears at twelve months follow-up (Stern, Iqbal et al. 2004). Concerns about the effects of a relatively high fat diet on cardiovascular risk factors appear to be unfounded over the short-term (Meckling, O'Sullivan et al. 2004; Volek, Sharman et al. 2004), even in individuals with established heart disease (Gann 2004) but little is known about the long-term effects. Low carbohydrate diets have been shown to decrease insulin levels and increase ketosis (Volek, Sharman et al. 2002; Volek and Westman 2002; Gann 2004) and these effects have been noted within 72 hours of starting the diet (Willi, Oexmann et al. 1998). There is no evidence that ketone production varies between diabetic and non-diabetic subjects, or for the role of ketosis in weight loss.

Although there is now evidence from randomised controlled trials for the effect of low carbohydrate diets in people without diabetes, there is limited evidence for people with Type 2 diabetes. The review above of previous studies in people with Type 2 diabetes reports that most studies have involved small numbers of subjects, lacked a control group, had high attrition rates, had short follow-up periods or used a relatively moderate carbohydrate restriction and have shown highly variable effects upon weight loss and glycaemic control . In response to these somewhat contradictory results, the study reported below was designed to evaluate the effect of a low carbohydrate diet (40g/day) on quality of life, appetite and

biomedical outcomes including body weight, glycaemic control, ketone production and lipid levels in subjects with and without type 2 diabetes and to compare this with subjects allocated a low fat diet.

3.4.2. Aims and Objectives

This study was designed with a primary aim to investigate the effect of a low carbohydrate ketogenic diet on weight loss, biomedical outcomes and quality of life in people with and without Type 2 diabetes. In addition, secondary outcomes included the investigation of the rate of development and concentration of ketone bodies in both diabetic and non-diabetic subjects following a low carbohydrate diet and to investigate the relationship between ketone production and weight loss.

3.4.3. Subject Selection

The study population was designed to include 24 overweight individuals, 12 with Type 2 diabetes and 12 non-diabetic individuals, who meet the inclusion/exclusion criteria.

3.4.7. Methods

The methodology for this study is explained fully in Chapter 2. Briefly, 26 (13 subjects with type 2 diabetes and 13 without) overweight or obese individuals were recruited from a volunteer database held in the research unit at OCDEM, from direct referral from a physician during attendance at a routine clinic visit or by means of advertisements placed around the hospital. All subjects expressing an interest were invited to attend an appointment at the research unit where the study was explained in detail and they were invited to participate. Subjects were randomly assigned either a low carbohydrate diet (40g/day) or a low fat, weight reducing diet. All subjects who agreed to take part in the study had not attempted weight loss in the previous twelve months and all non-diabetic subjects had never received formal dietary

advice. All subjects with diabetes had received dietary advice in line with Diabetes UK recommendations at diagnosis, but none had had formal dietary intervention in the preceding twelve months.

3.4.8. Results

Baseline physical characteristics of the 26 subjects recruited into the study are shown in Table 3.4 by dietary allocation and Table 3.5 by diagnosis of diabetes. There were no significant differences between the two groups for any of the baseline variables. Table 3.5 shows a comparison of the characteristics of the diabetic and non-diabetic subjects at baseline. There were no differences for any of these variables with the exception of waist:hip ratio and A1c, and this was, obviously, significantly higher in the diabetic population (7.3 v 6.0%, p=0.001).

Three-month data are presented for 22 (85%) of the 26 patients randomised into the study. Fig 3.2 shows that 12 subjects were randomised to the low carbohydrate diet and 14 to low fat advice. Analysis is by last observation carried forward at one or two months and this applies to one subject allocated to the low carbohydrate group and one subject allocated low fat advice. There were no drop-outs after randomisation in the low carbohydrate group, and this contrasts with four subjects in the low fat group who refused follow-up after randomisation. All four of these subjects expressed disappointment at being allocated to the low fat dietary arm of the study rather than the low carbohydrate diet, and their decision to withdraw was made as they did not receive their preferred diet.

The differences in the changes between the low carbohydrate and low fat groups at three months are shown in Table 3.6 and % changes from baseline are shown in Fig 3.3. There was a significant reduction in weight in both groups, but a greater reduction in the low carbohydrate group compared with the low fat group (6.9 v 2.1kg, p=0.003). There was also a

significant reduction in the amount of body fat in the group allocated the low carbohydrate diet (-1.5 v 0.6%, p=0.012). There were no differences between the groups for all other measured variables including A1c, lipid profiles, blood pressure, quality of life and hunger score.

Tables 3.7 and 3.8 show the differences in characteristics at baseline and three months and the differences from baseline at three months in the diabetic and non-diabetic subjects by dietary allocation.

Table 3.4. Baseline characteristics of 26 diabetic and non-diabetic subjects

Variable	Low carbohydrate (n=12) Mean (SD)	Low fat (n=14) Mean (SD)	All subjects (n=26) Mean (SD)
% male	17	36	23
Age (years)	55 (5)	50 (12)	52 (9)
Weight (kg)	95.6 (16.7)	97.0 (17.2)	96.3 (16.6)
BMI	35.1 (6.8)	35.0 (7.4)	35.1 (7.0)
Waist:hip ratio	0.90 (0.07)	0.91 (0.07)	0.90 (0.07)
Body fat (%)	47.3 (8.6)	43.9 (8.7)	43.5 (8.7)
A1c (%)	6.7 (1.3)	6.6 (1.0)	6.6 (1.1)
Total cholesterol (mmol/l)	5.1 (1.3)	5.1 (0.8)	5.1 (1.1)
HDL cholesterol (mmol/l)	1.28 (0.44)	1.37 (0.33)	1.32 (0.38)
LDL cholesterol (mmol/l)	3.1 (1.1)	3.1 (0.8)	3.1 (0.9)
Triglycerides (mmol/l)	1.55 (1.01, 2.35)*	1.12 (0.74, 1.72)*	1.48 (0.82, 2.14)*
Ketones (mmol/l)	0.0 – 0.2**	0.0 – 0.1**	0.0 – 0.2**
Blood pressure: Systolic Diastolic (mmHg)	138 (20.6) 79 (13.4)	138 (20.4) 84 (8.4)	138 (11.0) (11.0)
Quality of life (%)	60.7 (21.6)	49.4 (21.7)	54.6 (22.0)
Hunger score (Lickert scale)	3.8 (0.8)	4.5 (1.2)	4.2 (1.1)

SD standard deviation
* Triglyceride concentrations expressed as geometric means
** Ketone concentrations expressed as ranges

Table 3.5. Baseline comparison of diabetic and non-diabetic subjects

Variable	Diabetic subjects (n=13) Mean (SD)	Non-diabetic subjects (n=13) Mean (SD)	*p*-value
% male	30	23	
Age (years)	54 (9)	51(9)	0.331
Weight (kg)	99.0 (12.9)	93.5 (19.8)	0.412
BMI (kg/m^2)	34.8 (4.8)	35.8 (7.1)	0.874
Waist:hip ratio	0.93 (0.07)	0.87 (0.05)	0.016
Body fat (%)	43.6 (9.6)	47.3 (7.5)	0.284
A1c (%)	7.3 (1.3)	6.0 (0.3)	0.001
Total cholesterol (mmol/l)	4.8 (1.2)	5.4 (0.9)	0.184
HDL cholesterol (mmol/l)	1.34 (0.5)	1.31 (0.2)	0.837
LDL cholesterol (mmol/l)	2.8 (0.9)	3.4 (0.9)	0.090
Triglycerides (mmol/l)	1.51 (0.76, 2.27)*	1.47 (0.57, 2.04)*	0.862
Ketones (mmol/l)	0 – 0.1**	0 – 0.2**	
Blood pressure:			
Systolic	142 (20.5)	134 (19.5)	0.287
Diastolic (mmHg)	82 (10.4)	82 (12.2)	0.994
Quality of life (%)	62.2 (23.0)	47.1 (18.8)	0.734
Hunger score (Lickert scale)	4.1 (0.8)	4.2 (1.4)	0.734

SD standard deviation
* Triglyceride concentrations expressed as geometric means
** Ketone concentrations expressed as ranges

Fig 3.2. Consort Flowchart

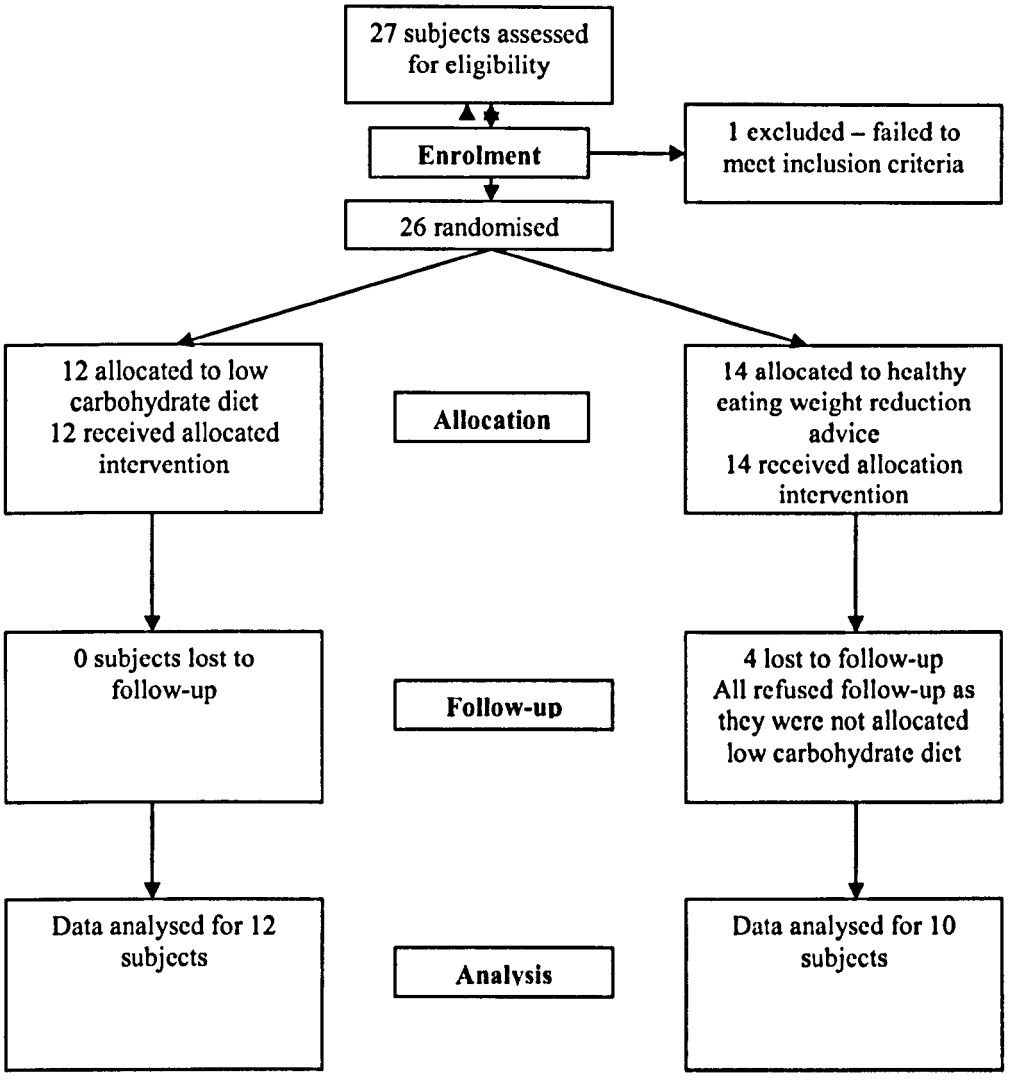


Table 3.6. Absolute changes from baseline at three months, low carbohydrate v low fat diet

Variable	Low carbohydrate diet	Low fat diet	*p*-value
Weight (kg)	-6.9	-2.1	0.003
BMI (kg/m^2)	-2.7	-0.8	0.001
Waist:hip ratio	-0.01	-0.01	0.990
Body fat (%)	-1.5	0.6	0.012
A1c (%)	-0.3	-0.2	0.582
Total cholesterol (mmol/l)	0.1	-0.1	0.282
HDL cholesterol (mmol/l)	0.09	-0.06	0.113
LDL cholesterol (mmol/l)	0.2	0	0.126
Triglycerides* (mmol/l)	-0.4	0	0.07
Ketones (mmol/l)	0.1	0	0.055
Blood pressure:			
Systolic	-0.9	-4.6	0.386
Diastolic (mmHg)	2.2	-3.4	0.186
Quality of life (%)	6	9	0.715
Hunger score (Lickert scale)	-0.6	0.6	0.106

* Triglyceride concentrations reported as changes in geometric means

Fig 3.3. % changes from baseline at three months, low carbohydrate v low fat diet

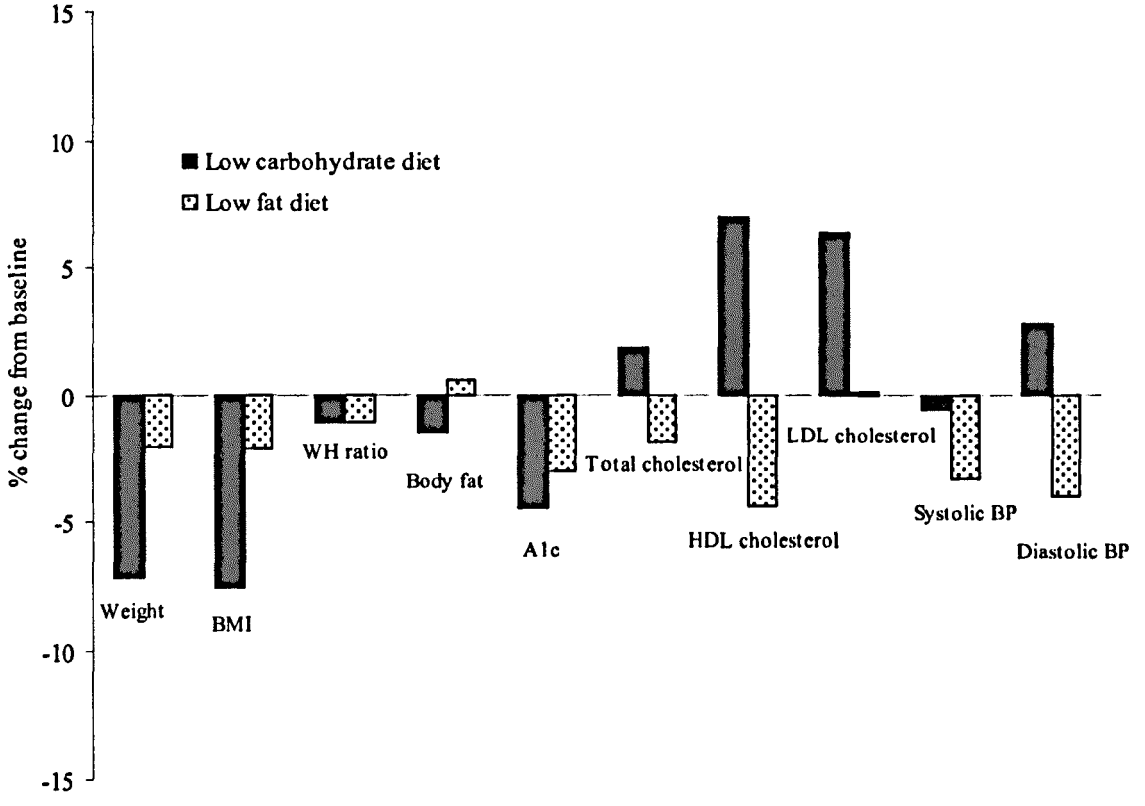


Table 3.7. Baseline and 3 months characteristics and absolute changes from baseline by dietary allocation and diagnosis of diabetes

Variable	Diabetes						Non-diabetes					
	Low carbohydrate (n = 6)			Low fat (n = 6)			Low carbohydrate (n = 6)			Low fat (n = 4)		
	Baseline	3 months	Change	Baseline	3 months	Change	Baseline	3 months	Change	Baseline	3 months	Change
Weight (kg)	99.7	91.7	-8.0	96.9	96.1	-0.8	91.1	85.3	-5.8	95.7	92.9	-2.8
BMI (kg/m^2)	36.5	33.4	-3.1	33.3	33.0	-0.1	33.7	31.5	2.2	37.3	36.2	-1.1
Waist:hip ratio	0.92	0.91	-0.02	0.93	0.93	0.0	0.87	0.87	0.0	0.88	0.87	-0.01
Body fat (%)	46.8	44.4	-2.4	41.8	42.2	0.4	47.9	47.4	-0.5	44.6	45.4	0.8
A1c (%)	7.2	6.8	-0.4	7.5	7.3	-0.2	6.1	5.9	-0.2	6.0	5.8	-0.2
Total cholesterol (mmol/l)	4.8	4.8	0	4.7	4.6	-0.1	5.5	5.7	+0.2	5.5	5.4	-0.1
HDL cholesterol (mmol/l)	1.24	1.32	+0.08	1.47	1.34	-0.13	1.32	1.40	+0.08	1.32	1.38	+0.06
LDL cholesterol (mmom/l)	2.70	2.94	+0.24	2.69	2.75	+0.06	3.46	3.60	+0.16	3.57	3.40	-0.17
Triglycerides (mmol/l)	1.8	1.2	-0.6	1.2	1.3	+0.1	1.6	1.5	-0.1	1.4	1.3	-0.1

Table 3.8. Baseline and 3 months characteristics and absolute changes from baseline by dietary allocation and diagnosis of diabetes

Variable	Diabetes						Non-diabetes					
	Low carbohydrate (n=6)			Low fat (n=6)			Low carbohydrate (n=6)			Low fat (n=4)		
	Baseline	3 months	Change	Baseline	3 months	Change	Baseline	3 months	Change	Baseline	3 months	Change
Blood pressure:												
Systolic	145	139	-6	138	133	-5	132	136	4	133	129	-4
Diastolic (mmHg)	76	78	3	86	83	-3	83	85	2	84	81	-4
Quality of life (%)	70	67	-3	51	56	5	51	66	15	49	65	16
Hunger (Lickert Scale)	4.0	4.2	-0.2	4.0	4.0	0.0	3.5	4.5	-1.0	5.5	4.0	1.5

Dietary intake is shown in Table 3.9. There were no significant differences in nutrient intake between the two groups at baseline. At the end of the study, dietary analysis showed a significant reduction in energy intake in both groups, and a greater reduction in calories in the low carbohydrate group (949 v 515 kcal/day, p=0.036). There were no differences in changes in absolute protein and fat intakes between the two groups, but there was a highly significant reduction in carbohydrate intake in the low carbohydrate group to 56.8g/day. This reduction in carbohydrate intake was reflected in significant changes in % energy from macronutrients.

Table 3.9. Changes in daily dietary intake over three months

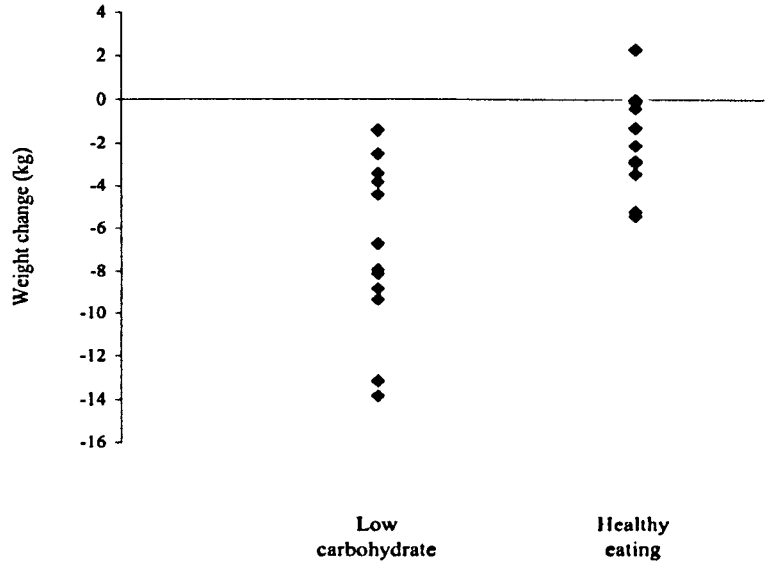
Variable	Baseline (n=26)	3 months		*p*-value
	Mean (SD)	Low carbohydrate (n=11)	Low fat (n=10)	(Changes in LC v LF)
Energy (kcal)	2130 (457)	1313 (205)	1593 (277)	0.036
Protein (g)	95.2 (18.4)	97.2 (18.9)	79.5 (16.6)	0.113
Fat (g)	92.5 (30.1)	69.3 (25.6)	62.7 (22.4)	0.634
Carbohydrate (g)	223.2 (62.0)	56.8 (26.5)	167.3 (60.4)	0.001
Protein (%energy)	18.4 (3.8)	31.1 (6.9)	19.8 (3.1)	<0.001
Fat (%energy)	38.6 (7.2)	46.2 (10.6)	34.4 (7.8)	0.033
Carbohydrate (%energy)	39.5 (6.8)	17.3 (9.7)	39.3 (12.8)	<0.001
Alcohol (%energy)	3.5 (5.0)	6.1 (9.3)	6.6 (6.6)	0.611

3.4.9. Discussion

3.4.9.1. Weight change

In common with other published work, this study shows a greater reduction in weight over three months in subjects allocated a low carbohydrate diet compared to a low fat regimen. Individual weight changes are shown in Fig 3.4. Weight losses in the low carbohydrate group ranged from 1.4 – 13.8kg over the three months of the study, with a mean weight loss of 6.9kg. The group allocated to the low fat diet showed a mean weight loss of 2.1 kg and a narrower range weight loss of 0.4 – 5.4 kg. Only 2 individuals in failed to lose weight, one of these showed no change in weight and another gained 2.4kg over the 3 months of the study. Both these individuals were allocated low fat advice. Significant weight loss in the low carbohydrate group was accompanied by significant loss of fat as measured by electrical impedance, the low carbohydrate group reduced body fat levels by 1.5% compared with an increase of 0.6% in the low fat group ($p=0.012$). It has been postulated that weight loss is more challenging in people with type 2 diabetes and that they lose less weight than people without diabetes (Wing, Marcus et al. 1987) but the results of this study do not appear to support these claims. Although this study was not powered to show differences between those with and without diabetes allocated to the 2 different regimens, Tables 3.7 and 3.8 show that there were no clinically significant differences between the diabetic and non-diabetic subjects.

Fig 3.4. Individual weight changes over 3 months. Low carbohydrate v low fat



3.4.9.2. Glycaemic control

Glycaemic control improved in both groups, with no difference between the two groups. This result may reflect the fact that this study included both subjects with and without diabetes. A sub-analysis showed that there was a clinical reduction in A1c in all the subjects with diabetes (-0.3%, p=0.07) but this failed to reach statistical significance. However, the non-diabetic subjects also showed a statistically significant reduction in A1c (-0.1%, p=0.038) although this is unlikely to be of clinical significance. Further analysis of all subjects, regardless of dietary allocation or diagnosis of diabetes showed a significant improvement in glycaemic control (-0.3% A1c, p= 0.012) over the three months of the study.

3.4.9.3. Lipid levels

There were no significant differences in the changes in lipid levels between the low carbohydrate and low fat groups, although HDL cholesterol significantly increased and triglyceride levels decreased in the low carbohydrate group over three months. Lipid levels were relatively low in the subjects with Type 2 diabetes and this is partly explained by the use of lipid-lowering medication, 5 of the 13 (38%) diabetic subjects were taking prescribed statin therapy compared with none of the non-diabetic subjects. There was no evidence of an adverse effect of a low carbohydrate diet on lipid profiles in either the diabetic or non-diabetic group.

3.4.9.4. Blood pressure

There were no significant changes in blood pressure during the 3 months of the study, either between the low carbohydrate groups and the healthy eating group, or in the diabetic and non-diabetic subjects. These data suggest that low carbohydrate diets do not increase cardiovascular risk over the short-term in people with and without diabetes.

3.4.9.5. Quality of life

There was a non-significant improvement in quality of life in both the low carbohydrate and the healthy eating group over the three months of the study, with no significant difference between the two groups.

3.4.9.6. Mode of action of low carbohydrate diets

This study has shown significant weight loss in those allocated a low carbohydrate diet compared to those allocated a low fat dietary plan, and the possible modes of action of low carbohydrate diets include appetite suppression associated with ketone production, large

initial weight losses associated with changes in body composition and diuresis and reduction in dietary intake caused by reduction in carbohydrate intake.

3.4.9.7. Appetite suppression, weight loss and ketones

The premise of low carbohydrate diets is that they are associated with increased ketone production and this supports weight loss by suppressing appetite. This supposition was investigated in this study by measuring appetite with a validated hunger-satiety scale (Roth 1993). This scale is based upon a visual analogue scale (VAS) and utilises a scoring system from 1-10, where 1 represents starving hungry and 10 absolutely full. The higher the number, the greater the satiety and lower numbers represent greater hunger. There were no significant changes in appetite or hunger in either the low carbohydrate or the low fat group over the three months of this study and no significant changes between groups. This suggests that appetite suppression is not the primary action of low carbohydrate diets. There was no association between ketone levels and hunger or appetite in the subjects in this study, further suggesting that ketones have little or no effect upon appetite at the levels recorded. Further analysis investigated the relationship between ketone levels and weight loss to determine if ketones do exert some effect.

All subjects in this study were required to measure ketone levels using a hand-held meter. Ketone measurements were taken four times daily at the following time-points; before breakfast (fasting levels), before the mid-day and evening meal and before bed. Measurements were taken at baseline while the subjects were on their usual diets and during the first week of the intervention, ketones levels were recorded daily. During the remainder of the study, ketones levels were measured and recorded four times daily on one day each week.

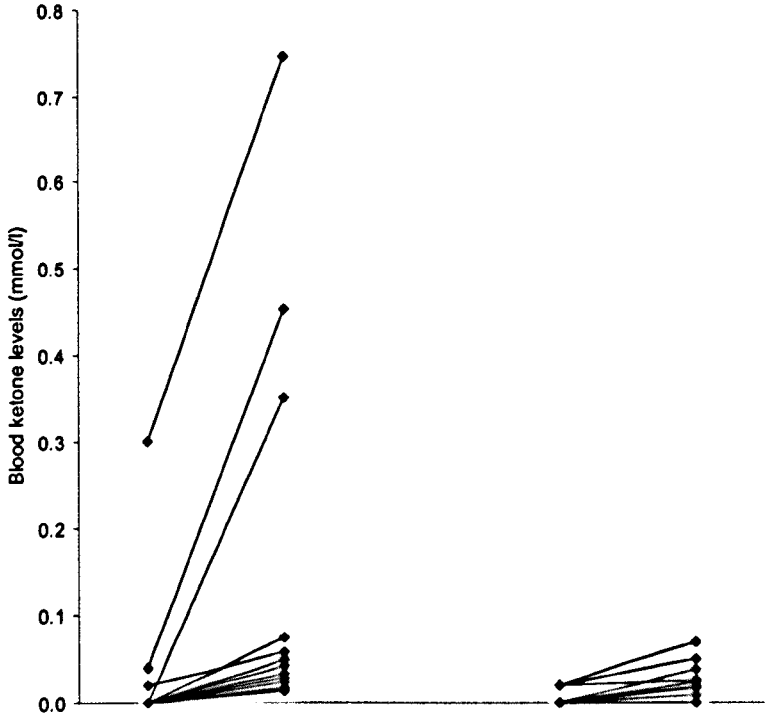
Baseline fasting ketone levels ranged from 0.0 – 0.2 mmol/l. A mean blood ketone level for each subject was calculated from all the available blood ketone levels measured during the 3-month study. Pearson's correlation coefficient was derived between mean blood ketone levels and weight loss from baseline at 3 months. Data were analysed by categorisation into % positive ketone tests and correlation was derived between weight loss and % positive ketone tests. 22 of 26 (84.6%) subjects completed the study at 3 months and provided a full record of ketone measurements. There were no significant differences in blood ketone levels either at baseline or over the 3 months of the study between the two dietary groups. Ketone production over three months was significantly higher in the low carbohydrate group from baseline ($p=0.05$), but failed to reach statistical significance when compared with the low fat group (Fig 3.5), and this can be largely explained by the three outliers in the low carbohydrate group who showed large increases in blood ketone levels over the course of the study.

Although there appeared to be more ketone production in the low carbohydrate group, it is interesting to note that 50% of the low fat group recorded positive ketones during the study period. As a proportion of the low fat group had also lost a significant amount of weight, the relationship between weight loss and ketone production was explored in all subjects, regardless of dietary allocation.

Further analysis of % positive ketone tests showed that the low carbohydrate group showed a higher frequency of positive tests than the low fat group (46.4 v 18.2%, $p=0.019$). An analysis including all subjects regardless of dietary allocation showed there was a significant correlation between mean blood ketone levels and weight loss over 3 months ($r = 0.72$, $p<0.01$) in all subjects (Fig 3.6). There was also a significant correlation between the frequency of positive ketone tests and weight loss ($r = 0.61$, $p<0.01$). Analysis of tertiles of

frequency of positive blood ketone tests and weight loss and showed that those in the lower tertile lost least weight [mean (SD)] with weight losses of 3.1 (2.8)kg, those in the middle tertile 4.9 (3.5)kg, and those in the upper tertile significantly more weight 11.7 (3.1)kg which was greater than either the weight losses of those in the lower ($p = 0.02$) or middle tertile ($p = 0.03$). `

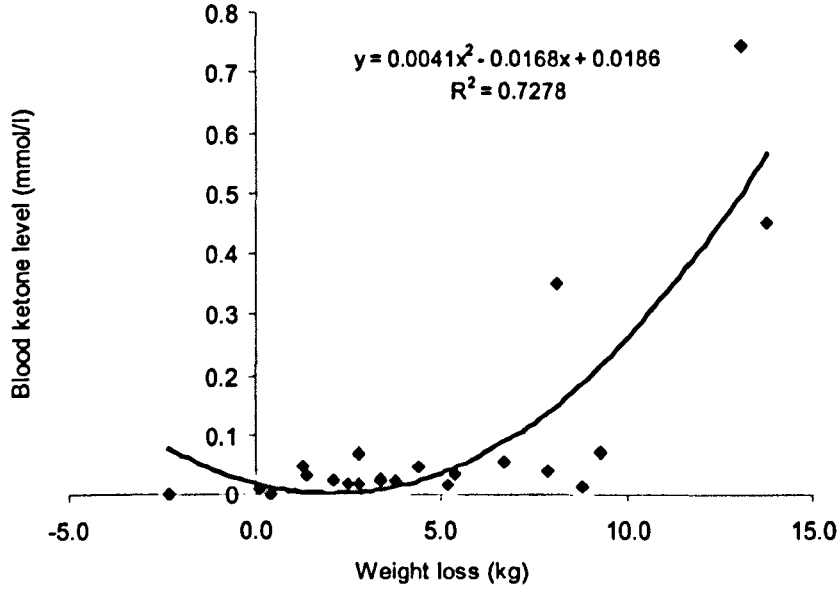
Fig 3.5. Changes in blood ketone levels over three months. Low carbohydrate v low fat



This analysis shows a positive association between self-reported blood ketone levels and degree of weight loss in people with and without Type 2 diabetes. Although it is assumed that

ketone production is higher in people adopting a low carbohydrate diet, this study fails to confirm that finding and suggests that mild ketosis is associated with weight loss regardless of carbohydrate intake. The only significant difference in blood ketone levels between those allocated a low carbohydrate diet and those adopting a healthy eating, low fat approach is that the frequency of positive blood ketone tests was higher in those adopting a low carbohydrate diet. This raises the question of a cut-off point for significant weight loss associated with the frequency of blood ketone testing. Further analysis revealed that subjects showing positive ketone tests 40% of the time had significantly greater weight loss that those who had lower frequencies (8.7 v 3.1 kg, $p = 0.01$). It could be postulated that recording blood ketones levels in individuals attempting weight loss may be a useful tool in identifying concordance and successful weight loss.

Fig 3.6. Correlation between blood ketone levels and weight loss in 22 individuals



3.4.9.8. Weight loss and fluid balance

The dramatic effects of low carbohydrate diets are often explained as fluid losses associated with utilisation of glycogen stores. Subjects in this study had body composition measured by electrical impedance to assess changes in body water and fat. There were no significant changes in body water during the three months of the study, but there were significant reductions in body fat in the low carbohydrate group, suggesting that the weight loss during the study was loss of fat and not fluid.

3.4.9.9. Dietary intake

The significant weight reduction in the low carbohydrate group appeared to be associated with a significant reduction in energy intake related to decreased carbohydrate intake and was not associated with an absolute increase in either protein or fat intake. Interestingly, subjects in both the low fat and the low carbohydrate group reported significant reductions in fat intake over the three months of the study, and this suggests that is possible to adopt a low carbohydrate diet without an increase in fat intake. This may reflect the fact that much of the fat eaten today is associated with carbohydrate for example in savoury snacks, biscuits, cakes and chocolate and that eliminating these foods reduces both carbohydrate and fat intake. There was no evidence from this study that either the low carbohydrate or the low fat diets resulted in any nutritional inadequacy, and there were no significant differences between the two diets for any vitamins or minerals. This may reflect the specific dietary advice given to the low carbohydrate group recommending 5 portions of low carbohydrate fruit and vegetables daily and the inclusion of 200ml milk.

3.5. Statistical analysis

In this set of analyses, there is the possibility of a type 2 error having occurred. A type 2 error may have arisen as the null hypothesis (in this case, that low carbohydrate diets have the same effect as low fat diets) has been accepted and this is, in fact, incorrect. There may well be differences between the groups, for example in A1c and lipid levels, although the data suggest that they are the same. A type 2 error may have arisen as the sample size is small and has been subject to multiple statistical tests.

3.6. Summary

This preliminary study shows that low carbohydrate diets are more effective than traditional low fat diets over the short term for weight reduction in overweight and obese people with and without diabetes. As has been shown in studies of people without diabetes, low carbohydrate diets induce weight loss by reduction in energy intake achieved by carbohydrate restriction. Although a review of studies has shown that low carbohydrate diets have no adverse effects and do no increase cardiovascular risk over the short term, little is known about the effects of these diets in the long term and further research is needed in this field. It appears that the primary mode of action for low carbohydrate diets on weight loss is that of energy reduction, but there may be an alternative mode of action to account for both weight reduction and improvements in glycaemic control. Low carbohydrate diets have a low glycaemic load, independent of the type of carbohydrate that is eaten. The diet used in this study, providing 40g carbohydrate from fruit, vegetables and milk products has an estimated daily glycaemic load of 15 (a low glycaemic load diet is considered to have a daily total <79). The effect of low glycaemic load diets, achieved by either adopting a diet of low

glycaemic index, or by reducing the total amount of carbohydrate eaten, may prove to be of benefit to people with diabetes.

Diabetes UK, the charity for people with diabetes, does not advocate low carbohydrate diets for weight loss and recommends that up to 60% of energy in the diet should be derived from carbohydrate. This study, illustrating the relative efficacy of low carbohydrate diets for weight loss for people with Type 2 diabetes, suggests that there may be room for more flexibility in the dietary interventions promoted for both weight loss and glycaemic control. This approach is explored further in Study 3, which investigates a more accomodating approach to carbohydrate in the diets of people with Type 1 diabetes. Flexibility in approach is also explored in Study 3 and in Study 2 with an investigation of the effects of lifestyle interventions delivered by means of video education rather than by traditional individual interview.

3.7. Publications

Two articles based upon work from Chapter 3 have been published and are included in the Appendices at the back of this volume. The first is entitled 'A low carbohydrate diet is more effective for reducing body weight than healthy eating in both diabetic and non-diabetic subjects'. This article was published in Diabetic Medicine in 2007 and can be found in Appendix 1. The second is entitled 'A review of low and reduced carbohydrate diets and weight loss in Type 2 diabetes' and was published in the Journal of Human Nutrition and Dietetics in 2008. A copy of this article can be found in Appendix 2.

Chapter 4

Health education for people with diabetes

4.0. Introduction

Webster's dictionary defines education as 'the process of educating or teaching', with a further definition of educating as 'developing knowledge, skills or character' (Merriam-Webster 2006). The Oxford English dictionary includes the following definition of education; 'The systematic instruction, schooling or training given to the young in preparation for the work of life; by extension, similar instruction or training obtained in adult age. Also, the whole course of scholastic instruction which a person has received. Often with limiting words denoting the nature of the predominant subject of the instruction or kind of life it prepares, as classical, legal, medical, technical, commercial or art education' (Oxford English Dictionary 1989). Both these definitions stress the purpose of education; that the individual should acquire some information, knowledge or training that was previously lacking. These traditional definitions of education suggest that the primary focus is on knowledge and teaching rather than on the learner, and that people undergoing an education process are expected to conform to the programme rather than the programme serving the needs of the learners.

Health education differs from education *per se* as it includes an implicit expectation that acquiring knowledge is not sufficient for improving health, and that some behaviour change is necessary to move the individual towards a state of optimal health. Health education has been defined as 'any combination of learning experiences designed to facilitate voluntary actions conducive to health' (Green and Kansler 1980). The theories and models underpinning health education have been summarised by the National Institute for Health in the US (NIH 2005)

and include the following models; social cognitive (learning) theory, theory of reasoned action and planned behaviour, health belief model, transtheoretical model, relapse prevention model, social support and ecological approaches.

4.1. Social cognitive theory

Most health education is based upon social cognitive theory with a central principle of self-efficacy. Self-efficacy reflects the estimate or personal judgement of an individual's ability and capacity to succeed in achieving specific goals. In addition, social cognitive theory includes the concepts of incentive and value from any health behaviour change.

4.2. Theory of reasoned action and planned behaviour

The theory of reasoned action and planned behaviour depends upon the individual attitudes and the influence of the social environment and includes the concept of perceived behavioural control. This concept is similar to that of self-efficacy.

4.3. Health belief model

The health belief model takes into account perceptions including perceptions of severity of any illness, individual susceptibility and the advantages and disadvantages of making a health behaviour change.

4.4. Transtheoretical model

The transtheoretical model is probably the best known and relates to readiness to change and embraces five key stages; precontemplation, contemplation, preparation, action and maintenance. The key to utilising this model effectively is matching the intervention to the stage of change.

4.5. Relapse prevention

Relapse prevention addresses the concept of adherence and examines the process of identifying high-risk situations and formulating solutions. It commonly involves four stages; indentifying the specific problem, brainstorming all possible solutions, evaluating each solution and committing to action.

4.6. Social support and ecological approaches

Social support and ecological approaches rely upon extrinsic models of health education and comprise the creation of supportive environments in both physical and emotional terms to support behaviour change. These approaches have been developed to support the individual behaviour change process relying upon intrinsic theories such as self-efficacy.

4.7 Health education for people with diabetes

There is some evidence that knowledge about diabetes is reflected in glycaemic control, but there is an acknowledged difference between knowledge and its relation to behaviour change (Panja, Starr et al. 2005). Outcomes from published studies vary greatly, with most education programmes for people with diabetes reporting greater effects on knowledge than on skills, behaviour change and metabolic effects (NICE 2005). GPs have reported that the biggest barrier to diabetes management is getting their patients to alter their lifestyles (Agarwal, Pierce et al. 2002) and this may well reflect that education is delivered within the parameters of the medical model (the health professional as the expert and in charge) rather than acknowledging the patients agenda and that lifestyle changes are completely under the patient's control (Meetoo and Gopaul 2004) . There is general agreement that it is necessary to provide lifestyle education as a package for people with diabetes, and a growing awareness that education should be combined with behavioural approaches in order to increase

knowledge and induce beneficial change in lifestyle. There has been very little work done on evaluating the effectiveness of lifestyle advice for people with diabetes. This is for two reasons, firstly that the provision of lifestyle advice is widely perceived as central to the diagnosis of diabetes and it is considered unethical to withhold this advice, making randomised, controlled trails impossible to conduct. Secondly, there is much confusion about whether to assess knowledge, behaviour change or metabolic outcomes from the delivery of diabetes education. Full evaluation of education should includes an assessment of cost effectiveness and there is evidence that people with diabetes who participate in diabetes education sessions have lower average costs than those who have little or no education (Duncan, Birkmeyer et al 2009). In addition, evidence shows that diabetes education is associated with cost saving and positive return on investment (Boren, Fitzner et al 2008). The total amount expended on diabetes education in the UK is unknown, but there is an imbalance between the amount spent on health education generally and the amount allocated by the food industry to promote processed foods. Globally, for every US dollar spent by the World Health Organisation (WHO) trying to improve nutrition of the world's population, $500 is spent by the food industry promoting processed food (Consumers International 2008). A recent health programme promoting fruit and vegetables in the US cost $9.55 million, compared with $11.26 billion promoting processed foods (CPEHN 2005).

Traditional, medically-centred models of health care delivery tend to concentrate upon the problem of 'non-compliance' and emphasise and evaluate the delivery of knowledge (Wolpert and Anderson 2001). It is becoming more widely accepted that this model should not be applied to chronic conditions like diabetes that depend upon self-management and that lifestyle advice for diabetes should be delivered using a more patient-centred approach

(Anderson and Funnell 2000). The rationale for a more patient-centred approach states that patients are constantly making decisions in their everyday life about food and physical activity that have a greater impact on their overall health than the decisions made by health professionals caring for them. If advice is framed from the patient's rather than the health professional's perspective, the patient may be more likely to regard that information as appropriate and should be more likely to incorporate it into their life. There is evidence that this more patient-centred approach can lead to significant improvements in self-management (Funnell and Anderson 2004).

The importance of structured education for people with diabetes has long been recognised, but until recently data have been lacking for the effectiveness of this education. Traditionally, diabetes education is delivered using a didactic, one-to-one model, but this is time consuming and requires a well-trained, motivated educator. This model is neither effective nor efficient and has led to frustration for educators and people with diabetes alike which resulted in an evaluation of and changes to the delivery of education during the 1990s. Amid growing recognition that the most effective approach was not an expert simply dispensing advice piece-meal to people with diabetes, there was a movement towards group education and utilisation of the expert experience of people with diabetes. At the same time, the Department of Health was developing the National Services Framework (NSF) (Department of Health 2001) for diabetes and recognised that self-management is the cornerstone for diabetes care (Department of Health 2003). In addition, Standard 3 of the NSF identifies structured education as a key intervention in encouraging partnership in decision-making, supporting self-management and helping people adopt and sustain a healthy life.

A review by the National Institute of Clinical Excellence (NICE 2003) supported the role of self-management as a fundamental part of diabetes care and went on to address the principles of education interventions. The Department of Health and Diabetes UK published a joint report in 2005 identifying the key components of structured education and stated that, from April 2006, structured education should be available to all people with diabetes in the UK (Diabetes UK 2005).

4.8. The development of education for people with diabetes in the UK

Recognition that education for people with diabetes should not consist solely of the medical professional dispensing advice, but should rely and build upon the existing knowledge and experience of the person with diabetes has led to a revolution in education for people with diabetes. This revolution has been supported by the Department of Health (DOH), the National Institute of Clinical Excellence (NICE) and Diabetes UK. The St Vincent Declaration, which was ratified by the World Health Organisation in 1991, first set goals for reducing the impact of diabetes and over twenty years later, the delivery strategy for the UK was published in 2003 in the NSF (Department of Health 2003). The NSF was established to improve services to people with diabetes by setting national standards, identifying the interventions and actions that will help meet each standard and proposing milestones by which service delivery will be measured. The NSF comprises twelve standards, ranging from preventing diabetes to managing complications, but the standard which underpins the education of people with diabetes is standard 3 which states: 'All children, young people and adults with diabetes will receive a service which encourages partnership in decision-making, supports them in managing their diabetes and helps them adopt and maintain a healthy lifestyle. This will be reflected in an agreed and shared care plan in an appropriate format and

language. Where appropriate, parents and carers should be fully engaged in this process.' It goes on to identify structured education as a key intervention to improve knowledge, blood glucose levels, body weight, dietary management, physical activity and psychological well-being in people with diabetes.

The National Institute for Clinical Excellence (NICE) published guidance on the use of patient education models for diabetes in 2003 (NICE 2003) and included a review of the education offered to people with diabetes in the UK. The review concluded that self-management is a fundamental part of diabetes care and that most people in England and Wales are offered some form of education at the time of diagnosis, but that this education varies greatly in content and length. However, this report also stated that very few individuals delivering education to people with diabetes had been formally trained for the purpose, and that there was a lack of evaluation of education. The conclusion was that there was insufficient evidence available to recommend a specific type of education or to provide guidance on the setting, delivery and frequency of educational interventions. The report did examine best practice and made some recommendations for interventions and recommended that they reflect established principles of adult learning, be provided by multi-disciplinary teams to groups of people with diabetes, ensure that the sessions are accessible to the broadest range of people by addressing differences in age, gender, culture, ethnicity and geography, that they are delivered in community or diabetes centres, use a variety of techniques to promote adult learning, specifically to engage with people by relating to their personal experience and are integrated into care in the long-term.

This report was followed by a publication recommending clinical guidelines for the management of both Type 1 (NICE 2004; NICE 2008) and Type 2 diabetes (NICE 2005),

specifically investigating effects of various interventions on glycaemic control. In terms of diabetes education, the report for people with Type 2 diabetes recommended that they receive a programme of structured education and that this should include modules designed to empower adults to participate in self-care. The report for people with Type 2 diabetes was the first to suggest the use of technology in delivering education and suggested that a variety of techniques could be utilised including picture charts, video techniques, computer packages, text messaging and e-mail tailored to the group or individual.

By 2005, when the Department of Health and Diabetes UK published a report from the patient education working group entitled 'Structured education in diabetes' (Diabetes UK 2005) it was well-established that education was fundamental to supporting self-management for people with diabetes. The DOH identified four key components to structured education programmes and they are as follows:

1. A structured, written curriculum including philosophy and theoretical principles
2. Trained educators
3. Quality assurance
4. Audit and evaluation of both biomedical and quality of life outcomes

More detailed criteria for education programmes were identified and included the recommendations that all programmes should be patient-centred and incorporate individual assessment, they should be reliable, valid, relevant and comprehensive, theory-driven and evidence-based, flexible and able to cope with diversity, able to utilise different teaching techniques, resource effective with supporting materials, written down (this included philosophy, aims and objective, timetables and detailed content) and finally that they should be subject to robust audit and evaluation.

These criteria apply more to structured education delivered in groups than to education delivered by leaflets or video, but the principles can be applied to all education. There are only three programmes in the UK which meet these standards, two designed for Type 2 diabetes (Diabetes Education and Self-Management for Ongoing and Newly Diagnosed - DESMOND and the X-PERT programme), and one for Type 1 diabetes (Dose Adjustment for Normal Eating – DAFNE). All three programmes have both shown a positive effect upon outcomes in terms of glycaemic control, weight, lipid levels and quality of life (DAFNE study group 2002; Deakin, Cade et al. 2006; Davies, Heller et al. 2008). The DAFNE programme for Type 1 diabetes is delivered to groups by specialist diabetes staff, predominately in secondary care. Both programmes for people with Type 2 diabetes offer education to groups and are delivered in primary care by trained health professionals, usually nurses and dietitians. Although these programmes have shown that they are effective, they do require resources and trained educators and may be beyond the reach of many primary health care trusts.

4.9. Innovation and delivery of education

The traditional model of delivering education is that of the one-to-one interview between the health care professional and the person with diabetes and is often based upon dietary histories and prescriptive advice. These didactic based approaches have been shown to produce modest improvements in outcomes, but patient education has been shown to be more effective if it has a behavioural element rather than being didactic (Brown, Lieberman et al. 1997). Both NICE and Diabetes UK suggest that innovative approaches may be needed to deliver education to people with diabetes (NICE 2003; Diabetes UK 2005). Patients with chronic disease including diabetes state that they would like information in as many formats as

possible and as early as possible after diagnosis (Corben and Rosen 2005). The challenges of instigating different approaches to education include the concept and design of suitable education programmes and evaluating the efficacy of education programmes. Two main studies were designed to address these issues; firstly Study 2 investigated the role of video education for people newly diagnosed with Type 2 diabetes, and Study 3 evaluated a structured, self-management education programme incorporating carbohydrate counting and insulin adjustment for people with Type 1 diabetes. Both Study 2 and 3 utilised novel, innovative methods to deliver education and both were subject to rigorous audit and evaluation to ensure that they both complied with the criteria set by NICE and Diabetes UK, and that they were shown to be effective in practice.

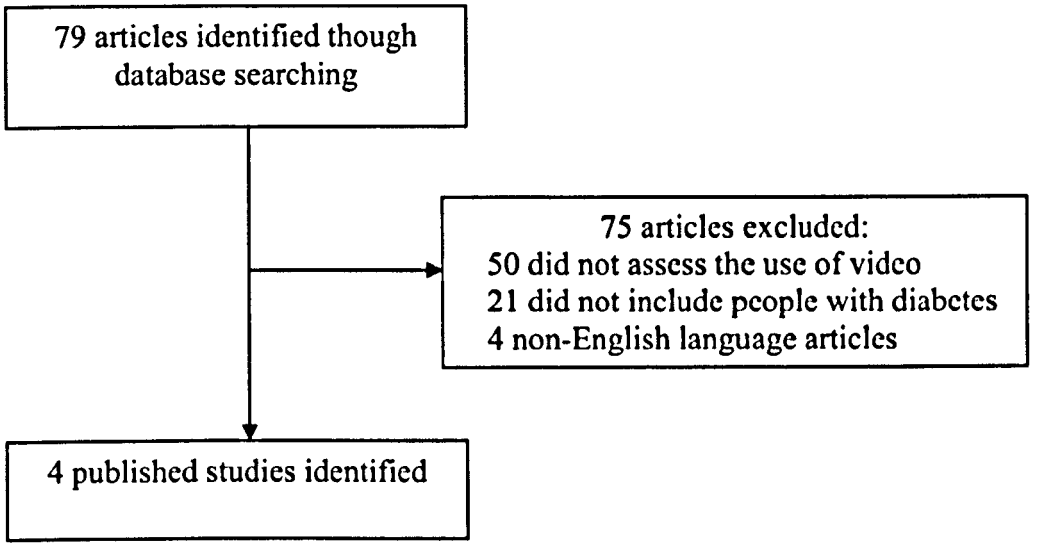
4.10. Rationale for innovative diabetes education

4.10.1. Video education and Type 2 diabetes

A variety of techniques have been suggested for providing health education for people with diabetes, including picture charts, video techniques, computer packages, text messaging and e-mail tailored to the group or individual (NICE 2005). These techniques are useful to support diabetes education provided by health professionals, and are not necessarily designed as stand-alone programmes. The published evidence for these innovative approaches is scarce. A recent systematic review of the contribution of teleconsultation and videoconferencing to diabetes care concluded that these are practical, cost-effective and reliable ways of delivering education to people with diabetes, but there is little evidence for benefit beyond that offered by more traditional approaches (Verhoeven, van Gemert-Pijnen et al. 2007) . Computer-assisted learning has been shown to have a positive effect on diabetes knowledge, but there is no evidence for weight reduction or glycaemic control (NICE 2005).

There are few studies examining the effects of video on knowledge or biomedical parameters in Type 2 diabetes, and most of these studies do not report metabolic outcomes. An electronic MEDLINE search in Pubmed, the Cochrane Library and EMBASE was undertaken from 1980 until December 2008 using the search terms *diabetes mellitus* and *video education*, see Fig 4.1.

Fig 4.1. Quorum flowchart of reviewing process for articles investigating video education and diabetes



Only four studies utilising video education for the treatment of people with diabetes were found (Wheeler, Wheeler et al. 1985; Brown, Lieberman et al. 1997; Day, Rayman et al. 1997; Gerber, Brodsky et al. 2005), and of these only two studies used video education for people with type 2 diabetes (Wheeler, Wheeler et al. 1985; Gerber, Brodsky et al. 2005). These two programmes consisted of a dietary education programme and general diabetes

education. The first, an evaluation of a computer-based diet education programme that provided meal-planning information by an individualised computer programme which was combined with an interactive videodisc system showed that knowledge increased significantly in the intervention group, but effects upon biomedical outcomes where not measured (Wheeler, Wheeler et al. 1985). A more recent randomised controlled trial investigating the effect of a computer multimedia system and which included audio/video sequences reported an increase in perceived susceptibility to diabetes complications but no significant change in either biomedical outcomes, self-efficacy or knowledge (Gerber, Brodsky et al. 2005). In addition, Fleming (Fleming, Simmons et al. 1995) reported a study in New Zealand examining the effects of a video on diabetes awareness, among local Maori and Pacific Island communities and reported that the video was highly rated and improved knowledge, but metabolic effects were not measured.

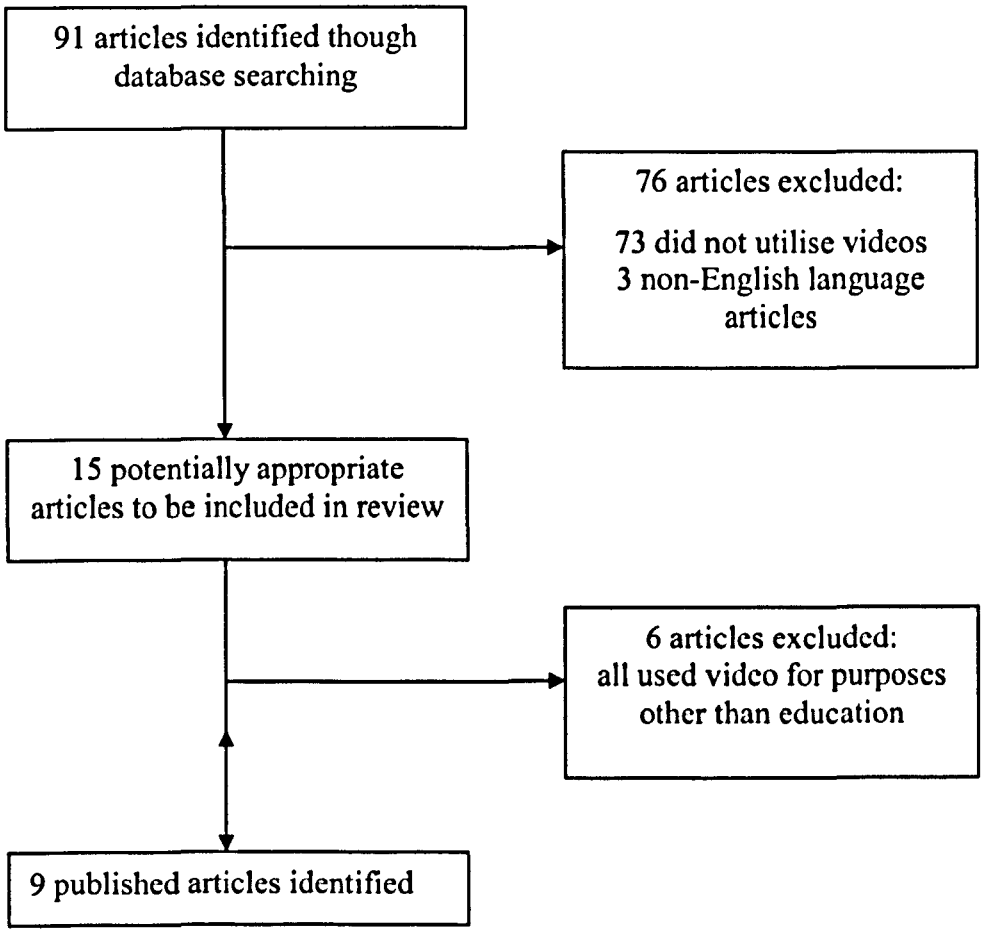
A search in both the American Diabetes Association (ADA) web-site and the National Institute of Health (NIH) web-site has shown that neither of these bodies produce videos designed for people with diabetes. Diabetes UK has recently introduced a 45-minute DVD intended for people with Type 2 diabetes and this is designed to answer many of the questions that surround the diagnosis of the condition. It uses an approach of advice from experts in the field intercut with case-studies and stories from people diagnosed with Type 2 diabetes. This DVD is available for sale, but the effectiveness of this approach to education has not been subject to audit or evaluation.

4.10.2. Evidence for video education and other chronic conditions

There is some evidence for the effects of video education on other chronic diseases, apart from diabetes. A further electronic search using MEDLINE in Pubmed, the Cochrane Library

and EMBASE was undertaken from 1980 until December 2008 using the search terms *chronic disease, illness* and *video education*, see Fig 4.2.

Fig 4.2. Quorum flowchart of reviewing process for articles investigating video education and chronic disease (excluding diabetes)



Nine studies were found reporting the effects of video education in the management of chronic diseases other that diabetes, but only four of these studies were designed as randomised controlled trials. The majority of studies do not report clinical outcomes, but use as an endpoint either changes in knowledge or behaviour. These nine studies show variable outcomes. Video teaching has been shown to improve:

- Patient satisfaction and reduce hospital expenditure in a preanaesthetic evaluation clinic (Yang, Wang et al. 2007)
- Knowledge of general health in native American people (Dick, Manson et al. 2007)
- Satisfaction in men with erectile dysfunction (Brock, Carrier et al. 2007)
- Knowledge in patients undergoing percutaneous cardiac interventions (Steffenino, Viada et al. 2007)
- Self-care behaviour leading to a reduction in signs and symptoms in people with heart failure (Albert, Buchsbaum et al. 2007)
- Reduction in pain rating and narcotic use in people with whiplash injury (Oliveira, Gevirtz et al. 2006)

A further study has reported that although video education can improve knowledge, it has little or no effect on adaptive behaviours in people with low vision (Goldstein, Dugan et al. 2007) and a report on managing asthma in South Asians in the UK (Hussein and Partridge 2002) also reported that video education was judged useful to both asthma sufferers and their families, but did not measure clinical outcomes.

4.10.3. Rationale for a trial of video education

Many recent reports have emphasised the importance of education for self-management of chronic conditions including Type 2 diabetes. There is evidence that patient education and knowledge can affect outcomes and that a patient-centred approach is beneficial. Didactic one-to-one teaching has been shown to be ineffective and demands huge resources. The increasing incidence of Type 2 diabetes and changes in health care structures have resulted in primary care taking responsibility for lifestyle education for people newly diagnoses with Type 2 diabetes. In the UK, there are now approximately 2 million people with Type 2 diabetes, and each of these should receive structured education at diagnosis and as an on-going procedure. Although the gold standard remains an individual appointment with a

diabetes specialist dietitian, lack of resources prevent this in Oxfordshire and other, more innovative, methods of supplying education should be investigated. Using new technologies to provide education for people with diabetes has not been fully explored or evaluated and this new approach involved investigating a video-based lifestyle education programme for people with Type 2 diabetes. This novel idea should increase access to education for people newly diagnosed with diabetes and offer equitable standards of care, including education about self-management, for all. Any new educational initiatives should be supported by sound evidence of efficacy and acceptability for people with diabetes and this was the rationale for the design and execution of this study.

4.10.4. Structured education and Type 1 diabetes

4.10.5. Evidence for structured education

There is wide recognition that education plays a central role in the management of Type 1 diabetes and has played a major part in the success of landmark studies designed to improve glycaemic control and reduce the risk of tissue damage (DCCT study group 1993; DAFNE study group 2002). Although education is regarded as a cornerstone in self-management of diabetes and is considered an integral part of treatment, there are few studies evaluating the overall effect of education and no systematic reviews specifically aimed at investigating self-management (NICE 2008).

The effects of education programmes for Type 1 diabetes was reviewed by a health technology assessment which reviewed four controlled trials and reported positive outcomes compared to normal care. End points differed between studies and included improvements in diabetes knowledge, glycaemic control, blood pressure, rates of diabetic ketoacidosis and hypoglycaemia (NICE 2002). However, most of these studies included small numbers of

subjects and had methodological limitations in design. Other randomised controlled trials have shown contradictory results, with one reporting improvements in glycaemic control (Lennon, Taylor et al. 1990) and another showing no effects of self-management education on either glycaemic control or quality of life (de Weerdt, Visser et al. 1991).

4.10.6. Rationale for trial of structured education

The day to day responsibility of diabetes control lies with the person with Type 1 diabetes; they are in charge of making the decisions about what and when to eat, how much insulin they inject and how much physical activity and blood glucose monitoring they undertake. In order to make appropriate choices about these various aspects of self-management, education is fundamental to enable the individual to acquire the relevant knowledge and skills to be successful in managing independently. Despite the recognition that education is the cornerstone in the treatment of diabetes, there is little evidence investigating the role of education in self-management. This study was designed to address this lack of evidence and investigated the effects of a structured education programme incorporating the concepts of carbohydrate counting and insulin adjustment for people with Type 1 diabetes.

4.11. Meeting the criteria for structured education

Both Study 2 and 3 were designed to meet the four key components for structured education (Diabetes UK 2005). These criteria are more relevant for teaching either individuals or groups as was the case for Study 3, but the basic principles can be applied to video education programmes. The four components including a structured curriculum with stated philosophy, trained educators, quality assurance and full audit were addressed.

4.11.1 Philosophy

The Thames Valley core philosophy for structured education states:

'Patients with diabetes have the right to take control of their care. All programmes delivered will enable patients with diabetes to develop skills, knowledge and the confidence to ensure that they can take responsibility for decisions they make regarding the daily management of their diabetes.'

In conjunction with the Diabetes Education Network, a national body operating under the umbrella of Diabetes UK, the core philosophy from the Thames Valley was further developed specifically for the healthy living programme and states:

'Diabetes is a complex condition, which is affected by, and can affect almost all daily activity. Most day to day decisions about lifestyle factors, including food choices and activity levels, which affect blood glucose levels are made by the person with diabetes. As such, people with diabetes are responsible for managing their condition (unless due to mental disability they are unable to make informed decisions). People with diabetes require knowledge and skills to enable them to understand the effects of lifestyle on their diabetes and vice versa, and how they can manipulate their diet and levels of physical activity to enable them to lead the lifestyle of their choice while maintaining stable blood glucose control and managing the associated risk factors of diabetes. The role of the health care professional is to provide a foundation to people with diabetes to enable them to develop realistic short term and long-term management goals, and to help them acquire the knowledge and skills necessary to achieve those goals.'

4.11.2. Core principles

The above philosophy was adopted for Studies 2 and 3 and was developed into core principles. These principles underpin the approach to health education adopted in the video education package and reflected the underlying philosophy. These core principles were developed over a series of workshops organised by the Diabetes Education Network. The core principles state that:

The person with diabetes has the right to expect the following from the education programme:

- Provision of non-judgemental, up-to-date, evidence-based information
- The development of practical skills to identify and address individual issues

The programme will achieve this by:

- Engaging with each individual by providing case-studies of people with diabetes
- Providing appropriate information to support decision-making
- Providing access to knowledge and skills needed to achieve self-care behaviour appropriate to that decision

On completion of the education programme, individuals will be able to:

- Utilise the information gained to formulate a self-management plan
- Identify and move towards personal targets and goals, whether biomedical or behavioural

4.11.3. Adult learning principles

The education programmes utilise a variety of learning theories including social learning theory (Bandura 1977), adult learning styles (Honey and Mumford 1982) and Lewin's cycle for adult learning (Kolb 1984).

4.11.3.1. Social learning theory

Social learning theory underpins the healthy living programme by utilising the concept that interaction between personal factors, behaviour and the environment dictate behaviour. Social learning theory focuses on the learning that occurs within a social context. It considers that people learn from one another and the environment and includes such concepts as observational learning, imitation and modelling.

General principles of social learning utilised in the education programmes:

- Observational learning. Participants can learn by observing and imitating the behaviour of others and the outcomes of this behaviour. This is the theoretical foundation for the use of case-studies as an educational tool
- Intrinsic reinforcement. Cognition plays a role in learning and mental states are important for learning to occur. A form of internal reward such a pride, satisfaction and a sense of accomplishment can provide positive reinforcement for behaviour change.
- Learning does not necessarily lead to behaviour change. Successful application of the modelling process to produce behaviour change includes addressing the concepts of attention, retention, reproduction and motivation. This theory supports the use of case studies.

4.11.3.2. Adult learning styles

Four main categories of adult learning styles have been identified:

- Activist. Relies on concrete experience, prefers doing and experimenting
- Reflector. Uses observation and reflection
- Theorist. Relies on abstract conceptualisation, wants to understand underlying concepts, reasons and relationships
- Pragmatist. Uses active experimentation, likes to try things out to see if they work

The education programmes address different learning styles by delivering information in various ways to match these different styles. This ensures that all individuals have the opportunity to learn in their preferred style.

4.11.3.3. Lewin's cycle of adult learning

This theory suggests that there are four stages of learning which follow on from each other:

- *Concrete Experience* - direct practical experience is followed by
- *Reflection* on that experience on a personal basis – what the experience means to person undergoing it. This reflection on personal experience may then be followed by
- *Abstract Conceptualisation* - the derivation of general rules describing the experience (comprehension), or the application of known theories to the experience and then to
- *Active Experimentation* - the construction of ways of modifying the next occurrence of the experience based upon the transformation of theory into practice and this leads in turn to the next *Concrete Experience.*

The education programme utilised this cycle by encouraging participants to reflect and learn from personal experience during the education.

4.11.4. Curriculum

4.11.4.1. Video education programme

Development of the curriculum of the video education programme involved identification of the knowledge and information required by people with diabetes. A small focus group of professionals (2 diabetes specialist nurses, 1 diabetes specialist dietitian, 1 specialist registrar and 2 volunteers with diabetes from the Oxford Patient Involvement Group) met and agreed the design and content of a form designed to elicit information from people with diabetes. This formed the basis of a pilot study aiming to identify the most important lifestyle issues for people with diabetes.

A small pilot study was conducted to investigate the preferences of people newly diagnosed with diabetes. Ten patients attending diabetes out-patient clinics at the Oxford Centre for Diabetes, Endocrinology and Metabolism were selected at random and asked to complete a form (Appendix 1) including specific questions and a box for free-text. Section 1 was designed to identify the areas of lifestyle that the patients considered the most important. All participants were encouraged to select as many categories as they liked from a selection. The number of patients, expressed as n (%), selecting each category is shown below.

Category	n (%)
Foods that affect blood glucose levels	10 (100%)
Foods that are related to heart disease	8 (80%)
Sugar	7 (70%)
Fruit and vegetables	8 (80%)
Fatty foods	7 (70%)
Salty foods	2 (20%)
Glycaemic index	1 (10%)
Alcohol	2 (20%)
Losing weight	10 (100%)
Physical activity (exercise)	9 (90%)

Section 2 asked the participants to record the three most important topics from the above list and from this most participants selected weight loss (90%), foods that affect blood glucose levels (100%) and physical activity (80%). Other topics selected once by 3 different patients were fatty foods, sugar and glycaemic index.

Section 3 asked participants for any other comments. Only 4 of the participants entered free text in the box provided and these comments are reproduced below:

'I'm not sure about all the pills I'm taking – it would be nice to know a bit more about these'
'You should talk much more about physical activity. My blood pressure is right down now since I started going to the gym'
'I think this is a very good idea – please could I have a copy of the videos?'
'If you can help me lose weight, that would be great!'

From the above feedback, a curriculum was developed based upon a mixture of expert opinion and a case-study approach. The feedback suggested that weight management, physical activity and the effect of foods on blood glucose levels were considered the most important topics, although the majority of patients were also interested in fatty foods and heart disease, sugar and fruit and vegetables. Most patients expressed little interest in the concept of glycaemic index and in salty foods and alcohol. The free-text comments suggested that there was patient support for the idea of video education. The patient who was unsure about his medication was given an individual appointment to discuss this as it was not seen a relevant part of this lifestyle programme. As a result of this survey, it was decided that three 10-15 minute videos would be produced and that they would be entitled 'Food choices', 'Physical Activity' and 'Weight Management' reflecting the participant's feedback. The video entitled 'Food choices' would include sections on carbohydrate foods that affect blood glucose levels, fruit and vegetables, low fat options, reducing sugar intake and would not address glycaemic index, salt and alcohol. The company employed to produce the videos, Joose TV, were involved in the design of the videos as they had extensive expertise in the field and they recommended a case-study approach.

Curriculum of 'Food choices' section

- The dietitian educator will introduce the concept of food and diabetes and explain the importance of diet. A specialist General Practitioner (GP) will offer a summary of basic principles of nutrition for people with diabetes.

- The role of sugar in the diet of people with diabetes will be explored and the concept of the ideal diet and quality of life will be introduced and discussed.

- The effect of different foods on blood glucose levels will be explained and a list of carbohydrate-containing foods will be provided

- The educator will explore the issue of a healthy diet and clarify the ingredients of a healthy diet for diabetes including recommendations for 5 portions of fruit and vegetables daily, moderate intake of starchy carbohydrate foods, moderate intake of low fat protein foods, low total and saturated fat intakes and inclusion of small amounts of sugary foods.

- All participants will be actively engaged throughout the programme by being encouraged to reflect on their experiences through the use of case studies of people with Type 2 diabetes who will share their experiences of dietary change.

Curriculum of 'Weight management' section

- The dietitian educator will introduce the concept of body weight and diabetes and explain the importance of weight management.

- A case study will be utilised to offer personal experience of weight loss. The case study will review the lifestyle changes necessary to induce weight loss.

- A review of the different methods to achieve weight loss will be described including adopting a healthy, low fat diet, calorie counting, joining a commercial weight loss programme

- All participants will be actively engaged throughout the programme by being encouraged to reflect on their experiences through the use of a case study of a person with type 2 diabetes who will share their experiences of weight loss.

Curriculum of 'Physical activity' section

- The dietitian educator will introduce the concept of physical activity and diabetes and two specialist GPs will explain the importance of increased physical activity and describe the benefits for blood glucose control, cardiovascular function, weight maintenance and quality of life.

- The type of activity will be discussed and the factors affecting choice of activity will be explored, including practical tips for increasing general daily activity.

- The recommended procedure for increasing physical activity will be explained and safety factors for people with diabetes will be clarified, including recommendations to check with a physician before starting exercise, wearing supportive footwear, never exercising when unwell and specific advice about preventing potential hypoglycaemia.

- All participants will be actively engaged throughout the programme by being encouraged to reflect on their experiences through the use of a case study of a person with type 2 diabetes who will share their personal experiences of increased physical activity.

4.11.4.2. Structured education for Type 1 diabetes

The curriculum for the structured education programme for Type 1 diabetes was developed in accordance with the recommendations of the Diabetes Education Network and is reproduced in full in Appendix 13.

4.12. Trained educators

It was considered of importance that the educator designing this programme should have recognised qualifications in both diabetes and education. In Oxford, all educators are required

to fulfil the following criteria:

- A professional, recognised medical qualification in the field of medicine, nursing or dietetics
- At least 2 years experience of the management and education of people with diabetes
- Currently employed at a specialist level, whether in primary or secondary care

The dietitian organising and delivering the programme holds State Registration and has 25 years experience of diabetes education. She also holds a recognised education qualification. All experts (hospital consultants, general practitioners and diabetes nurses) who appear in the video are experienced in diabetes and diabetes education.

4.13. Quality assurance

4.13.1. Video education programme

It is challenging to apply quality assurance to a one-off education session. Quality assurance commonly relates to the maintenance of the programme over time, and it was decided in this case to measure quality assurance by assessing the videos by the stated aims and objectives, the philosophy and the principles. This was a subjective process completed by all experts involved in the video production. Verbal feedback indicated that those involved in the process felt that the videos complied with the agreed principles.

4.13.2. Structured education programme for Type 1 diabetes

A quality assurance (QA) programme was developed for the structured education programme in conjunction with the Diabetes Education Network. As very little work has been done in this area, the QA process was designed to be trialled and re-assessed after an initial period of six months. The aim of the QA process was to ensure our local education programme for people

with Type 1 diabetes met the key criteria and matched the written philosophy, aims, objectives and curriculum. The objectives of the QA process were to demonstrate that programme educators met the documented philosophy and curriculum, to show that the programme provided the education it was designed to deliver and to ensure that the facilitation was of an appropriate standard.

The QA process was conducted by a trained educator from another site who attended the third week of the four-week programme. Week three was chosen for pragmatic reasons, firstly because all group participants were familiar with carbohydrate counting and insulin adjustment and secondly because it included a session exploring hypoglycaemia and most people with Type 1 diabetes were fully engaged during this session. The outside educator observed the complete session and completed a QA form for each of the topics covered. At the end of the session, the two educators delivering the course completed self and peer reflection forms and the outside educator then facilitated a reflection based upon all completed forms and any recommendations for action were agreed and recorded. The completed forms were then filed.

In addition, a dot test was undertaken during the hypoglycaemia session, which lasted 45 minutes. The aim of the session was to explore hypoglycaemia, and the dot test was devised to test the philosophy of the programme that patients are supported and encouraged to develop their own strategies to treat hypoglycaemia. The dot test was designed to assess the amount of time the subject or educator spends talking. The observer notes whether a participant or educator is talking at ten-second intervals throughout the 45 minutes session with the aim that the educators talk no more than 50% of the time.

4.14. Audit and evaluation

There were three areas subject to audit and evaluation including biomedical outcomes, well-being and diabetes knowledge. In addition, subjective evaluation of the videos and structured education programmes were collected from the participants.

Biomedical outcomes included glycaemic control measured by glycated haemoglobin (A1c), body weight and body mass index (BMI) and blood lipid levels. The video education study also included % body fat, blood pressure and insulin resistance measured by homeostatis model assessment (HOMA) (Levy, Matthews et al. 1998). In addition, the video education study also included measurement of dietary intake by validated 3-day food diary (Toeller, Buyken et al. 1997) and levels of physical activity by pedometer (Bravata, Smith-Spangler et al. 2007) were used to assess lifestyle changes. Well-being measurements included a general measurement of quality of life using a World Health Organisation questionnaire (WHO-5) (World Health Organisation 1998) and the EQ-5D health related quality of life questionnaire (Brazier 1993) in the case of the video education study and the Problem Areas in Diabetes (PAID) scale for the structured education programme (Polonsky, Anderson et al. 1995). Changes in diabetes knowledge in the video education study were assessed using a validated knowledge questionnaire, the ADKnowl (Speight and Bradley 2001).

Subjective feedback from the video education study was collected in two stages. Firstly, the videos were filmed, produced and edited into a rough cut and these were assessed by a panel of people with Type 2 diabetes for further refinement. A panel of 10 people with Type 2 diabetes were recruited from the OCDEM database and were given the videos to watch in their own time. They were asked to complete an assessment form (Appendix 7) and the videos were modified according to this feedback. The acceptance of the videos were marked using a

Lickert scale with 0 indicating lower acceptance and 10 higher acceptance. The scores were converted to % and are reported below:

Question	Score (%)
How useful were these videos?	82
What do you think of the idea of using videos?	93
What do you think of the presentation?	81
What do you think of the ease of use?	95
What do you think of the amount of information?	92

The majority of patients expressed an overwhelmingly positive response to the videos and felt that the amount of information contained in the videos was about right (although 2 patients felt that there was not sufficient detail about weight loss) and that this was an excellent method of presentation about the lifestyle issues of managing diabetes.

All patients expressed the opinion that the video entitled 'Food choices' was very clear and needed no amendment.

The case study in the video entitled 'Weight Management' produced a positive response, but some patients asked for further details of methods of weight management. In response to this, information about the web-sites of commercial slimming groups was added to the end of the video.

The video entitled 'Physical activity' was found to be the least acceptable. The majority of the panel felt that as the case-study was French, his experience may not be applicable to English people, and that the accent of the dubbed voice was difficult to understand. As it was impossible to film another case study, due to lack of resources, the French case study was retained, but the dubbed translation was changed to another voice which was easier to understand.

In addition, all subjects who took part in the study were invited to complete the assessment form and the results of this evaluation are presented in Chapter 5.

Subjective feedback from the structured education study for Type 1 diabetes was collected by means of a form based upon a visual analogue scale. The results are presented in Chapter 6.

4.15. Summary

Health education based upon established theories and models is recognised as fundamental for people with diabetes and this is supported by the Department of Health, who have recommended that structured education should be available to all people with diabetes. The traditional model of patient education with the expert dispensing advice is ineffective in inducing behaviour change, but there are few studies exploring the role of different approaches in delivering patient education. Most diabetes centres in the UK offer structured education either to individuals or in small groups, but these programmes do not always fulfil the key criteria for structured education and are not subject to rigorous evaluation as they are seen as part of routine care. Study 3 is designed to evaluate a structured self-management education programme for people with Type 1 diabetes incorporating carbohydrate counting and insulin adjustment and the results are described in Chapter 6.

Most education programmes rely on personal interaction and teaching and ignore innovative new technologies such as video and DVD education. The challenge is to produce an effective, resource-efficient education programme for people with diabetes that complies with Department of Health recommendations and which is subject to audit and evaluation. In response to this challenge, OCDEM designed and implemented a video-based education intervention for people with newly diagnosed Type 2 diabetes which was fully evaluated over

a 6 month period. Evaluation investigated both objective and subjective measures, including changes in metabolic outcomes, diabetes knowledge and quality of life. The results of this study are presented in Chapter 5.

Chapter 5

Video education for people newly diagnosed with Type 2 diabetes

5.0. Introduction

It is recognised that education plays a key role in development of self-management skills, but gaps in service delivery have meant that few people with diabetes have access to education programmes. One of the gaps in service has been identified as a lack of innovative, effective education programmes that are subject to robust evaluation and this led to the development of a video-based lifestyle education programme for people newly diagnosed with Type 2 diabetes.

5.1. Aims and Objectives

The aim of this study is to investigate the effect of a video-based lifestyle education programme on biomedical outcomes, diabetes knowledge and quality of life in people newly diagnosed with Type 2 diabetes

5.2. Methods

See Chapter 2 for full details.

5.3. Results

Results were collected for both objective and subjective outcomes. Objective outcomes included diabetes knowledge, quality of life and biomedical indices and are presented in the first section of the results and subjective outcomes included feedback on the videos by the subjects themselves using the evaluation sheet devised and applied in the pilot study.

5.3.1. Objective results

46 subjects were referred to the study and 42 agreed to take part. Figure 5.1 shows the consort flowchart for the subjects in the healthy living study. Of the 42 subjects randomised into the study, 21 were allocated to the video intervention group and 21 allocated to the control group. All 21 of the intervention group completed the study and 18 (86%) of the control group completed the study. The overall drop-out rate was 7%, meaning that 93% of the subjects recruited into the study completed it. This drop-put rate compares well with many other dietary or lifestyle studies where the attrition rate is typically much higher (Dansinger, Gleason et al. 2005).

Baseline characteristics of the subjects are shown in Tables 5.1 – 5.6 and include demographic details, physical characteristics, biochemical indices, dietary intake, quality of life and knowledge scores for lifestyle from the ADKnowl questionnaire. There were no significant differences between the groups for any baseline variables.

Fig 5.1. Consort Flowchart

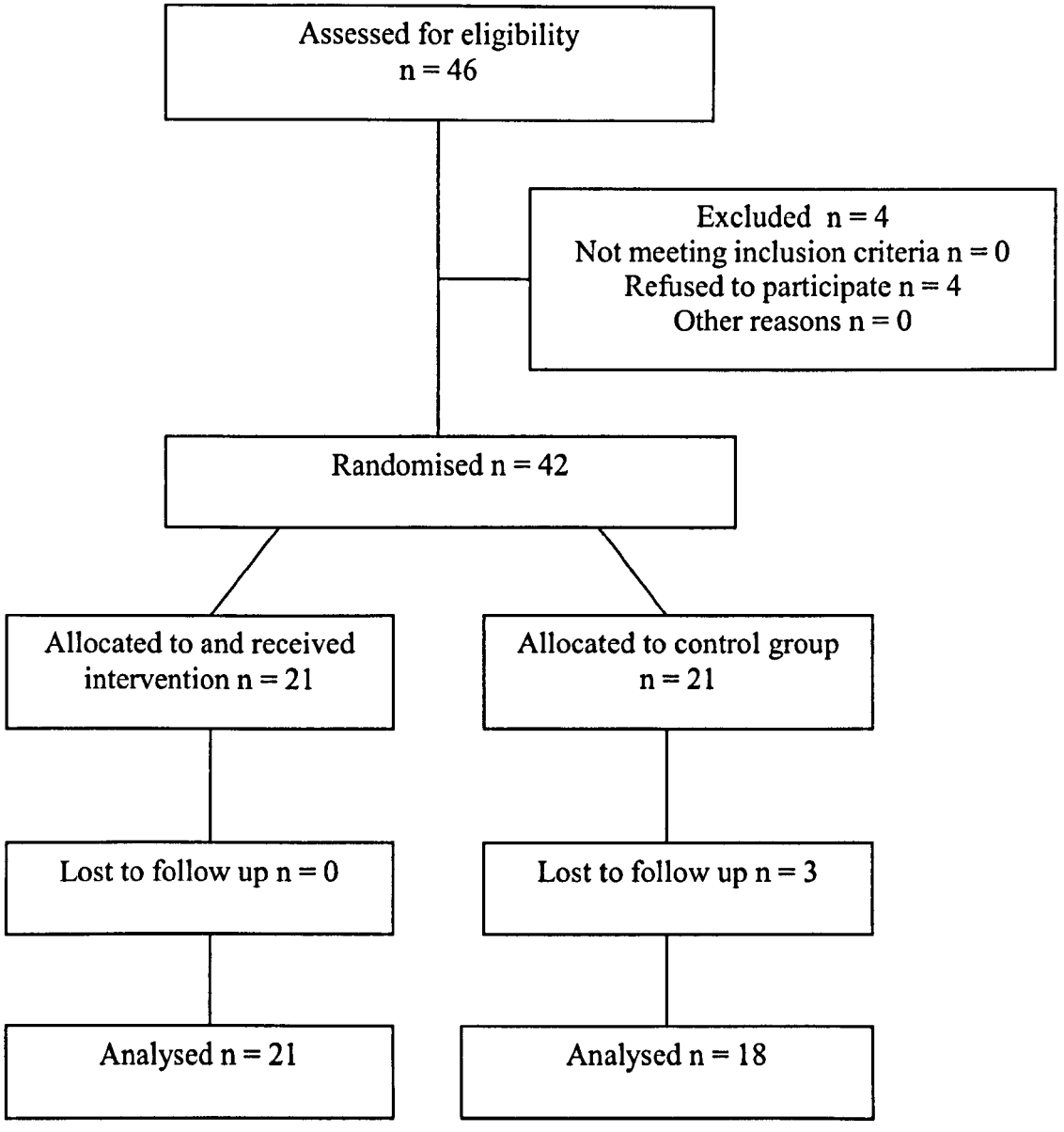


Table 5.1. Baseline demographic details of 42 people with newly diagnosed type 2 diabetes

Variable	All subjects Mean (SD)	Intevention Mean (SD)	Control Mean (SD)	*p*-value
Number	42	21	21	
Male/female	18/24	8/13	10/11	
Age (years)	60.8 (9.6)	58.6 (9.2)	62.9 (9.5)	0.141
Duration of diabetes (months)	3.6 (2.5)	3.8 (2.7)	3.3 (2.3)	0.539

Table 5.2. Physical characteristics at baseline

Variable	All subjects Mean (SD) (n=42)	Intervention Mean (SD) (n=21)	Control Mean (SD) (n=21)	*p*-value
Weight (kg)	89.5 (15.5)	90.7 (16.0)	88.3 (15.1)	0.616
BMI (kg/m^2)	31.3 (5.1)	31.9 (5.4)	30.6 (4.9)	0.410
Waist/hip ratio	0.94 (0.06)	0.93 (0.05)	0.95 (0.06)	0.314
% body fat	41.2 (9.2)	41.5 (9.0)	40.9 (9.5)	0.837
Blood pressure:				
Systolic	143 (21.1)	139 (21.2)	147 (20.7)	0.234
Diastolic	80 (12.3)	81 (13.1)	79 (11.7)	0.730

Table 5.3. Biochemical indices at baseline

Variable	All subjects Mean (SD) (n=42)	Intervention Mean (SD) (n=21)	Control Mean (SD) (n=21)	*p*-value
A1c (%)	7.4 (1.7)	7.6 (1.3)	7.2 (2.0)	0.555
Total cholesterol (mmol/l)	4.7 (1.2)	4.7 (1.2)	4.8 (1.2)	0.811
HDL cholesterol (mmol/l)	1.15 (0.34)	1.13 (0.28)	1.17 (0.39)	0.669
Triglycerides (mmol/l)	1.8 (1.0)	1.9 (1.1)	1.7 (0.9)	0.697
LDL cholesterol (mmol/l)	2.8 (1.0)	2.7 (1.1)	2.8 (0.9)	0.733

Table 5.4. Dietary intake and physical activity at baseline

Variable	All subjects Mean (SD) (n=42)	Intervention Mean (SD) (n=21)	Control Mean (SD) (n=21)	*p*-value
Energy (kcal)	1861 (439)	1825 (427)	1896 (458)	0.608
Protein (g)	82.0 (19.2)	81.0 (12.2)	82.9 (24.8)	0.748
Fat (g)	72.9 (25.3)	69.9 (22.0)	75.9 (27.4)	0.448
Carbohydrate (g)	219.0 (58.9)	213.8 (57.3)	224.1 (61.5)	0.576
Total sugars (g)	82.0 (33.0)	74.6 (29.8)	89.3 (35.1)	0.151
Saturated fat (g)	24.3 (10.2)	23.1 (10.7)	25.6 (9.8)	0.427
Monounsaturated fat (g)	23.8 (9.2)	22.1 (6.8)	25.6 (10.9)	0.223
Polyunsaturated fat (g)	15.8 (7.2)	15.1 (6.5)	16.5 (8.0)	0.539
Dietary fibre (g)	16.2 (4.7)	15.5 (3.5)	17.0 (5.6)	0.314
% energy from: Protein	17.9 (3.3)	18.1 (2.7)	17.7 (3.8)	0.632
Fat	34.8 (6.3)	34.1 (6.7)	35.5 (5.9)	0.469
Carbohydrate	44.2 (7.8)	44.0 (7.7)	44.5 (8.0)	0.833
Alcohol	3.1 (4.6)	3.8 (4.9)	2.3 (4.3)	0.313
Pedometer reading (steps/day)	5721 (3446)	6097 (3457)	5346 (3483)	0.498

Table 5.5. Quality of life at baseline

Variable	All subjects Mean (SD) (n=42)	Intervention Mean (SD) (n=21)	Control Mean (SD) (n=21)	*p*-value
QOL:				
WHO-5 (%)	65.8 (18.3)	64.6 (18.5)	67.0 (18.4)	0.666
EQ-5D VAS	75.6 (14.0)	76.6 (13.2)	74.6 (15.0)	0.657
EQ-5D Dimensions		**% reporting any problems**		
Dimension 1 (Mobility)	30.0	33.3	26.3	0.681
Dimension 2 (Self-care)	2.5	0.0	5.3	0.753
Dimension 3 (Usual activity)	17.5	9.5	26.3	0.419
Dimension 4 (Pain/discomfort)	38.4	40.0	39.8	0.862
Dimension 5 (Anxiety/depression)	30.7	23.8	38.8	0.232

Table 5.6. Diabetes knowledge at baseline

Variable	All subjects (n=42)	Intervention (n=21)	Control (n=21)	*p*-value
	% of subjects identifying correct answer			
Item 1 (General diabetes)	70.4	75.0	65.8	0.548
Item 2 (Physical activity)	51.1	55.0	47.3	0.686
Item 3 (Food and blood glucose)	41.9	44.3	39.4	0.901
Item 4 (Food)	68.4	71.7	65.2	0.666
Item 5 (Alcohol)	51.7	58.3	45.0	0.700
Item 6 (Tissue damage)	83.8	81.7	86.0	0.400
Item 7 (Regular examination)	84.5	87.0	82.0	0.222

The baseline characteristics of the 42 subjects entered into the healthy living study show that 43% were male, they were aged over 60 years and had been diagnosed with diabetes just over three months prior to recruitment. They were overweight with body weight of 89.5 kg and BMI 31.3 kg/m^2, had A1c levels of 7.4% and had both blood pressure and lipid levels within the normal range. Mean quality of life scores suggested no major problems, although depression or anxiety was reported by over 30% of subjects at baseline and this reflects the reported increased prevalence of depression in people with diabetes (Anderson, Freedland et al. 2001; Li, Ford et al. 2008). Diabetes knowledge was measured at baseline and Table 5.6 shows that although general diabetes knowledge including the effects of complications of diabetes and the need for regular examinations was good, the majority of subjects were unable to identify correctly the effects of different foods on blood glucose levels.

Six-month data are presented for 39 (93%) of the 42 patients randomised into the study. Fig 5.1 shows that 21 subjects were randomised to immediate video education and 21 to a control group. Data analysis was performed on all subjects completing the study and this applies to all subjects randomised to the lifestyle intervention and 18 (86%) of subjects in the control group. There were no drop-outs after randomisation in the video education group and this contrasts with three subjects allocated to the control group who failed to attend six-month follow-up after randomisation.

5.3.1.1. Biomedical indices

Changes at six months from baseline for both the video intervention and control groups are shown in Table 5.7. There were significant reductions in A1c, total cholesterol and LDL cholesterol at six months in the video intervention group and a significant increase in the amount of physical activity measured by pedometer, but no

significant change in the control group. Fig 5.2 shows % changes from baseline at sic months for the intervention and control groups. A comparison of absolute changes from baseline showed no significant differences in changes over six months between the groups (Table 5.8), despite the significant changes in the intervention group from baseline.

5.3.1.2. Dietary intake

Changes from baseline to six months in dietary intake in both the video intervention and the control groups are shown in Tables 5.9 and 5.10. There were no significant differences between intakes of any nutrients in either groups and no differences between the groups.

5.3.1.3. Quality of life

Changes in quality of life measured by WHO 5 Well-Being Index and the EQ-5D are shown in Table 5.11 and 5.12. There were no significant changes in general quality of life or in any specific areas of mobility, self-care, usual activity, pain or discomfort or anxiety or depression from baseline to six months follow-up and no significant differences between the two groups.

5.3.1.4. Diabetes Knowledge

Changes in knowledge of diabetes are shown in Tables 5.13 and 5.14. Knowledge was measured by the ADKnowl questionnaire, a validated questionnaire developed by Professor Clare Bradley and which can be adapted to match the specific areas of knowledge changed anticipated in a particular study. For the purposes of this study, general questions about diabetes were included together with specific questions about diet, physical activity and alcohol. Although there were no significant changes in overall diabetes knowledge in either the intervention or the control group from

baseline to six months, there was a trend for increased knowledge in the video intervention group and decreased knowledge in the control group (Table 5.15). Table 5.16 shows that when the changes in knowledge from baseline to six months were compared between the groups, there was a highly significant increase in overall diabetes knowledge in the intervention group ($p=<0.0001$).

Analysis of individual items shows that there were no significant changes in the two groups from baseline to six months for any of the seven different items, but when comparing changes from baseline between the two groups, there were significant differences between the groups for knowledge about food ($p=<0.01$) and the need for regular examinations ($p=<0.05$).

Table 5.7. Mean changes from baseline at six months: video intervention v control

Variable	Video intervention group (n=21) Mean (SD)				Control group (n=18) Mean (SD)			
	Baseline	6 months	Change	*p*-value	Baseline	6 months	Change	*p*-value
A1c (%)	7.6 (1.3)	6.8 (1.0)	-0.7 (1.4)	0.024*	7.2 (2.1)	6.5 (0.6)	-0.6 (1.8)	0.177
Weight (kg)	89.8 (16.0)	88.4 (16.5)	-1.4 (4.4)	0.165	87.8 (15.7)	88.0 (17.9)	0.2 (3.7)	0.801
BMI (kg/m^2)	31.7 (5.4)	31.3 (5.2)	-0.4 (1.7)	0.264	30.4 (5.0)	30.4 (5.6)	0.0 (1.3)	0.927
Waist/hip ratio	0.93 (0.05)	0.93 (0.06)	0.0 (0.03)	0.899	0.94 (0.06)	0.93 (0.07)	-0.01 (0.03)	0.195
% body fat	41.0 (9.0)	40.3 (9.2)	-0.8 (3.8)	0.381	40.8 (9.2)	40.3 (9.5)	-0.5 (4.6)	0.676
Blood pressure:								
Systolic	140 (21.7)	137 (19.7)	-3 (17.8)	0.505	147 (20.7)	150 (24.8)	2 (20.0)	0.625
Diastolic	81 (13.5)	79 (10.8)	-2 (12.1)	0.449	80 (12.7)	83 (13.7)	3 (11.7)	0.288
Total cholesterol (mmol/l)	4.7 (1.2)	4.2 (1.1)	-0.5 (0.8)	0.017*	4.7 (1.1)	4.5 (1.2)	-0.2 (1.1)	0.543
HDL cholesterol (mmol/l)	1.13 (0.29)	1.20 (0.30)	0.07 (0.18)	0.096	1.22 (0.42)	1.29 (0.47)	0.07 (0.19)	0.191
Triglycerides (mmol/l)	1.9 (1.1)	1.9 (1.1)	0.0 (1.0)	0.887	1.6 (0.7)	1.7 (0.8)	0.1 (0.5)	0.633
LDL cholesterol (mmol/l)	2.7 (1.1)	2.1 (0.8)	-0.5 (0.9)	0.018*	2.8 (0.9)	3.1 (2.1)	0.3 (2.0)	0.530
Pedometer reading (steps/day)	5140 (2727)	6401 (3343)	1266 (2526)	0.043*	5566 (4240)	4844 (4039)	-721 (3383)	0.439

* $p<0.05$

Table 5.8. Absolute changes from baseline at six months: video intervention v control group

Variable	Video intervention (n=21)	Control group (n=18)	*p*-value
A1c (%)	-0.7	-0.6	0.843
Weight (kg)	-1.4	0.2	0.223
BMI (kg/m^2)	-0.4	0.0	0.347
Waist/hip ratio	0.0	-0.01	0.416
% body fat	-0.8	-0.5	0.825
Blood pressure:			
Systolic	-3	2	0.416
Diastolic	-2	3	0.194
Total cholesterol (mmol/l)	-0.5	-0.2	0.347
HDL cholesterol (mmol/l)	0.07	0.07	0.939
LDL cholesterol (mmol/l)	-0.5	0.3	0.100
Triglycerides (mmol/l)	0.0	0.1	0.737
Pedometer reading (steps/day)	1266	-721	0.063

Fig 5.2. % change from baseline at six months, video intervention v control group

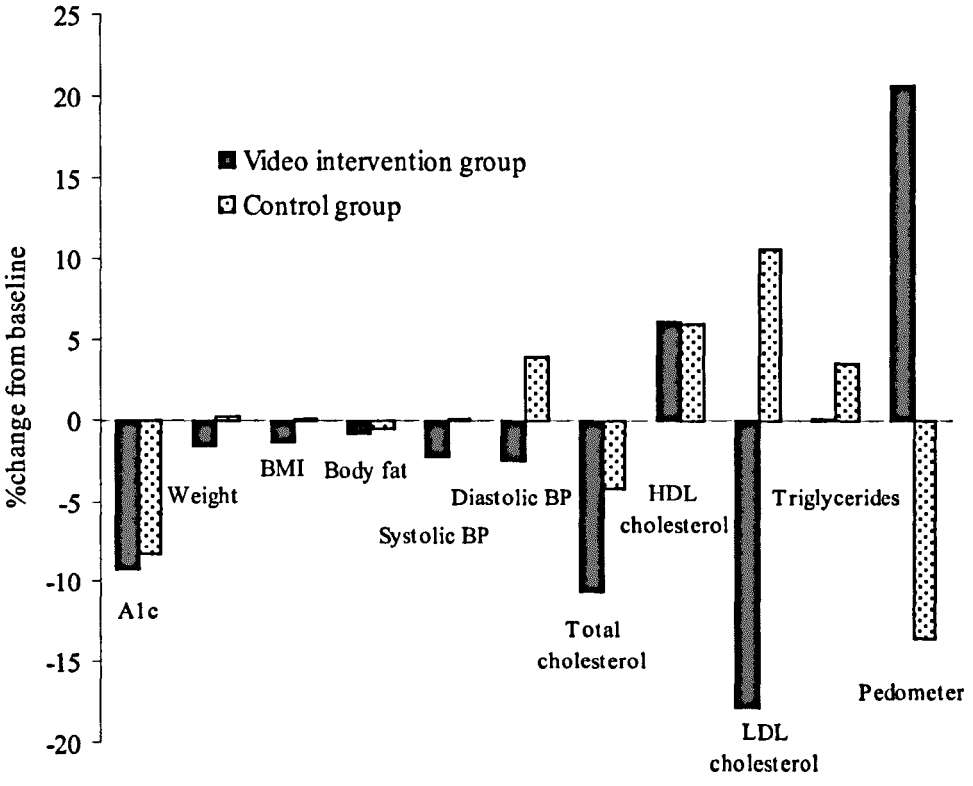


Table 5.9. Mean changes in dietary intake from baseline at six months: video intervention v control

Variable	Video intervention group (n=21) Mean (SD)				Control group (n=18) Mean(SD)			
	Baseline	6 months	Change	*p*-value	Baseline	6 months	Change	*p*-value
Energy (kcal)	1854 (431)	1813 (299)	-40 (532)	0.744	1970 (429)	1871 (390)	-98 (294)	0.187
Protein (g)	81.6 (12.4)	82.9 (14.3)	1.3 (19.6)	0.378	84.3 (25.8)	79.7 (19.0)	-4.6 (19.5)	0.349
Fat (g)	71.7 (22.4)	66.3 (20.9)	-5.4 (30.8)	0.456	79.4 (28.3)	70.5 (16.6)	-8.9 (25.8)	0.173
Carbohydrate (g)	215.9 (59.8)	211.4 (52.7)	-4.5 (61.8)	0.753	231.9 (61.9)	228.1 (62.1)	-3.9 (50.5)	0.756
Sugar (g)	71.9 (30.1)	73.9 (24.3)	2.1 (20.8)	0.669	92.4 (34.0)	91.5 (29.5)	-0.9 (24.0)	0.879
Saturated fat (g)	24.1 (10.7)	22.8 (11.0)	-1.3 (13.2)	0.664	26.0 (9.6)	23.8 (7.0)	-2.2 (10.0)	0.376
Monounsaturated fat (g)	22.6 (6.7)	21.7 (7.0)	-0.9 (8.6)	0.639	26.7 (11.2)	22.2 (6.2)	-4.4 (9.4)	0.070
Polyunsaturated fat (g)	15.4 (6.1)	12.7 (5.0)	-2.7 (8.5)	0.179	18.0 (8.1)	14.8 (4.8)	-3.2 (6.9)	0.073
Dietary fibre (g)	15.6 (3.3)	15.2 (5.3)	-0.4 (4.6)	0.716	17.9 (8.0)	16.1 (7.6)	-1.8 (5.2)	0.160
% energy from:								
Protein	18.0 (2.7)	18.4 (3.0)	0.4 (3.9)	0.635	17.1 (3.9)	17.2 (3.1)	0.1 (3.7)	0.913
Fat	34.5 (6.8)	32.7 (7.4)	-1.8 (8.2)	0.341	35.8 (6.4)	33.6 (5.1)	-2.2 (7.3)	0.235
Carbohydrate	43.6 (7.8)	43.5 (7.4)	-0.1 (5.6)	0.967	44.2 (8.9)	45.6 (8.1)	1.4 (7.2)	0.437
Alcohol	3.9 (5.1)	5.4 (8.5)	1.5 (5.5)	0.262	2.9 (4.7)	3.2 (4.5)	0.3 (2.8)	0.616

Table 5.10. Absolute changes in dietary intake from baseline at six months: video intervention v control group

Variable	Video intervention (n=21)	Control group (n=18)	*p*-value
Energy (kcal)	-40	-98	0.693
Protein (g)	1.3	-4.6	0.378
Fat (g)	-5.4	-8.9	0.714
Carbohydrate (g)	-4.5	-3.9	0.972
Sugars (g)	2.1	-0.9	0.879
Saturated fat (g)	-1.3	-2.2	0.828
Monounsaturated fat (g)	-0.9	-4.4	0.254
Polyunsaturated fat (g)	-2.7	-3.2	1.861
Dietary fibre (g)	-0.4	-1.8	0.382
% energy from: Protein	0.4	0.1	0.795
Fat	-1.8	-2.2	0.897
Carbohydrate	-0.1	1.4	0.501
Alcohol	1.5	0.3	0.458

Table 5.11. Mean changes in quality of life from baseline at six months: video intervention v control

Variable	Video intervention group (n=21) Mean (SD)				Control group (n=18) Mean (SD)			
	Baseline	6 months	Change	*p*-value	Baseline	6 months	Change	*p*-value
WHO 5 Well-Being (%)	64.0 (18.8)	63.8 (19.4)	-0.2 (13.2)	0.947	64.7 (19.5)	68.0 (19.2)	3.3 (15.0)	0.378
EQ-5D thermometer (%)	77.1 (11.5)	76.7 (15.8)	-0.4 (10.4)	0.876	75.5 (16.1)	77.2 (16.6)	1.7 (17.4)	0.706
	% reporting some problems							
EQ-5D 1 Mobility	35.0	25.0	-10.0	0.602	26.7	26.7	0.0	1.000
EQ-5D 2 Self-care	0.0	0.0	0.0	1.000	6.7	6.7	0.0	1.000
EQ-5D 3 Usual activity	10.0	15.0	5.0	0.799	28.6	21.4	-4.5	0.768
EQ-5D 4 Pain/discomfort	36.8	47.4	10.6	0.582	33.3	33.3	0.0	1.000
EQ-5D 5 Anxiety/depression	25.0	25.0	0.0	1.000	38.8	21.4	-17.4	0.264

Table 5.12. Absolute changes in quality of life from baseline at six months: video intervention v control group

Variable	Video intervention (n=21)	Control group (n=18)	*p*-value
WHO 5 Well-Being (%)	-0.2	3.3	0.456
EQ-5D Thermometer (%)	-0.4	1.7	0.668
EQ-5D 1 Mobility	-10.0	0.0	0.633
EQ-5D 2 Self-care	0.0	0.0	1.000
EQ-5D 3 Usual activity	5.0	-4.5	0.569
EQ-5D 4 Pain/discomfort	10.6	0.0	0.836
EQ-5D 5 Anxiety/depression	0.0	-17.4	0.323

Table 5.13. Mean changes in total diabetes knowledge from baseline at six months: video intervention v control

Variable	Video intervention group (n-21) % reporting correct answers			Control group (n=18) % reporting correct answers		
	Baseline	6 months	*p*-value	Baseline	6 months	*p*-value
All items (1-7)	66.8	74.3	0.121	60.7	56.4	0.439

Table 5.14. Absolute change in total diabetes knowledge from baseline at six months: video intervention v control group

Variable	Video intervention (n=21)	Control group (n=18)	*p*-value
All items (1-7, inclusive)	7.5	-4.3	0.6^{-5} *

* p=<0.0001

Table 5.15. Mean changes in diabetes knowledge by item from baseline at six months: video intervention v control

Variable	Video intervention group (n=21) % reporting correct answer				Control group (n=18) % reporting correct answer			
	Baseline	6 months	Change	*p*-value	Baseline	6 months	Change	*p*-value
Item 1 General diabetes	75.0	79.0	4.0	0.690	65.8	61.0	-4.8	0.841
Item 2 Physical activity	55.0	57.5	2.5	0.886	47.3	36.8	-10.5	0.685
Item 3 Food and blood glucose levels	44.3	52.1	7.9	0.620	39.4	37.0	-2.4	0.949
Item 4 Food	71.7	80.6	8.9	0.386	65.2	58.8	-6.4	0.604
Item 5 Alcohol	58.3	66.7	8.3	0.827	45.0	50.7	5.7	0.827
Item 6 Tissue damage	81.7	93.3	11.7	0.400	86.0	78.0	-8.0	0.400
Item 7 Regular examinations	87.0	96.0	9.0	0.055	82.0	80.8	-1.2	0.841

Table 5.16. Absolute changes in diabetes knowledge by item from baseline at six months: video intervention v control group

Variable	Video intervention (n=21)	Control group (n=18)	*p*-value
Item 1 General diabetes	4.0	-4.8	0.309
Item 2 Physical activity	2.5	-10.5	0.343
Item 3 Food and blood glucose	7.9	-2.4	0.165
Item 4 Food	8.9	-6.4	0.005**
Item 5 Alcohol	8.3	5.7	0.827
Item 6 Tissue damage	11.7	-8.0	0.100
Item 7 Regular examinations	9.0	-1.2	0.031*

* p=<0.05
** p=<0.01

5.3.2. Subjective analysis

Feedback about the videos was obtained by using an evaluation form designed and adapted from the pilot study. 17 of the 21 subjects (80%) allocated to the video intervention group completed and returned the evaluation form. Evaluation was by means of a Lickert linear scale and the responses were coded from 0% (negative) to 100% (positive). The general evaluations of the innovative approach utilising videos are given in Table 4.17.

Table 5.17. Overall evaluation of the videos

Question	0.....100		Mean score (%)
How useful were these videos?	Completely useless	Very useful	77.1
What do think of the idea of using videos to give this type of information?	Poor	Excellent	89.9
What do you think of the presentation of the videos?	Poor	Excellent	78.1
What do you think of the ease of use of the videos?	Poor	Excellent	83.2
What do you think of the amount of information in the videos?	Too little	Too much	59.0

These results show positive response to this approach to delivering information. A box asking for any general remarks about using videos for diabetes education was completed by 12 subjects and included the following statements:

1. I think the three videos provide useful information for early diabetes.
2. It was good to have 'live' patients talking about their experience of being diabetic. I thought them very believable. The presentation was very easy to follow. I liked the idea of headings followed by an explanation. The length and amount of information did not overwhelm you. I would find these videos good to watch about 1 month after being diagnosed - giving enough time to adjust but at a time I might need good advice and reassurance.
3. The tapes were very informative, short and to the point. I found them easy to understand and short enough to keep my attention. Well done!
4. They seem to me to be well done.

5. Well presented and professional looking program. All the information would be better and easier to access if all on just one video or DVD.

6. Would have thought all the information could have been put on one video. Would like to see more emphasis on water intake and its beneficial effects.

7. The information is not detailed enough, giving no real guidance as to how to proceed with any actual positive actions.

8. I found these DVDs very useful and interesting to watch.

9. Good idea but content extremely basic.

10. Very informative and well presented and confirms the information provided in the diabetes information provided by the practice nurse

11. A very good idea but they could have contained more info for each category. They seemed to just repeat the same message.

12. All had a very natural and pleasant way of delivering an important series of guidelines within an aid of living with diabetes.

The majority of responses were positive and confirmed the scores from the Lickert scale showing that this approach to health education is acceptable to most patients. The highest score was given for the idea of using videos to deliver this information, and this suggests that using this innovative format was very acceptable to all the subjects. Then lowest score from the overall evaluation was given for the amount of information contained in the videos and the free-text feedback suggests that more information on each topic could have been included with 3 subjects stating that they thought the content was very basic and that more details could have been included.

The second part of the evaluation form was designed to collect information about each separate video and subjects were encouraged to select and provide feedback about a topic of

their choice. 10 (59%) chose to evaluate the video entitled 'Food Choices', 4 (23%) chose 'Physical Activity', none chose 'Weight Management', 1 (6%) chose to evaluate all 3 videos and 2 (12%) did not complete this section of the evaluation form.

Results of the evaluation of the videos covering food and physical activity are given. The format was similar to that of the overall evaluation and asked similar questions with additional free-text boxes encouraging subjects to give their answers to the following questions:

1. Are there any changes you could suggest to improve this video?
2. Is there anything you would leave out?
3. Are there any topics you would like to see included n another video?

5.3.2.1. Evaluation of 'Food Choices' video

The results for the videos about food are shown in Table 4.18.

Table 5.18. Evaluation of the video entitled 'Food Choices'

Question	0.....100		Mean score (%)
What do think of the idea of using videos to give this type of information?	Poor	Excellent	95.3
What do you think of the presentation of the videos?	Poor	Excellent	86.1
What do you think of the ease of use of the videos?	Poor	Excellent	90.1
What do you think of the amount of information in the videos?	Too little	Too much	60.1

This table shows that the video approach was well received by all subjects, but that there was not sufficient detailed information given. This is supported by the free-text comments to the

questions about improvements to the video. 5 of the 10 subjects selecting the food video for evaluation completed the free-text box and their answers to each question are shown below:

Are there any changes you could suggest to improve the food video?

1. There is no idea of quantity - lots of small amount can be quite meaningless. What is a portion of fruit 1 grape or 100 grapes? Detail is needed.

2. More info about food management as the video only gives a general idea for eg amount of calories, portion etc.

3. This format could be used to give more detailed information. This could be menu driven so that users can choose content.

4. Expand more on meal tips and the idea for someone trying to make recipes for diabetics was good. Show more meals to show portion size and variety it stays better in your mind.

5. Slightly longer and therefore more of the useful content available.

Is there anything you would leave out of the food video?

All of the subjects stated that nothing included in the food video should be omitted and one added that if anything, more details could have been given. Another subject suggested using a more positive case study, where the person seemed happier.

Are there any topics you would like to see included in another video?

1. How to control diet on special occasions - eg Xmas, celebrations which include a set meal; travel and holidays. Also the effect other illnesses have on diabetes- eg vomiting and diarrhoea, coughs, colds.

2. More information about the beneficial effects of particular food like individual vegetables or herbs and seeds. The difference between type 1 and type 2 diabetes.

3. No- just more content on diet.

4. One more CD just on diabetes itself.

5. As a salutary measure - I think maybe a further educational message into the consequences of not heeding the advice proffered in these three videos.

5.3.2.2. Evaluation of 'Physical activity' video

Evaluation for the physical activity video is shown in Table 4.19. As only 4 subjects reported feedback on this topic it is difficult to draw conclusions from the results given. All 4 of these subjects provided feedback in the free-text boxes on the evaluation sheet and their responses are given below.

Table 5.19. Evaluation of the video entitled 'Physical Activity'

Question	0..... 100		Mean score (%)
What do think of the idea of using videos to give this type of information?	Poor	Excellent	86.4
What do you think of the presentation of the videos?	Poor	Excellent	65.0
What do you think of the ease of use of the videos?	Poor	Excellent	55.7
What do you think of the amount of information in the videos?	Too little	Too much	52.0

Are there any changes you could suggest to improve the physical activity video?

1. There will be patients watching this who do not use insulin (eg controlling diabetes by diet alone) and may not have the means to check their blood glucose levels before and after exercise, as suggested.

2. No, I thought it was pitched about right.

3. I would put all the information on just one DVD. All the information is relevant and interesting so would be useful.

4. More advice to people on alternative exercise, if patients have difficulty walking e.g. hip problems. More emphasis on the benefits of swimming as an alternative.

Is there anything you would leave out of the physical activity video?

All four subjects stated that there was nothing they would leave out of this video and one subject added; 'As it's presented to Oxfordshire patients it would be better filmed and presented in England. I had difficulty in understanding the accent of the people. They were however also very inspiring.' This comment related to the fact that the case study was French and spoke in French with an English voice-over translation added at the editing stage. The case-study was filmed in Paris.

Are there any topics you would like to see included in another video?

1. I think it would be good to explain a bit more of the underlying science in another video.

2. Warning signs of side effects.
3. May be a bit more on exercises for not so fit (elderly).
4. Could all three discs be incorporated onto a single disc?

5.4. Discussion

This intervention was designed to increase knowledge and improve outcomes for people with newly diagnosed Type 2 diabetes and included assessment of glycaemic control, body weight and cardiovascular risk factors. The results show that a short video intervention delivering lifestyle education significantly increased overall knowledge of diabetes compared to the control group, but that it had no significant effect on changes in biomedical outcomes or quality of life when compared to the control group. However, it is of interest that there were some significant positive changes from baseline in the intervention group compared to no change in any of the outcomes measured in the control group over the six months of the study.

The completion rate of subjects recruited into this study was high (93%) and compares well with many other dietary and lifestyle studies where the attrition rate is typically much higher

(Dansinger, Gleason et al. 2005). This suggests that lifestyle education delivered by means of video is acceptable to the majority of people with diabetes. This is further supported by subjective evaluation of the videos. Subjects receiving videos were asked to complete an evaluation form including visual analogue scales and free-text comments and all patients completed and returned this form suggesting that they had watched the videos. Reactions to this innovative approach to providing diabetes education were overwhelmingly positive with the subjects giving a mean score of over 90% for the idea of delivering lifestyle advice by means of video.

The apparent lack of difference between the control and the intervention group in terms of biomedical indices and quality of life may be due to a variety of reasons. Firstly, the sample size was small and this may well have resulted in a type 2 statistical error, producing a false negative result. The small sample size was due to problems associated with recruitment to the study. The power calculations had indicated that 80 subjects were necessary to show a statistically significant difference between the intervention and the control group, but it proved difficult to recruit this number to the study. The main reason for this was the introduction of the Quality and Outcome Framework (Department of Health 2005) in the UK during the period of recruitment. QOFs were designed to provide financial incentives for GPs to improve outcomes and meet targets, including glycaemic control and cardiovascular risk factors, in people with diabetes and health professionals in primary care proved reluctant to refer people with diabetes out of their care into this study.

Secondly, it has been demonstrated that the introduction of QOFs has improved treatment for people with diabetes. It is likely that both the control group and the intervention group received more intensive treatment than they otherwise might have obtained before introduction of QOFs, and this may have obscured any differences between the groups.

Fourteen (33%) subjects reported that they had been prescribed Metformin at diagnosis without a trial of lifestyle interventions as recommended by the then current NICE guidelines (NICE 2005). At six months follow-up, of the 39 completers, 12 (32%) were taking Metformin as single therapy, 10 (27%) taking a combination of Metformin and sulphonylurea and 1 taking triple therapy, leaving 16 (41%) subjects taking no glucose lowering agents. This intensification of treatment improved glycaemic control in this study and mean A1c values decreased significantly from 7.4% to 6.7% (p=0.009) over the study period, regardless of allocation to video education. The QOF also includes cardiovascular risk assessment and this is reflected by a significant reduction in total cholesterol from 4.7mmol/l to 4.3 mmol/l (p=0.04) and an increase in HDL cholesterol from 1.17 mmol/l to 1.24 mmol/l (p=0.03) in all subjects. Other parameters such as body weight, quality of life and diabetes knowledge are not assessed as part of the QOF and it is interesting to note that these indices showed no significant change for all subjects in this study.

Thirdly, most studies have shown that any intervention at diagnosis of Type 2 diabetes appears to be effective (UKPDS 1990; Davies, Heller et al. 2008) and that it may be that six months is too soon for any benefit of the education to show. There may be additional benefit of education in the long-term for people with Type 2 diabetes, but at present there are no studies to support this supposition.

5.4.1. Glycaemic control

Over the six month of the study, glycaemic control measured by A1c decreased in both the intervention and the control group (-0.7 vs -0.6%, ns) although only the intervention group showed a significant difference from baseline to six months. Most studies of people with newly diagnosed Type 2 diabetes show that glycaemic control improves after diagnosis. The UKPDS reported a reduction in A1c of 2% (from 9.1% to 7.0%) in three months following

diagnosis with all 5102 subjects receiving dietary advice alone (UKPDS 1990) and a more recent large-scale intervention trial, DESMOND (Diabetes Education and Self-Management in Newly diagnosed and On-going Diabetes), that was designed to deliver intensive education in primary care compared to standard care failed to show a difference in 842 subjects allocated to either the educational intervention or control group, although both groups showed significant reductions in A1c over the 12 months of the study (Davies, Heller et al. 2008). This may well suggest that, at diagnosis, people with Type 2 diabetes will improve glycaemic control by means of combination of standard lifestyle education and medication. However, Type 2 diabetes is characterised by progressive loss of beta cell function accompanied by a rise in A1c and it could be postulated that the benefits of intensive education may not be seen for many years. Most education programmes have short-term follow-up (6-12 months) and this may not be sufficient time for the benefits of education to be observed. In addition, at the time of recruitment for this study, the Quality and Outcomes Framework (QOF) was introduced in to General Practice and this provides incentives for reducing A1c in people with diabetes and this may account for the lack of difference in A1c between the two groups in this study.

5.4.2. Body weight

At entry to the study the majority of subjects were either overweight or obese (mean weight 89.5 kg, BMI 31.3 kg/m^2). Only 6 of the 42 participants (14%) were within the normal weight range (BMI 18-24.9 kg/m^2). There were no significant differences either within the groups or between the groups at six months follow-up. It has been established that weight loss is beneficial to people with Type 2 diabetes who are overweight or obese (Aucott, Poobalan et al. 2004) and for this reason, one of the lifestyle videos was entitled 'Weight management'. Weight loss is notoriously challenging for people with diabetes and there is evidence that people with diabetes find weight loss more difficult to achieve than those without diabetes

(Wing, Marcus et al. 1987). In addition, the majority of medication prescribed for Type 2 diabetes, with the exception of Metformin (which is weight-neutral), is associated with significant weight gain (UKPDS 1998). Despite this, the video intervention group reduced body weight by 1.4kg, compared with a weight gain of 0.2kg in the control group. The more intensive education delivered by the DESMOND programme (Davies, Heller et al. 2008) was associated with significant weight loss over 12 months (-2.98kg vs -1.8kg, p=0.027) and this may represent the importance of formal, structured education programmes for weight loss in people with newly diagnosed Type 2 diabetes.

5.4.3. Cardiovascular risk

There were no significant changes between the groups from baseline to six months in cardiovascular risk factors including lipid profiles, blood pressure and physical activity measured by pedometer, although total and LDL cholesterol was significantly reduced in the video intervention group, and they also showed an increase in number of steps recorded by the pedometer. Type 2 diabetes is associated with increased cardiovascular risk (Selvin, Marinopoulos et al. 2004) and this is reflected in abnormal lipid profiles. This group of newly diagnosed Type 2 subjects showed lipid levels and blood pressure within the normal range, suggesting that there was not much scope for improvement. These levels of cardiovascular risk may be a result of the QOFs, which specifically target lipid levels and blood pressure in people with diabetes, and this is reflected in the treatment of the subjects in the study as 62% were taking lipid-lowering medication and 69% anti-hypertensive agents at entry to the study.

5.4.4. Dietary intake

There were no significant changes in dietary intake either within or between the two groups. This may be reflected by the fact that all subjects had received dietary advice at diagnosis from their practice nurse before referral to the study and most had adopted a reduced fat diet.

However, the majority of the subjects reported a relatively low energy intake, and this may reflect the previously-reported phenomenon of a strong inverse association between reported food intake and BMI (Heitmann and Lissner 1995). However, although absolute food intake may not have been fully reported, it is likely that any change in intake may have been identified and this is supported by the fact that there was no significant change in either body weight or reported energy intake. One of the three videos was entitled 'Food and diabetes' and was designed to educate the subjects about the effect of carbohydrate foods on blood glucose levels and to encourage a reduction in fat intake. There were no differences between baseline and six months in either group and no differences between the two groups. This may be due to the relatively low fat intake in the subjects at baseline (<35% total energy from fat) and the fact that they had already received healthy eating advice from a practice nurse at diagnosis.

5.4.5. Quality of life

There were no significant changes in quality of life measured by either WHO-5 Well-Being Index or the EQ-5D either within or between the groups.

5.4.6. Diabetes knowledge

There were no differences between the groups at baseline for diabetes knowledge assessed by the ADKnowl questionnaire. Comparisons between baseline and six months in both groups showed non-significant improvements in knowledge in the video intervention group for all 7 items and non-significant deterioration in knowledge in the control group for 6 of the 7 items with the exception of alcohol. Comparisons of the changes between the two groups from baseline to six months showed a highly significant increase in overall diabetes knowledge in the video intervention group compared with the control group (p=<0.0001) and specifically in the areas of food (p=<0,001) and the importance of regular examinations (p=<0.05). These

results indicate that delivering education by means of a video intervention have a positive effect on diabetes knowledge in people newly diagnosed with Type 2 diabetes.

5.4.7. Subject evaluation

The response of the subjects to this innovative approach to providing diabetes education was overwhelmingly positive with the subjects giving a mean score of over 90% for the idea of delivering lifestyle advice by means of video. This acceptance is supported by the fact that only 3 subjects failed to attend their follow-up visit at six months and all 3 were in the control group; none of the video intervention group failed to attend their follow-up visit. Evaluation for the video education process was high for all aspects except the content of the videos, with some subjects stating they would have like more detail on all three topics although there were others who felt that the information content was sufficient.

This subjective evaluation suggests that innovative approaches to education are acceptable to people newly diagnosed with Type 2 diabetes, although people would welcome more detailed information, especially about how to relate theory to practice.

5.5. Summary

These results show that a short video intervention delivering lifestyle education to people newly diagnosed with Type 2 diabetes significantly increased overall knowledge of diabetes, especially about food and the need for regular examinations, but that it had no significant effect on biomedical outcomes, including glycaemic control, cardiovascular risk and body weight, or quality of life when compared to a control group. The lack of differences between the groups may have been caused by the fact that the sample size was too small, that the recently introduced Quality and Outcomes Framework had improved diabetes care in general practice and obscured any potential differences between the intervention and the control

group and that the positive effects of education may take more than six months to manifest themselves.

At present, there are no diabetes education programmes delivered by video that have been subject to any assessment, one produced by Diabetes UK provides information to people with Type 2 diabetes but has not been formally evaluated. This study has demonstrated that video-based interventions are highly rated by people with newly diagnosed Type 2 diabetes and that there may be a role for this type of education in the future.

Study 1 has shown that a low carbohydrate diet is effective for weight loss in people with Type 2 diabetes, and this study (Study 2) that a novel approach to lifestyle education increases knowledge and may have some effect on weight loss and physical activity. This suggests that educational approaches other than the traditional individual appointment may benefit people with diabetes, and that more flexibility in the amount of carbohydrate included in the diet may improve weight loss. These two components of innovative approaches to education and manipulating carbohydrate intake were combined for Study 3, investigating the effect of carbohydrate counting and insulin adjustment delivered in a group setting for people with Type 1 diabetes.

5.6. Publications

An article based upon work from Chapter 5 has been published and is included in the Appendices at the back of this volume. This publication is entitled 'An assessment of lifestyle video education for people newly diagnosed with Type 2 diabetes' and was published in the Journal of Human Nutrition and Dietetics in 2010. It can be found in Appendix 3.

Chapter 6

Carbohydrate counting, insulin adjustment and Type 1 diabetes

6.0. Introduction

Type 1 diabetes is associated with significantly increased mortality and morbidity (Diabetes UK 2004); a diagnosis of Type 1 diabetes is estimated to reduce life expectancy by more than 20 years (Department of Health 2001), although improvements in treatment have shown that life expectancy has improved over the past decade (Ioacara, Lichiardopol et al. 2009). Despite this, mortality rates remain up to five times higher for people with diabetes (Kanters, Banga et al. 1999).

Morbidity in people with Type 1 diabetes includes macrovascular (cardiovascular) disease and microvascular disease including kidney disease (nephropathy), eye disease (retinopathy) and nerve damage (nephropathy). Although cardiovascular disease is rare in people with Type 1 diabetes in the 30 years following diagnosis, after 40 years of exposure to diabetes, cardiovascular disease accounts for 30% of deaths. Diabetes is now the leading cause of end stage renal failure in the UK, with approximately 20% of people with Type 1 diabetes reaching end stage kidney disease. The risk of kidney damage increases with the duration of diabetes, after twenty five years of exposure the risk is 40-50% for both types of diabetes. Blindness is more prevalent in people who have Type 1 diabetes, and twenty years after diagnosis nearly all people with Type 1 diabetes will have some form of retinopathy. Amputation is a complication caused by damage to the nerves and blood vessels that serve the limbs. In the UK, diabetes is the second most common cause of lower limb amputation, and the most common cause of non-traumatic amputation (Diabetes UK 2004).

Improvements in treatment leading to tighter glycaemic control have been shown to significantly reduce the morbidity associated with Type 1 diabetes (DCCT study group 1993). The Diabetes Complications and Control Trial (DCCT) showed that intensive management of blood glucose levels resulted in significant reductions in the risk of tissue damage. The results of the trial showed that a reduction in A1c from 9% to 7% was accompanied by a 34% reduction in incidence of microalbuminuria, a marker for kidney damage, and the development and progression of retinopathy was reduced by 27% and 34% respectively. Although the evidence for intensive management of Type 1 diabetes is compelling, the DCCT achieved this through close supervision by health professionals of the subjects' dietary intake, insulin adjustment and management of physical activity. In addition intensive insulin management had some deleterious effects; significantly more hypoglycaemia and weight gain were recorded in the intensively managed group during the course of the study. The resources needed to achieve the results seen in the DCCT, the increased rates of hypoglycaemia and weight gain have presented barriers to the adoption of this approach in routine clinical care. As a result, studies exploring alternative approaches to managing blood glucose levels in people with Type 1 diabetes have been carried out (DAFNE study group 2002). These studies have postulated that alternative methods of education which emphasise self-management skills may be a better use of resources, and may be accompanied by less hypoglycaemia and weight gain. The process of education for people with Type 1 diabetes has been identified as needing further exploration and in the UK this has led to the concept of structured education for people with diabetes (Diabetes UK 2005). A full description of the development of structured education for people with diabetes is given in Chapter 4.

6.1. Content of structured education programmes for Type 1 diabetes

There is general agreement in the UK from the Diabetes Education Network that education programmes for people with Type 1 diabetes should include information about dietary intake, insulin dose adjustment, managing physical activity, hypoglycaemia and sick day rules.

6.1.1 Dietary intake

Traditionally, education for people with Type 1 diabetes aimed to match carbohydrate to a prescribed insulin dose at each meal or snack. Although there was recognition that people with Type 1 diabetes did not have to avoid carbohydrate entirely, there was also the recommendation that those using insulin to treat diabetes may need to consider amount and timing of carbohydrate foods (Nuttall 1980). People with diabetes were taught to identify carbohydrate-containing foods and were given various strategies to assess the amount of carbohydrate in different portions, including exchange lists which indicated the amount of food containing 10-15g carbohydrate (Franz, Barr et al. 1987). A prescription for the amount of carbohydrate to be consumed at each meal and snack was dispensed by the health professional and the person with diabetes was expected to adopt this. Different strategies were employed to facilitate the idea of carbohydrate control; the strategies used in the DCCT included healthy food choices, carbohydrate exchanges, carbohydrate counting and calculating total available carbohydrate (Anderson, Richardson et al. 1993). In the UK, the recommended approach for people with Type 1 diabetes was that of carbohydrate prescription by means of 10g carbohydrate exchange lists (Nutrition sub-committee of the British Diabetic Association 1982). This approach was driven by the medical model of focussing on the metabolic outcomes of diabetes management and especially glycaemic control, which contrasts with the patient's

agenda where the emphasis is on daily management of diabetes and the challenge of integrating treatment into daily life (Wolpert and Anderson 2001). One of the most challenging aspects of diabetes management from the patient's perspective is that of dietary restriction (Bradley and Speight 2002), and it has been suggested that changing the emphasis of dietary restriction to dietary freedom accompanied by intensive insulin management may improve both biomedical outcomes and quality of life (DAFNE study group 2002). The basis of this approach is that the person with diabetes assesses the amount of carbohydrate eaten at each meal and snack and injects insulin to match the amount eaten. This approach was first introduced in Germany and was shown to be effective in improving glycaemic control (Muhlhauser, Bruckner et al. 1987), and is the general strategy for most structured education programmes.

6.1.2. Insulin dose adjustment

Insulin dose adjustment for people with Type 1 diabetes depends upon individual testing and titration. The main aim of insulin treatment is to mimic insulin secretion in people without diabetes. Typically, this is achieved by one injection of long-acting background or basal insulin and injections of short-acting insulin before each meal (Williams and Pickup 2004). As insulin requirements are subject to large individual variation, there are no current published recommendations for insulin doses for people with Type 1 diabetes, although some authorities have produced guidelines in the past (International Diabetes Federation 1998; American Diabetes Association 2009). Algorithms for insulin treatment of Type 1 diabetes are challenging in use due to this individual variation, and as a result very few centres publish these, although some centres employ this technique (Texas Department of State Health Services 2010). Traditionally, people with diabetes take set doses of insulin at each meal time, the amount of which is calculated by trial and error, and this can result in large

fluctuations in blood glucose levels if differing amounts of carbohydrate are eaten form day to day. The emphasis of the new approach is to provide flexibility in the amount of insulin taken at meal times and, by definition, this will change from day to day and meal to meal.

6.1.3. Physical activity

Management of physical activity in people with Type 1 diabetes is challenging owing to the increased risk of hypoglycaemia. Increased physical activity is recommended for general health for all people with diabetes (Weltman, Saliba et al. 2009), although for people with Type 1 diabetes the recommendation is made in terms of reducing cardiovascular risk rather than improving glycaemic control (Kavookjian, Elswick et al. 2007). Published reviews and recommendations state that the altered physiological response in diabetes means that more thought must go into preparation for exercise in people with Type 1 diabetes (Gallen 2006; Lumb and Gallen 2009). Before exercise, it is recommended that blood glucose levels should be between 7-12 mmol/l. If levels are <7 mmol/l, extra carbohydrate should be taken. In the absence of ketosis and with glucose levels >12 mmol/l glucose replacement during exercise should be delayed. In the presence of ketosis, exercise should be avoided. It is recommended that blood glucose levels are monitored before, during and after exercise at thirty minute intervals to establish an individual's blood glucose response to exercise.

To prevent hypoglycaemia during exercise, additional carbohydrate is advocated as needed. As a general guide, for moderate intensity endurance activities, high glycaemic index carbohydrate should be consumed after thirty minutes of exercise at a rate of up to approximately 1g/kg/hr. Lower intensity activities or intermittent high

intensity activities are likely to require smaller rates of carbohydrate supplementation. In addition, adequate hydration is essential.

6.1.4. Hypoglycaemia

Hypoglycaemia is generally accepted as a blood glucose level <3.5 mmol/l (Amiel 2009), although in clinical practice it is recommended that any blood glucose level <4.0 mmol/l is treated as hypoglycaemia (American Diabetes Association (ADA) Workgroup on Hypoglycemia 2005). Many people with Type 1 diabetes find hypoglycaemia challenging to manage and struggle to establish the balance between improved glycaemic control and increased hypoglycaemia (Heller 2008). The DCCT showed that reducing A1c levels was accompanied by an increase in hypoglycaemia (DCCT study group 1993), but subsequent studies relating insulin to carbohydrate intake have shown no increase in hypoglycaemia with improved glycaemic control (Muhlhauser, Bruckner et al. 1987; DAFNE study group 2002).

6.1.5. Sick day rules

Illness and infection in people with Type 1 diabetes is frequently associated with raised blood glucose and ketone levels and if left untreated can lead to diabetic ketoacidosis (DKA). DKA is correlated with significant morbidity and mortality and requires close monitoring during episodes of illness (Weber, Kocher et al. 2009). To support the management of illness in people with Type 1 diabetes, general recommendations have been devised and these are commonly known as 'sick day rules' (Laffel 2000; Campbell and Alford 2006).

6.2. Process of structured education

A review of the education process is provided in Chapter 4 and the principles of structured education as recommended by both the Department of Health and Diabetes

UK have been adopted by many education programmes in the UK (DAFNE study group 2002; Deakin, Cade et al. 2006; Davies, Heller et al. 2008). All education programmes are required to comply with the four criteria for structured education programme including a structured, written curriculum with explicit philosophy and theoretical principles, utilising trained educators, a process of quality assurance and audit and evaluation of both biomedical and quality of life outcomes.

6.3. Summary

It appears that there are many barriers to improving glycaemic control in people with Type 1 diabetes, including lack of resources, increased hypoglycaemia and weight gain. Structured education programmes may improve outcomes by promoting self-management skills. This chapter reviews published literature and evaluates the available data and then describes the application of a structured education programme for people with Type 1 diabetes in clinical practice.

6.4. A review of structured education programmes for people with Type 1 diabetes

6.4.1. Background

There is unequivocal evidence that improved glycaemic control in people with Type 1 diabetes reduces both the risk and progression of tissue damage associated with hyperglycaemia (DCCT study group 1993). However, although targets of 6.5-7% for A1c levels are now routinely recommended (American Diabetes Association 2009), many people find this challenging to achieve in practice. For example, the DCCT reported that only 5% of the intervention group were able to maintain A1c at target levels throughout the study period of 6.5 years, despite the level of support and advice that was available (DCCT study group 1993). Unpublished data from Diabetes UK has reported that in 2000 the mean A1c of people with Type 1 diabetes was 8.6%.

There are a number of factors that may explain suboptimal diabetes control including inflexible insulin therapy with associated hyper and hypoglycaemia, lack of provision of appropriate healthcare by the diabetes team and the lack of education and associated self-management skills of people with diabetes (Davies 2004).

6.4.2. Insulin therapy and hypoglycaemia

Strong evidence for optimal insulin therapy for Type 1 diabetes is lacking, but most centres now recommend a basal prandial regimen with one injection of basal (background) analogue insulin, often taken at night, and three or more injections of rapid-acting analogue insulin (prandial) with meals and snacks. There is little evidence that this regimen is more effective that two injections daily of mixed insulin, but there is some evidence that using analogue insulins rather than human soluble insulin can significantly reduce both A1c and hypoglycaemia (Jacobsen, Henriksen et al. 2009).

The main obstacle to reducing A1c for people with Type 1 diabetes is the fear of hypoglycaemia (Davis and Alonso 2004). Reducing A1c levels is associated with increasing insulin doses and the increased risk of hypoglycaemia, and patient's perceptions of this increased risk compromises glycaemic control (Gonder-Frederick, Clarke et al. 1997).

6.4.3. Healthcare provision

Healthcare professionals have a responsibility to provide optimal medical and educational advice to their patients. Healthcare professionals usually formulate advice based upon evidence from clinical trials and concentrate on biomedical outcomes and medical management, rather than the perspective of people with Type 1 diabetes, who

are more concerned with the day-to-day concerns of living with diabetes (Wolpert and Anderson 2001).

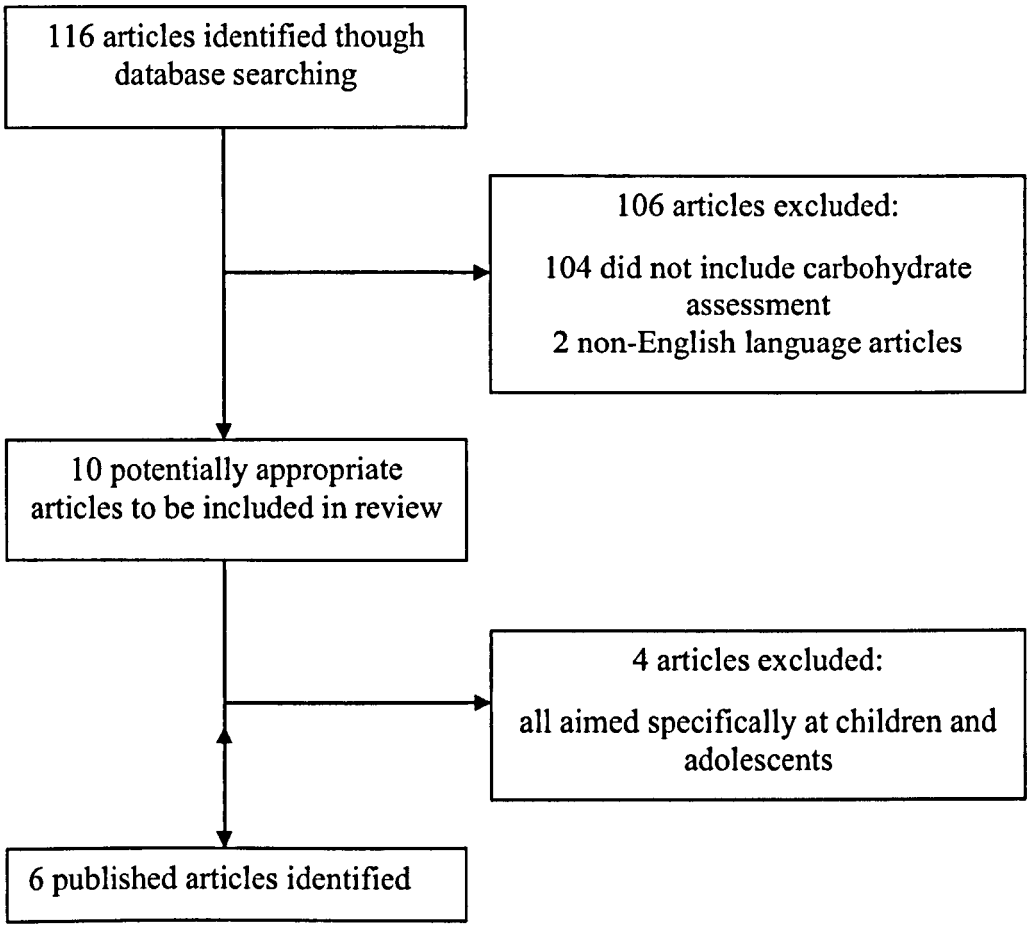
6.4.4. Education and self-management

Most authorities agree that self-management is fundamental for success for people living with Type 1 diabetes (DAFNE study group 2002). However, there is little evidence for the most effective method of delivering education to support and encourage self-management skills. It has been established that didactic teaching is effective in clinical trials, but is no longer effective once the trial ends. This has been demonstrated in the DCCT, where the intervention group, who received intensive insulin and education during the study, showed a rise in A1c once the study had finished and the education was no longer available (2002). Alternatives to didactic teaching include structured education incorporating dietary freedom, carbohydrate assessment and intensive insulin management. This approach was first adopted in Germany (Muhlhauser, Bruckner et al. 1987) and has since been trialled successfully in the UK (DAFNE study group 2002).

6.4.5. Methods

A literature search was undertaken to identify studies assessing structured education including carbohydrate counting and insulin adjustment in people with Type 1 diabetes. An electronic search was performed using MEDLINE (1966 – March 2009), EMBASE (1988 – March 2009) and the Cochrane Central Register of Controlled Trials (1991 – March 2009) using the search terms *structured education, Type 1 diabetes, carbohydrate counting* and *insulin adjustment*, see Fig 6.1. All studies relating to intervention trials of structured education in people with Type 1 diabetes were included.

Fig 6.1. Quorum flowchart of reviewing process for articles investigating video education and diabetes



Six studies were identified and are included in this review. Two of the studies were designed as randomised controlled trials, one as a delayed randomised controlled trial (DAFNE) and the other with block randomisation (BITES). Three other studies were designed as intervention trials and the remaining study was a retrospective audit. Results for the five studies are reported at intervals ranging from six weeks to one year. Table 6.1 summarises the characteristics of the six published studies.

Table 6.1 Details of structured education programmes for people with Type 1 diabetes

Programme name	Subjects (Number)	Age (years) Mean (SD)	Duration of diabetes Mean (SD)	Male/Female	Type of study	Duration of study
DAFNE	141	40 (9)	16.6 (9.6)	69/72	Delayed randomised controlled trial	6 months
REACCT	80		Data not reported		Intervention	6 weeks
NEP	137	47 (15)	15 (26.2)	61/76	Intervention	4 and 12 months
BITES	114	41 (11)	195 (11.8)	51/63	Block randomised controlled trial	12 months
RECLAIM	298	44(14)	Data not reported		Retrospective audit	12 months
FIT	45	41 (Range 18-79)	10 (Range 1-49)	21/24	Intervention	6 and 18 months

DAFNE: Dose Adjustment for Normal Eating (DAFNE study group 2002)
REACCT: Re-education and Carbohydrate Counting Training (Ulahannan, Ross et al. 2007)
NEP: Newcastle Empowerment Programme (Lowe, Linjawi et al. 2008)
BITES: Brief Intervention in Type 1 diabetes, Education for Self-efficacy (George, Valdovinos et al. 2008)
RECLAIM: Royal Infirmary of Edinburgh Carbohydrate Learning and Insulin Management (Teoh, Anderson et al. 2010)
FIT: Flexible Insulin Therapy (Falconnier Bendik, Keller et al. 2009)

6.4.6. Results

Structured education programmes for people with Type 1 diabetes incorporating carbohydrate counting and insulin adjustment are postulated to have positive effects on glycaemic control, quality of life and rates of hypoglycaemia without increasing body weight. These factors are discussed more fully below.

6.4.6.1. Glycaemic control and body weight

Table 6.2 shows changes in A1c and body weight from baseline for the six studies investigating the effects of structured education in people with Type 1 diabetes. The results are somewhat contradictory, with three studies showing a significant improvement in glycaemic control and the remaining three studies indicating a small but non-significant improvement. The DAFNE study showed the greatest reduction in A1c (1%), with other studies reporting reductions of 0.1-0.9%. There appears to be a trend towards greater improvement in those subjects with a higher A1c at baseline, and this is supported by data from one of the DAFNE centres showing that there was a greater mean fall in A1c (-0.7 v -0.3%, p=<0.001) for 102 individuals with a baseline A1c >8.5% (Lawrence, Hopkins et al. 2008).

None of these studies indicate that structured education is associated with deterioration in glycaemic control in people with Type 1 diabetes, and this is particularly important within the context of relaxation of dietary prescription. All these studies promote a more flexible approach to food and eating than was previously the case, and this is particularly true of the DAFNE study where the study is promoted under the by-line 'Eat what you like, like what you eat.' Although none of the studies attempted to evaluate dietary intake in people with Type 1 diabetes, subjective feedback from the studies suggest that most subjects imposed no limits on

their carbohydrate intake and many began to include larger quantities of sugar-containing foods into their diet.

It has long been known that improvements in glycaemic control in people with Type 1 diabetes are associated with significant weight gain (Zinman 1998). The DCCT trial, for example, reported significant weight gain in the intensively treated group and reported that during the first year of the study, mean weight gain in the intensively treated group was 5.1kg, compared with 2.4kg in the conventionally treated group ($p<0.0001$) (DCCT study group 1988). The studies reported here that investigated the effects of structured education have reported no significant change in weight, even when there is a reported significant improvement in glycaemic control. This suggests that structured education programmes enable people with Type 1 diabetes to improve glycaemic control and enjoy greater dietary freedom without a significant increase in weight. This may be important for future health, as follow-up data from the DCCT showed that those with the greatest weight gain exhibited significantly increased cardiovascular risk, namely raised blood pressure and changes in lipid levels (Purnell, Hokanson et al. 1998).

Table 6.2. Changes from baseline in body weight, BMI, and A1c in studies of structured education programmes for people with Type 1 diabetes

Programme name	A1c (%)			Weight (kg)			BMI (kg/m^2)		
	Baseline	Change	P value	Baseline	Change	P value	Baseline	Change	P value
DAFNE	9.4	-1.0	<0.001	80.5	1.0	0.11		Data not reported	
REACCT	8.37	-0.09	NS		Data not reported			Data not reported	
NEP					Data not reported			Data not reported	
4 months	8.27	-0.17	0.04						
12 months	8.27	-0.19	0.04						
BITES	8.7	-0.3	0.94		Data not reported		26.3	0.07	0.77
RECLAIM	8.91	-0.6	<0.001	79.3	0.45	NS		Data not reported	
FIT									
6 months	7.2	-0.1	NS	68.1	1.3	NS	23.0	23.4	NS
18 months	7.2	-0.1	NS	68.1	0.1	NS	23.0	23.0	NS

NS, no significant difference

DAFNE: Dose Adjustment for Normal Eating (DAFNE study group 2002)
REACCT: Re-education and Carbohydrate Counting Training (Ulahannan, Ross et al. 2007)
NEP: Newcastle Empowerment Programme (Lowe, Linjawi et al. 2008)
BITES: Brief Intervention in Type 1 diabetes, Education for Self-efficacy (George, Valdovinos et al. 2008)
RECLAIM: Royal Infirmary of Edinburgh Carbohydrate Learning and Insulin Management (Teoh, Anderson et al. 2010)
FIT: Flexible Insulin Therapy {Falconnier Bendik, 2009)

6.4.6.2. Hypoglycaemia rates

Improvements in glycaemic control in people with Type 1 diabetes are associated with an increase in the rate of hypoglycaemia. People with Type 1 diabetes report that hypoglycaemia is one of their greatest fears (Davies 2004), and avoidance of hypoglycaemia plays a large part in decision-making about treatment and targets for blood glucose levels (Davis and Alonso 2004). The DCCT reported that levels of severe hypoglycaemia in the intensively treated group were three times higher that those of the conventionally treated group, and that hypoglycaemia was more common in the intensively treated group at all comparable levels of A1c (DCCT study group 1997). Structured education programmes aim to improve glycaemic control without increasing the risk and frequency of hypoglycaemia.

Three of the published studies describing the effects of structured education in people with Type 1 diabetes reported effects on rates of hypoglycaemia. REACCT, NEP and RECLAIM did not assess or report hypoglycaemia rates and the three remaining studies used different measures to assess frequency and fear of hypoglycaemia. DAFNE assessed frequency of hypoglycaemia by means of the diabetes satisfaction questionnaire (DTSQ) (Bradley 2003) and showed that there was no increase in the frequency of hypoglycaemia in the intervention group despite a significant improvement in glycaemic control (DAFNE study group 2002). BITES measured both frequency and fear of hypoglycaemia using records of pre-defined hypoglycaemia and the hypoglycaemia fear scale (HFS) (Cox, Irvine et al. 1987). Hypoglycaemia was pre-defined as a recorded episode of blood glucose values <2.7 mmol/l or the subject required assistance from a third party to treat clinical symptoms. There were no significant differences in rates of severe hypoglycaemia between the

intervention and the control groups. In addition, this study reported no change in fear of hypoglycaemia between the two groups (George, Valdovinos et al. 2008). The FIT study measured frequency of hypoglycaemia defined as any episode requiring assistance by a third party, and this was the only study to report a decrease in hypoglycaemia as a result of the education intervention. Severe hypoglycaemia decreased by ten-fold as a result of the intervention, decreasing from a mean of 0.66 episodes per patient per year to <0.05 episodes per patient per year. The explanation offered for this was that patients were able to adjust their insulin doses more accurately by accounting for self-monitored blood glucose levels, amount of carbohydrate eaten and effects of physical activity (Falconnier Bendik, Keller et al. 2009).

These studies suggest that structured education programmes can improve glycaemic control in people with Type 1 diabetes without increasing the risk of hypoglycaemia, and may help prevent severe hypoglycaemia.

6.4.6.3. Quality of life

A large, global study of over 5000 people with diabetes has shown that psychological distress is common in people with diabetes. The Diabetes Attitudes Wishes and Needs (DAWN) study has shown that 41% of people with diabetes report poor psychological well-being and experience emotional distress related to their diabetes (Peyrot, Rubin et al. 2005). Diabetes related distress is especially common at diagnosis, with 82.5% of this sample reporting high levels of distress (Funnell 2006). In addition, depression is more prevalent amongst those with diabetes, with twice as many people with diabetes reporting they are depressed and this has effects on self-management and diabetes care (Diabetes UK 2006). Structured education programmes aim to improve

quality of life and self-management skills in people with diabetes. Measurement of quality of life in people with diabetes presents methodological problems, typically different studies use different techniques to measure quality of life and this makes comparisons difficult (Speight, Reaney et al. 2009). Table 6.3 shows the effects of the different assessments of quality of life and diabetes distress in the studies under review. Five of the six studies assessed quality of life and although many different tools are available to measure quality of life and diabetes distress, all these reported studies show that structured education programmes significantly improved quality of life, independent of any change in either glycaemic control or hypoglycaemia.

The DAFNE study used the ADDQoL questionnaire (Bradley and Speight 2002) to measure quality of life and the W-BQ12 (Bradley and Speight 2002) to measure psychological well-being. The ADDQoL produces a diabetes impact rating that includes 18 domains of life. Scores range from +9, representing the maximum positive effect of diabetes, to -9, representing the maximum negative effect. The W-BQ12 is a series of 12 questions, each with a maximum score of 3, and higher scores indicate better psychological welfare. DAFNE reported significant improvement in overall quality of life measured by ADDQoL, especially in the areas exploring the negative impact of diabetes and dietary freedom (DAFNE study group 2002). The REACCT study did not use a validated quality of life questionnaire, although subjective evaluation showed that 56% of subjects reported improved quality of life (Ulahannan, Ross et al. 2007).

The NEP measured quality of life using the ADDQoL questionnaire and assessed self-efficacy by means of a diabetes empowerment scale (DES) and which includes three sub-scales; managing psychological aspects, goal-setting and readiness to change

(Anderson, Funnell et al. 2000). DES is designed to assess self-efficacy and is rated by a score of 1-5 for each question. A higher score indicates greater self-efficacy. NEP showed that quality of life measured by ADDQoL improved significantly at 4 months and this improvement was maintained at 12 month's follow-up. Self-efficacy measured by DES improved significantly at 4 months, but the effects were lost at 12 months' follow-up (Lowe, Linjawi et al. 2008). BITES assessed self-efficacy using the DES and a diabetes health profile (DHP) which includes three sub-scales measuring psychological distress, barriers to activity and disinhibited eating (Meadows, Steen et al. 1996). The DES showed significant improvement in two areas, but not in readiness to change and the DHP showed no differences between the groups except for reduced barriers to activity in the intervention group (George, Valdovinos et al. 2008). The FIT study used the same questionnaire as that used for DCCT, the diabetes quality of life questionnaire (DQOL) (DCCT study group 1988) and which includes 46 questions. Total scores range from 46-230, with higher scores indicating poorer quality of life. There was a significant reduction in total scores at the end of the study, indicating improved quality of life (Falconnier Bendik, Keller et al. 2009).

In summary, these studies under review show that structured education for people with Type 1 diabetes improves quality of life and diabetes related distress, although different studies have used different methods of assessment.

Table 6.3. Mean changes in quality of life in studies of structured education in people with Type 1 diabetes

Programme name	Assessment tool	Baseline value	Change	P value
DAFNE	ADDQoL	Not reported	0.4	<0.01
	W-BQ12	20.94	3.4	<0.01
REACCT	Subjective questionnaire	Not reported	Not reported	Not reported
NEP	DES:			
	4 months		0.19	0.001
	12 months		0.03	0.19
	ADDQoL:	Not reported		
	4 months		1.9	0.05
	12 months		3.9	0.005
BITES	DES:			
	Managing psychological aspects:		4.6	0.005
	Setting and achieving goals:		3.7	0.02
	Readiness to change:	Not reported	2.87	0.12
	DHP:			
	Psychological distress		-2.3	0.93
	Barriers to activity		-3.5	0.02
	Disinhibited eating		-3.6	0.12
FIT	DQOL:			
	6 months	91.8	-6.1	<0.001
	18 months	91.8	-6.2	<0.001

ADDQoL: diabetes dependent quality of life (Bradley and Speight 2002)
W-BQ 12: 12-item well-being questionnaire (Bradley 2000)
DES: diabetes empowerment scale (Anderson, Funnell et al. 2000)
DHP: diabetes health profile (Meadows, Steen et al. 1996)
DQOL: diabetes quality of life (DCCT study group 1988)

6.4.7. Conclusions

In summary, the available evidence shows that in four intervention studies and two randomised controlled trials of structured education programmes in people with Type 1 diabetes, the majority showed significant improvement in glycaemic control and quality of life. No study published to date has shown a deleterious effect on glycaemic control, body weight or risk of severe hypoglcaemia, but these findings should be interpreted with caution as there were no control groups in the majority of studies. In addition, the majority of studies report that their education programmes are designed to promote dietary freedom and flexibility, but this is not measured or reported in any of the studies to date. In an attempt to identify the role of structured education in people with Type 1 diabetes, we designed and evaluated an education programme for people with Type 1 diabetes and assessed its effects on glycaemic control, body weight, cardiovascular risk and quality of life.

6.5. Structured education for people with Type 1 diabetes – the InSight programme

6.5.1. Introduction

The day to day responsibility for diabetes control lies with the individual with Type 1 diabetes, but without appropriate education and support from the healthcare team, self management can be challenging. Education is fundamental to enable the person with diabetes to acquire the relevant knowledge and skills to be successful in managing independently.

The Diabetes Control and Complications Trial demonstrated the benefits of intensive glucose control in reducing long term complications (DCCT study group 1993), but was associated with significantly increased rates of hypoglycaemia and higher body weight, which in turn increased cardiovascular risk (DCCT study group 1988). To

facilitate improved glycaemic control in routine clinical practice without these undesirable side-effects, there is a need for self-management educational interventions. The aim is to improve self-management skills by providing knowledge and improving skills relating to carbohydrate assessment, insulin adjustment, and management of physical activity, illness and hypoglycaemia.

Structured education has been recognised as a fundamental aspect of supporting people with diabetes. National policy in the UK now reflects this importance and education requirements are documented within the NSF for diabetes (Department of Health 2003) and specific NICE guidance (NICE 2003). There is an increasing core of evidence that structured education can improve both psychosocial and clinical outcomes, through developing self management skills.

The development of structured education in the UK was initiated with the DAFNE trial (DAFNE study group 2002) based on the Dusseldorf model (Muhlhauser, Bruckner et al. 1987). Although DAFNE provided the template for skills training for people with Type 1 diabetes in the UK, initially there were significant resource issues and many routine diabetes clinics had financial restrictions preventing its application. The challenge was to develop a programme within existing resources that could be integrated into routine clinical service. Other clinical diabetes teams were also developing programmes and in order to share good practice, evaluate outcomes and ensure quality processes, the Type 1 education network, now known as the Diabetes Education Network (www.diabetes-education.net), was founded.

The InSight programme was developed in Oxford and is a skills-based programme addressing carbohydrate assessment, insulin adjustment and self-care management of

hypoglycaemia, exercise and illness. Evaluation of the Insight programme is presented as an observational study assessing education in routine clinical care.

6.5.2. Aims and objectives

The aims of the InSight programme are for participants to develop skills and techniques to enable them to understand the effects of lifestyle on their diabetes and vice versa, and how they can manipulate their treatment to enable them to lead the lifestyle of their choice and improve glycaemic control.

6.5.3. Methods

A full description of the methods is provided in Chapter 2. Briefly, this study received Chairman's approval from the local ethics committee and was designed to facilitate skills for matching insulin to carbohydrate intake based upon reflection from self-monitoring diaries and to support self-management of hypoglycaemia, hyperglycaemia, illness and exercise.

6.5.4. Results

Baseline characteristics of the 51 InSight subjects who completed the education programme are shown in Table 6.4. One year data are presented for 48 (94%) of the subjects. Changes in A1c, weight and lipid levels at six and twelve months follow-up are shown in Table 6.5. PAID and hypoglycaemia questionnaires were fully completed by all 51 (100%) of the subjects at baseline, 46 (90.1%) at six months and 45 (88.2%) at one year. Changes in diabetes distress measured by PAID and changes in rates and severity of hypoglycaemia are shown in Table 6.6.

Glycaemic control, assessed by A1c showed a significant improvement at six months (-0.4%, p=<0.006) and this was maintained at twelve months follow-up (-0.3%,

p=0.03). There were no changes in other biometric measurements including body weight, BMI and lipid levels. There was a highly significant reduction in diabetes related distress by six months measured by PAID (mean score 33% v 25%) and this positive effect was maintained at one year. This reduction in scores was significant at six months and one year compared to baseline (p=<0.01 and p=0.019 respectively). In addition, there was a significant reduction in the number of subjects reporting both moderate and severe hypoglycaemic events and an improvement in awareness of hypoglycaemia.

Table 6.4 Baseline characteristics of 51 InSight subjects

Variable	Subjects Mean (SD)
Number	51
% Male	34
Age (years)	42.5 (11.3)
Diabetes duration (years)	21.0 (13.5)
A1c (%)	8.5 (1.4)
Body weight (kg)	76.4 (12.9)
BMI (kg/m^2)	26.2 (3.8)
Total cholesterol (mmol/l)	4.6 (0.8)
HDL cholesterol (mmol/l)	1.7 (0.5)
Triglycerides (mmol/l)	0.9 (0.8)
LDL cholesterol (mmol/l)	2.5 (0.6)
PAID score (%)	33.7 (16.9)
Hypoglycaemia	
% reporting 1 or more episodes of severe hypoglycaemia in the past year	21.5
% reporting 1 or more episodes of moderate hypoglycaemia in the six months	41.2
% reporting hypoglycaemic unawareness	37.2

Table 6.5. Changes in biomedical variables in InSight subjects at six and twelve months follow-up.

Variable	Change at 6m	*p*-value	Change at 12m	*p*-value
A1c (%)	-0.4	0.006	-0.3	0.028
Body weight (kg)	-0.3	0.292	0.2	0.708
BMI (kg/m^2)	-0.1	0.501	0.1	0.638
Total cholesterol (mmol/l)	-0.03	0.679	0.04	0.757
HDL cholesterol (mmol/l)	-0.04	0.126	-0.05	0.094
Triglycerides (mmol/l)	0.01	0.786	-0.12	0.136
LDL cholesterol (mmol/l)	0.0	0.926	0.11	0.196

Table 6.6. Changes in PAID scores and rates of hypoglycaemia in InSight subjects at six and twelve months follow-up.

Variable	Change at 6m	*p*-value	Change at 12m	*p*-value
PAID score (%)	-8.2	<0.01	-7.3	0.019
% reporting 1 or more episodes of severe hypoglycaemia in the past year	-13.0	<0.01	-13.3	<0.01
% reporting 1 or more episodes of moderate hypoglycaemia in the six months	-15.3	<0.01	-15.5	<0.01
% reporting hypoglycaemic unawareness	-21.7	<0.001	-24.5	<0.001

6.5.5. Discussion

6.5.5.1. Glycaemic control

This study showed a significant improvement in glycaemic control in subjects who attended a structured education course incorporating carbohydrate counting and insulin adjustment. At twelve month's follow up A1c levels had decreased by 0.3% to 8.2%, and although this may be statistically significant, there remain some questions about the clinical significance of this improvement in glycaemic control. Diabetes UK and the American Diabetes Association have both recommended target A1c levels of <7.0% for people with diabetes, with some authorities recommending lower levels of 6.5% (Rodbard, Blonde et al. 2007). In this context, A1c levels of 8.2% may be considered unsatisfactory. In comparison DAFNE, the best-known randomised, controlled trial of this approach to structured education, showed a significant reduction in A1c of 1% over six months (DAFNE study group 2002). However, only subjects with an A1c >7.5% were included in the study and the mean A1c at entry was 9.4%. This is higher than that of the InSight participants whose mean A1c was 8.5% at baseline. The A1c end-point of DAFNE was equivalent to the starting point of InSight. This may suggest that there is greater glycaemic benefit for those with a higher A1c at entry to the programme. A sub-analysis of the InSight data, using an arbitary cut-off of 7.5% showed that 12 individuals (24%) with A1c levels below 7.5% at baseline showed an increase in A1c at one year (6.8 v 7.0%, p=0.2). Conversely, 36 subjects with A1c levels above 7.5% showed a larger reduction in A1c at one year compared to the full cohort (9.0 v 8.5%, p=0.003). In addition, DAFNE have reported one year follow-up data from use in routine clinical practice and these data show a reduction in A1c from 8.5% to 8.2% (Lawrence, Hopkins et al. 2008). Although this reduction was statistically significant, it does not match the magnitude

of reduction reported in the trial data and is comparable to the results from the InSight trial.

6.5.5.2. Medication

All InSight subjects had optimised prandial basal insulin regimens with analog insulins before entry to the study and continued with this during the study period. This is in contrast to both DAFNE and published data from Germany (Muhlhauser, Bruckner et al. 1987) where changes in insulin regimen, especially changes from twice daily biphasic insulin regimens to basal prandial regimens were common place. Any recorded effect of InSight was due solely to the education intervention.

6.5.5.3. Diabetes related distress

In common with other clinical programmes, the InSight programme showed significantly positive effects on well-being. PAID was used as a marker of diabetes distress and there were significant improvements at six months which were maintained at one year follow-up.

6.5.5.4. Hypoglycaemia

Improvements in glycaemic control in people with Type 1 diabetes and are associated with increased hypoglycaemia. The DCCT reported that hypoglycaemia was more common in the intensively treated group (DCCT study group 1997). People with Type 1 diabetes report that hypoglycaemia is one of their greatest fears (Davies 2004), and avoidance of hypoglycaemia plays a large part in decision-making about treatment and targets for blood glucose levels (Davis and Alonso 2004). The InSight programme successfully reduced both A1c and hypoglycaemia. There were significant reductions in rates of both moderate and severe hypoglycaemia and an increase in awareness of the symptoms of hypoglycaemia.

6.5.6. Summary

The introduction of the InSight programme has shown that it is feasible to integrate a local structured education programme, which meets the current Department of Health guidelines, into routine clinical practice. This programme has shown a significant improvement in glycaemic control, diabetes related distress and hypoglycaemia without compromising general health or increasing body weight, supporting the theory that more flexibility in both educational approaches and carbohydrate manipulation may improve outcomes in people with Type 1 diabetes.

Chapter 7

Overall conclusions and recommendations for further work

7.0 Conclusions

Lifestyle education, especially dietary advice, has been shown to have a positive impact on the treatment of diabetes. There are two components to dietary education, the content of any education programme and the method of delivery of that programme. Both these aspects have been addressed in this thesis, with the aim of improving the provision and quality of education available for people with diabetes.

The content of education programmes, and especially dietary education, has been traditionally dictated by a consensus of expert opinion, rather than relying upon evidence from randomised controlled trials. It is now generally agreed that evidence-based recommendations for treatment of all diseases, including diabetes, should be adopted as the gold standard. However, the assessment of dietary interventions for people with diabetes has proved challenging as randomised trials that take place in free-living populations cannot be blinded, and may lead to issues with contamination between intervention and control groups and with concordance and compliance. In addition, many studies have involved small numbers of subjects, lacked a control group, have high attrition rates, short follow-up periods and shown relatively small changes in end-points. The impact this has for people with diabetes is that there is little hard evidence for most dietary components of education programmes, and in addition, adopting the recommendations to eat a low fat, high carbohydrate diet may not necessarily lead to the desired improvements in blood glucose levels or weight loss.

The emphasis of dietary advice for people with diabetes over the past twenty years has been aiming to reduce the risk of chronic disease, especially cardiovascular disease, by reducing total and saturated fat intake and obtaining the majority of energy from carbohydrate. This strategy may have an adverse affect upon glycaemic control as carbohydrate foods have been shown to increase blood glucose levels after eating, and large amounts of carbohydrate are associated with postprandial hyperglycaemia. There may be more effective dietary approaches which reduce the glycaemic load and this may be achieved by adopting a diet of low glycaemic index, reducing total carbohydrate intake or both. This thesis aimed to show that reducing the glycaemic load by adopting a low carbohydrate diet would be effective for the treatment of Type 2 diabetes. A review of the available evidence showed that there was limited evidence for low carbohydrate diets in the treatment of Type 2 diabetes and Study 1 was designed as a randomised controlled trial to evaluate the effect of a low carbohydrate diet on glycaemic control and body weight. This study showed that weight loss was significantly greater in the low carbohydrate group and although there were no significant differences between the control group and the intervention group, glycaemic control improved with weight loss. The assumption that low carbohydrate diets are less healthy than conventional diets as they are higher in fat and protein was also disproved, as was the commonly held belief that low carbohydrate diets increase cardiovascular risk. This study showed for the first time that low carbohydrate diets are as effective in people with Type 2 diabetes as in people without diabetes, they induce significantly greater weight loss than conventional low fat diets and do not increase cardiovascular risk. These results suggest that low carbohydrate diets may be of benefit to people with Type 2 diabetes who are overweight or obese. The limitations of this study were that it included small numbers and was a short-term

study with three months, follow-up. There is no evidence that low carbohydrate diets are safe and effective over the longer term, and weight loss, glycaemic control, cardiovascular risk and adherence may be of issue in the longer term. Further studies in people with diabetes using low carbohydrate interventions over the longer term may show benefits in both glycaemic control and body weight, and elucidate the safety and efficacy of these diets.

The process of delivery of education can also affect outcomes in people with diabetes. The traditional medical model is widely used to deliver lifestyle education to people with diabetes, with few studies investigating the efficacy and safety of education. Education is seen as a cornerstone to self-management for people with diabetes, yet there are few studies investigating and evaluating the effect of education for people with diabetes. Individual consultations, with the health professional seen as the expert, is the most widely adopted model of education delivery, especially in primary care, despite the fact that there is emerging evidence that structured education programmes delivered in groups can improve outcomes in people with diabetes.

A review of education programmes for people with diabetes was performed and showed that, although education has been shown to be effective for the treatment of diabetes, there was little evidence to support alternative methods of education, largely because of lack of evaluation of different strategies. In practical terms, application of the structured education programmes delivered in research studies in routine clinical care requires resources well beyond that of the majority of health care systems. This suggests that there is a role for other methods of education delivery within clinical practice and this should be subject to evaluation. Innovative education programmes using alternative approaches, for example video education, have not been evaluated

in clinical practice and Study 2 was designed as a randomised controlled trial to assess a novel education programme delivered by video for people newly diagnosed with Type 2 diabetes in primary care. At six months follow-up, there was a significant increase in knowledge in the intervention group receiving the video and although there were significant improvements in glycaemic control, lipid levels and physical activity in the intervention group and no significant change in the control group, these failed to reach significance between groups. This study showed for the first time that education delivered by video improved knowledge in people with diabetes and had a positive impact upon health. The major limitations of this study are the small numbers recruited, the lack of effect compared to the control group and that follow-up lasted only six months. In terms of chronic conditions such as diabetes, it may be that the benefits of education are apparent over the longer term and that this type of education may be useful in primary care. However, it is worth noting that videos are fast becoming out-dated, and it may be of more benefit to explore the use of interactive, web-based education programmes and social networking sites to deliver diabetes education.

The theme of investigating alternative methods of education and examining the role of carbohydrate in the diet of people with diabetes was continued in the third study, which examined group education with an emphasis on peer support and including carbohydrate counting and insulin adjustment in people with Type 1 diabetes. Traditionally, people with Type 1 diabetes have been advised to monitor and regulate their carbohydrate intake to a prescribed insulin dose, resulting in lack of flexibility with associated effects upon quality of life and issues with adherence. Previous studies applying this traditional but inflexible approach have shown significant improvements in glycaemic control, but accompanied by increased hypoglycaemia,

weight gain and increased cardiovascular risk. Study 3 applied carbohydrate counting and insulin adjustment in routine clinical practice and showed significant improvements in glycaemic control, diabetes related distress and hypoglycaemia rates in people with Type 1 diabetes without associated weight gain or any other adverse effects including any dietary restriction. These findings suggest that more flexibility in educational programmes, carbohydrate intake and insulin doses can improve outcomes for people with Type 1 diabetes. The limitations of this study were that follow-up was relatively short-term over one year, and that the lack of a control group prevented any firm conclusions being drawn from this intervention.

Although there remains little firm evidence about the most effective lifestyle interventions to improve management of diabetes and its associated risk factors, this thesis has shown that flexibility in carbohydrate intake may improve glycaemic control and some risk factors in people with both Type 1 and Type 2 diabetes. In addition, more innovative methods of delivering education including video education and group-based education sessions can improve outcomes when compared to the traditional medical model. The results from the three studies making up this thesis suggest that there should be more flexibility in the dietary management of diabetes, both in terms of carbohydrate intake and educational strategies. Diabetes UK, the charity for people with diabetes, has published nutritional guidelines for diabetes and these recommend that people with diabetes should adopt a relatively high carbohydrate diet. This thesis challenges that view and would propose that overweight and obese people with Type 2 diabetes who are aiming to lose weight may benefit from reducing carbohydrate intake and that people with Type 1 diabetes can be more flexible in their carbohydrate intake if adjusting insulin to intake. In addition, the

traditional medical model of diabetes education can be improved by adopting more innovative approaches including delivering education by video and utilising peer support in group education.

7.1. Further studies

- Investigate the potential effects of reduced carbohydrate diets in people with Type 1 diabetes with special reference to glycaemic control, body weight, risk of complications, insulin dose and quality of life
- Determine the effects of diets of low glycaemic load, whether low carbohydrate or of low glycaemic index, on glycaemic control and cardiovascular risk in people with both Type 1 and Type 2 diabetes
- Assess the optimum amount of dietary carbohydrate for health in people with diabetes
- Explore the application of web-based interactive education for people with Type 1 and Type 2 diabetes
- Examine the application of video-based diabetes education for people of different ethnic origins

Anderson RM, Funnell MM, Fitzgerald JT, Marrero DG (2000). The Diabetes Empowerment Scale: a measure of psychosocial self-efficacy. *Diabetes Care* **23** (6): 739-43.

Anderson RM and Funnell MM (2000). Compliance and adherence are dysfunctional concepts in diabetes care. *Diabetes Educ* **26** (4): 597-604.

Anon (2009). Diabetes--a global threat. *Lancet* **373** (9677): 1735.

Arora SK and McFarlane SI (2005). The case for low carbohydrate diets in diabetes management. *Nutr Metab (Lond)* **2**: 16.

Astrup A, Meinert Larsen T, Harper A (2004). Atkins and other low-carbohydrate diets: hoax or an effective tool for weight loss? *Lancet* **364** (9437): 897-9.

Aucott L, Poobalan A, Smith WC, Avenell A, Jung R, Broom J, Grant AM (2004). Weight loss in obese diabetic and non-diabetic individuals and long-term diabetes outcomes--a systematic review. *Diabetes Obes Metab* **6** (2): 85-94.

Bandura A. (1977). Social learning theory. Eaglewood Cliffs, NJ, Prentice Hall.

Bantle JP, Wylie-Rosett J, Albright AL, Apovian CM, Clark NG, Franz MJ, Hoogwerf BJ, Lichtenstein AH, Mayer-Davis E, Mooradian AD, Wheeler MG (2008). Nutrition recommendations and interventions for diabetes: a position statement of the American Diabetes Association. *Diabetes Care* **31** Suppl 1: S61-78.

Barclay AW, Petocz P, McMillan-Price J, Flood VM, Prvan T, Mitchell P, Brand-Miller JC (2008). Glycemic index, glycemic load, and chronic disease risk--a meta-analysis of observational studies. *Am J Clin Nutr* **87** (3): 627-37.

Barth JH, Marshall SM, Watson ID (2008). Consensus meeting on reporting glycated haemoglobin (HbA1c) and estimated average glucose (eAG) in the UK: report to the National Director for Diabetes, Department of Health. *Diabet Med* **25** (4): 381-2.

Bassuk SS and Manson JE (2005). Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease. *J Appl Physiol* **99** (3): 1193-204.

Bazzano LA, Li TY, Joshipura KJ, Hu FB (2008). Intake of fruit, vegetables, and fruit juices and risk of diabetes in women. *Diabetes Care* **31** (7): 1311-7.

Bingham SA (1997). Dietary assessments in the European prospective study of diet and cancer (EPIC).*Eur J Cancer Prev* **6** (2): 118-24.

Boden G, Sargrad K, Homko C, Mozzoli M, Stein TP (2005). Effect of a low-carbohydrate diet on appetite, blood glucose levels, and insulin resistance in obese patients with type 2 diabetes. *Ann Intern Med* **142** (6): 403-11.

Bolen S, Feldman L, Vassy J, Wilson L, Yeh HC, Marinopoulos S, Wiley C, Selvin E, Wilson R, Bass EB, Brancati FL (2007). Systematic review: comparative effectiveness and safety of oral medications for type 2 diabetes mellitus. *Ann Intern Med* **147** (6): 386-99.

Boren SA, Fitzner KA, Panhalkar PS, Specker J (2008). Costs and benefits associated with diabetes education: A review of the literature. *Diabetes Educ* **35**: 72-96.

Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ (2001). Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. *JAMA* **286** (10): 1218-27.

Boule NG, Kenny GP, Haddad E, Wells GA, Sigal RJ (2003). Meta-analysis of the effect of structured exercise training on cardiorespiratory fitness in Type 2 diabetes mellitus. *Diabetologia* **46** (8): 1071-81.

Bradley C. (2000). The 12-Item Well-Being Questionnaire: origins, current stage of development, and availability. *Diabetes Care* **23** (6): 875.

Bradley C and Speight J (2002). Patient perceptions of diabetes and diabetes therapy: assessing quality of life. *Diabetes Metab Res Rev* **18** Suppl 3: S64-9.

Bradley C (2003). The diabetes treatment satisfaction questionnaire: DTSQ. Handbook of psychology and diabetes: a guide to psychological measurement in diabetes research and practice. Hove, Psychology Press.

Brand-Miller J, Hayne S, Petocz P, Colagiuri S (2003). Low-glycemic index diets in the management of diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care* **26** (8): 2261-7.

Bravata DM, Sanders L, Huang J, Krumholz HM, Olkin I, Gardner CD (2003). Efficacy and safety of low-carbohydrate diets: a systematic review. *JAMA* **289** (14): 1837-50.

Bravata DM, Smith-Spangler C, Sundaram V, Gienger AL, Lin N, Lewis R, Stave C, Olkin I, Sirard JR (2007). Using pedometers to increase physical activity and improve health: a systematic review. *JAMA* **298** (19): 2296-304.

Brazier J (1993). The SF-36 health survey questionnaire--a tool for economists. *Health Econ* **2** (3): 213-5.

Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA (2003). A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. *J Clin Endocrinol Metab* **88** (4): 1617-23.

Brock G, Carrier S, Casey R, Tarride JE, Elliott S, Dugre H, Rousseau C, D'Angelo P, Defoy I (2007). Can an educational program optimize PDE5i therapy? A study of Canadian primary care practices. *J Sex Med* **4** (5): 1404-13.

Brookes R, Rabin R, De Charro F (2003). The measurement and validation of health status using EQ-5D: A European perspective. Kluwer Academic Press.

Brown SJ, Lieberman DA, Germeny BA, Fan YC, Wilson DM, Pasta DJ (1997). Educational video game for juvenile diabetes: results of a controlled trial. *Med Inform (Lond)* **22** (1): 77-89.

Buyken AE, Toeller M, Heitkamp G, Karamanos B, Rottiers R, Muggeo M, Fuller J H (2001). Glycemic index in the diet of European outpatients with type 1 diabetes: relations to glycated hemoglobin and serum lipids. *Am J Clin Nutr* **73** (3): 574-81.

California Pan-Ethnic Health Network (2005). Out of balance. San Francisco, CPENH, Consumer Union of the United States.

Campbell LV and Alford J (2006). Sick day management for patients with diabetes. *Aust Fam Physician* **35** (6): 405-6.

Clarke WL, Cox DJ, Gonder-Frederick LA, Julian D, Schlundt D, Polonsky W (1995). Reduced awareness of hypoglycemia in adults with IDDM. A prospective study of hypoglycemic frequency and associated symptoms. *Diabetes Care* **18** (4): 517-22.

Consumers International (2008). International code on marketing food to children: Marketing spend figures. London, Consumers International.

Corben S and Rosen R (2005). Self-management for long-term conditions. London, King's Fund

Cox DJ, Irvine A, Gonder-Frederick L, Nowacek G, Butterfield J (1987). Fear of hypoglycemia: quantification, validation, and utilization. *Diabetes Care* **10** (5): 617-21.

DAFNE study group (2002). Training in flexible, intensive insulin management to enable dietary freedom in people with type 1 diabetes: dose adjustment for normal eating (DAFNE) randomised controlled trial. *BMJ* **325** (7367): 746.

Daly ME, Paisey R, Millward BA, Eccles C, Williams K, Hammersley S, Macleod KM, Gale TJ (2006). Short-term effects of severe dietary carbohydrate-restriction advice in Type 2 diabetes-a randomized controlled trial. *Diabet Med* **23** (1): 15-20.

Daly ME, Piper J, Paisey R, Darby T,Paisey R, George L, Ball C, Vaezi A, Williams K, Gale TJ (2006). Efficacy of carbohydrate restriction in obese type 2 diabetes patients. *Diabet Med* **23** (Suppl 2): 26-7.

Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ (2005). Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA* **293** (1): 43-53.

Dansinger ML, Tatsioni A, Wong JB, Chung M, Balk EM (2007). Meta-analysis: the effect of dietary counseling for weight loss. *Ann Intern Med* **147** (1): 41-50.

Davies M (2004). The reality of glycaemic control in insulin treated diabetes: defining the clinical challenges. *Int J Obes Relat Metab Disord* **28** Suppl 2: S14-22.

Davies MJ, Heller S, Skinner TC, Campbell MJ, Carey ME, Cradock S *et al* (2008). Effectiveness of the diabetes education and self management for ongoing and newly diagnosed (DESMOND) programme for people with newly diagnosed type 2 diabetes: cluster randomised controlled trial. *BMJ* **336**(7642): 491-5.
Davis S and Alonso MD (2004). Hypoglycemia as a barrier to glycemic control. *J Diabetes Complications* **18** (1): 60-8.

Day JL, Rayman G, Hall L, Davies P (1997). "Learning Diabetes'--a multi-media learning package for patients, carers and professionals to improve chronic disease management. *Med Inform (Lond)* **22** (1): 91-104.

Day NE, Wong MY, Bingham S, Khaw KT, Luben R, Michels KB, Welch A, Wareham NJ (2004). Correlated measurement error--implications for nutritional epidemiology. *Int J Epidemiol* **33** (6): 1373-81.

DCCT study group (1988). Reliability and validity of a diabetes quality-of-life measure for the diabetes complications and control trial (DCCT). *Diabetes Care* **11**: 725-32.

DCCT study group (1988). Weight gain associated with intensive therapy in the diabetes control and complications trial. The DCCT Research Group. *Diabetes Care* **11** (7): 567-73.

DCCT study group (1993). The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. The Diabetes Control and Complications Trial Research Group. *N Engl J Med* **329** (14): 977-86.

DCCT study group (1997). Hypoglycaemia in the diabetes control and complications trial. *Diabetes* **46**: 271-86.

Deakin TA, Cade JE, Williams R, Greenwood DC (2006). Structured patient education: the diabetes X-PERT Programme makes a difference. *Diabet Med* **23** (9): 944-54.

Delahanty LM and Halford BN (1993). The role of diet behaviors in achieving improved glycemic control in intensively treated patients in the Diabetes Control and Complications Trial. *Diabetes Care* **16** (11): 1453-8.

de Munter JS, Hu FB, Spiegelman D, Franz M, van Dam RM (2007). Whole grain, bran, and germ intake and risk of type 2 diabetes: a prospective cohort study and systematic review. *PLoS Med* **4** (8): e261.

Denke MA (2001). Metabolic effects of high-protein, low-carbohydrate diets. *Am J Cardiol* **88** (1): 59-61.

Department of Health (2001). National Services Framework for Diabetes: Standards. London, DOH.

Department of Health (2003). The National Service Framework for Diabetes: Delivery Strategy. London, DOH.

Department of Health (2005). Health Survey for England. London, DOH

Department of Health (2005). Quality and Outcomes Framework (QOF). London, DOH.

de Weerdt I,Visser AP, Kok GJ, de Weerdt O, van der Veen EA (1991). Randomized controlled multicentre evaluation of an education programme for insulin-treated diabetic patients: effects on metabolic control, quality of life, and costs of therapy. *Diabet Med* **8** (4): 338-45.

Diabetes UK (2000). Recommended core staffing levels for an average district specialist diabetes care team. London, Diabetes UK.

Diabetes UK (2004). Diabetes in the UK 2004. London, Diabetes UK.

Diabetes UK (2005). Structured Patient Education in Diabetes. London, Diabetes UK and Department of Health.

Diabetes UK (2006). State of the Nation 2006. London, Diabetes UK.

Diabetes UK (2007). Diabetes: State of the Nations 2006. Progress made in delivering the national diabetes framework. London, Diabetes UK

Dick RW, Manson SM, Hansen A. L, Huggins A, Trullinger L (2007). The Native Telehealth Outreach and Technical Assistance Program: a community-based approach to the development of multimedia-focused health care information. *Am Indian Alsk Native Ment Health Res* **14** (2): 49-66.

Duncan I, Birkmeyer C, Coughlin S, Li QE, Sherr D, Boren s (2009). Assessing the value of diabetes education. *Diabetes Educ* **35**: 752-60.

EASD (2000). Recommendations for the nutritional management of patients with diabetes mellitus. *Eur J Clin Nutr* **54** (4): 353-5.

EURODIAB IDDM Complications Study Group (1994). Microvascular and acute complications in IDDM patients: the EURODIAB IDDM complications study. *Diabetologia* **37** (3): 278-285.

Fagot-Campagna A, Pettitt DJ, Engelgau MM, Burrows NR, Geiss LS, Valdez R, Beckles GL, Saaddine J, Gregg EW, Williamson DF, Narayan KM (2000). Type 2 diabetes among

North American children and adolescents: an epidemiologic review and a public health perspective. *J Pediatr* **136** (5): 664-72.

Falconnier Bendik C, Keller U, Moriconi N, Gessler A, Schindler C, Zulewski H, Ruiz J, Puder JJ (2009). Training in flexible intensive insulin therapy improves quality of life, decreases the risk of hypoglycaemia and ameliorates poor metabolic control in patients with type 1 diabetes. *Diabet Res Clin Prac* **83**: 327-33.

Fitzsimons B, Wilton L, Lamont T, McCulloch L, Boyce J (2002). The Audit Commission review of diabetes services in England and Wales, 1998-2001. *Diabet Med* **19** Suppl 4: 73-8.

Fleming C,Simmons D, Leakehe L, Voyle J (1995). Ethnic differences in the perception of a video developed for a multiethnic diabetes prevention programme in south Auckland, New Zealand. *Diabet Med* **12** (8): 701-7.

Forestfield Software Ltd (2002). Dietplan 5 for Windows. Horsham, Forestfield Software Limited.

Forouhi NG, Merrick D, Goyder E, Ferguson BA, Abbas J, Lachowycz K, Wild SH (2006). Diabetes prevalence in England, 2001--estimates from an epidemiological model. *Diabet Med* **23** (2): 189-97.

Foster GD, Wyatt HR, Hill JO, McGuckin BG, Brill C, Mohammed BS, Szapary PO, Rader DJ, Edman JS, Klein S (2003). A randomized trial of a low-carbohydrate diet for obesity. *N Engl J Med* **348** (21): 2082-90.

Franz MJ, Barr P, Holler H, Powers MA, Wheeler ML, Wylie-Rosett J (1987). Exchange lists: revised 1986. *J Am Diet Assoc* **87** (1): 28-34.

Freedson PS and Miller K (2000). Objective monitoring of physical activity using motion sensors and heart rate. *Res Q Exerc Spor* **71** (Suppl 2): S21-9.

Fuller MF, Fowler PA, McNeill G, Foster MA (1994). Imaging techniques for the assessment of body composition. *J Nutr* **124** (8) Suppl: 1546S-1550S.

Funnell MM (2006). The Diabetes, Attitudes, Wishes and Needs (DAWN) study. *Clin Diabetes* **24** (4): 154-5.

Funnell MM and Anderson RM (2004). Empowerment and self-management of diabetes. *Clinical diabetes* **22**: 123-27.

Gallen I (2006). Diabetes and sport: managing the complex interactions. *Br J Hosp Med (Lond)* **67** (10): 512-5.

Gann D (2004). A low-carbohydrate diet in overweight patients undergoing stable statin therapy raises high-density lipoprotein and lowers triglycerides substantially. *Clin Cardiol* **27** (10): 563-4.

Gannon MC, Nuttall FQ, Saeed A, Jordan K, Hoover H (2003). An increase in dietary protein improves the blood glucose response in persons with type 2 diabetes. *Am J Clin Nutr* **78** (4): 734-41.

Gannon MC and Nuttall FQ (2004). Effect of a high-protein, low-carbohydrate diet on blood glucose control in people with type 2 diabetes. *Diabetes* **53** (9): 2375-82.

George JT, Valdovinos AP, Russell I, Dromgoole P, Lomax S, Torgerson DJ, Wells T, Thow JC (2008). Clinical effectiveness of a brief educational intervention in type 1 diabetes: results from the BITES (Brief Intervention in Type 1 diabetes, Education for Self-efficacy) trial. *Diabet Med* **25** (12): 1447-53.

Gonder-Frederick LA, Clarke WL, Cox DJ (1997). The Emotional, Social, and Behavioral Implications of Insulin-Induced Hypoglycemia. *Semin Clin Neuropsychiatry* **2** (1): 57-65.

Gumbiner B, Wendel J.A, McDermott MP (1996). Effects of diet composition and ketosis on glycemia during very-low-energy-diet therapy in obese patients with non-insulin-dependent diabetes mellitus. *Am J Clin Nutr* **63** (1): 110-5.

Gerber BS, Brodsky IG, Lawless KA, Smolin LI, Arozullah AM, Smith EV, Berbaum ML, Heckerling PS, Eiser AR (2005). Implementation and evaluation of a low-literacy diabetes education computer multimedia application. *Diabetes Care* **28** (7): 1574-80.

Goldstein RB, Dugan E, Trachtenberg F, Peli E (2007). The impact of a video intervention on the use of low vision assistive devices. *Optom Vis Sci* **84** (3): 208-17.

Green LW and Kansler CC (1980). The professional and scientific literature on patient education. Detroit, Dale Research Co.

Gutierrez M, Akhavan M, Jovanovic L, Peterson CM (1998). Utility of a short-term 25% carbohydrate diet on improving glycemic control in type 2 diabetes mellitus. *J Am Coll Nutr* **17** (6): 595-600.

Ha TK and Lean ME (1998). Recommendations for the nutritional management of patients with diabetes mellitus. *Eur J Clin Nutr* **52** (7): 467-81.

Hamer M. and Chida Y (2007). Intake of fruit, vegetables, and antioxidants and risk of type 2 diabetes: systematic review and meta-analysis. *J Hypertens* **25** (12): 2361-9.

He FJ, Nowson CA, MacGregor GA (2006). Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. *Lancet* **367** (9507): 320-6.

He FJ, Nowson CA, Lucas M, MacGregor GA (2007). Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. *J Hum Hypertens* **21** (9): 717-28.

Healthcare commission (2007). Managing diabetes: improving services for people with diabetes. London, Commission for Healthcare Audit and Inspection.

Heitmann BL and Lissner L (1995). Dietary underreporting by obese individuals--is it specific or non-specific? *BMJ* **311** (7011): 986-9.

Heller SR (2008). Minimizing hypoglycemia while maintaining glycemic control in diabetes. *Diabetes* **57** (12): 3177-83.

Henderson L, Gregory J, Irvine I, Swan G (2003). The National Diet and Nutrition Survey: adults aged 19-64 years. Energy, protein, carbohydrate, fat and alcohol intake. London, HMSO.

Hippisley-Cox J and Pringle M (2004). Prevalence, care, and outcomes for patients with diet-controlled diabetes in general practice: cross sectional survey. *Lancet* **364** (9432): 423-8.

Holman RR, Thorne KI, Farmer AJ, Davies MJ, Keenan JF, Paul S, Levy JC (2007). Addition of biphasic, prandial, or basal insulin to oral therapy in type 2 diabetes. *N Engl J Med* **357** (17): 1716-30.

Honey P and Mumford A (1982). Manual of learning styles. London, P. Honey.

Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB (2001). Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med* **161** (12): 1542-8.

Hu FB, Li TY, Colditz GA, Willett WC, Manson JE (2003). Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA* **289** (14): 1785-91.

Hu G, Jousilahti P, Barengo NC, Qiao Q, Lakka TA, Tuomilehto J (2005). Physical activity, cardiovascular risk factors, and mortality among Finnish adults with diabetes. *Diabetes Care* **28** (4): 799-805.

Hussein S and Partridge M (2002). Perceptions of asthma in South Asians and their views on educational materials and self-management plans: a qualitative study. *Patient Educ Couns* **48** (2): 189-94.

International Diabetes Federation (1998). A desktop guide to Type 1 (insulin dependent) diabetes. IDF.

Ioacara S, Lichiardopol R, Ionescu-Tirgoviste C, Cheta D, Sabau S, Guja C, Farcasiu E, Tiu C (2009). Improvements in life expectancy in type 1 diabetes patients in the last six decades. *Diabetes Res Clin Pract* **86** (2): 146-51.

Jacobsen IB, Henriksen JE, Hother-Nielsen O, Vach W, Beck-Nielsen H (2009). Evidence-based insulin treatment in type 1 diabetes mellitus. *Diabetes Res Clin Pract* **86** (1): 1-10.

James WP, Bingham S.A, Cole TJ (1981). Epidemiological assessment of dietary intake. *Nutr Cancer* **2** (4): 203-12.

Janssen I, Katzmarzyk PT, Ross R (2002). Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* **162** (18): 2074-9.

Jenkins DJ, Wolever TM, Taylor RH, Barker H, Fielden H, Baldwin JM, Bowling AC, Newman HC, Jenkins AL, Goff DV (1981). Glycemic index of foods: a physiological basis for carbohydrate exchange. *Am J Clin Nutr* **34** (3): 362-6.

Johnston S, Tjonn SL, Swan PD, White A, Hutchins H, Sears B (2006). Ketogenic low-carbohydrate diets have no metabolic advantage over nonketogenic low-carbohydrate diets. *Am J Clin Nutr* **83** (5): 1055-61.

Kanters SD, Banga JD,Stolk RP, Algra A (1999). Incidence and determinants of mortality and cardiovascular events in diabetes mellitus: a meta-analysis. *Vasc Med* **4** (2): 67-75.

Kavookjian J, Elswick BM, Whetsel T (2007). Interventions for being active among individuals with diabetes: a systematic review of the literature. *Diabetes Educ* **33** (6): 962-88; discussion 989-90.

Kennedy RL, Chokkalingam K, Farshchi HR (2005). Nutrition in patients with Type 2 diabetes: are low-carbohydrate diets effective, safe or desirable? *Diabet Med* **22** (7): 821-32.

Kerstetter JE, O'Brien KO, Caseria DM, Wall DE, Insogna KL (2005). The impact of dietary protein on calcium absorption and kinetic measures of bone turnover in women. *J Clin Endocrinol Metab* **90** (1): 26-31.

Khunti K, Gadsby R, Millett C, Majeed A, Davies M (2007). Quality of diabetes care in the UK: comparison of published quality-of-care reports with results of the Quality and Outcomes Framework for Diabetes. *Diabet Med* **24** (12): 1436-41.

Kipnis V, Midthune D, Freedman L, Bingham S, Day NE, Riboli E, Ferrari P, Carroll RJ (2002). Bias in dietary-report instruments and its implications for nutritional epidemiology. *Public Health Nutr* **5** (6A): 915-23.

Knowler WC, Pettitt DJ, Savage PJ, Bennett PH (1981). Diabetes incidence in Pima Indians: contributions of obesity and parental diabetes. *Am J Epidemiol* **113** (2): 144-56.

Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* **346** (6): 393-403.

Kolb DA (1984). Experiential learning._Eaglewood Cliffs, NJ, Prentice Hall.

Laffel L (2000). Sick-day management in type 1 diabetes. *Endocrinol Metab Clin North Am* **29** (4): 707-23.

Lawrence IG, Hopkins D, Mansell P, Thompson G, Amiel S, Heller S (2008). DAFNE (Dose Adjustment for Normal Eating) training delivered in routine clinical care is associated with a significant improvement in glycaemic control and a reduction in severe hypoglycaemia. *Diabet Med* **25** (Suppl 1): 72.

Lean ME (2003). The implementation of nutritional advice for people with diabetes. *Diabet Med* **20** (10): 783-5.

Lennon GM, Taylor KG, Debney L, Bailey CJ (1990). Knowledge, attitudes, technical competence, and blood glucose control of Type 1 diabetic patients during and after an education programme. *Diabet Med* **7** (9): 825-32.

Levy JC, Matthews DR, Hermans MP (1998). Correct homeostasis model assessment (HOMA) evaluation uses the computer program. *Diabetes Care* **21** (12): 2191-2.

Li C, Ford ES, Strine TW, Mokdad AH (2008). Prevalence of depression among U.S. adults with diabetes: findings from the 2006 behavioral risk factor surveillance system. *Diabetes Care* **31** (1): 105-7.

Liu S, Serdula M, Janket SJ, Cook NR, Sesso HD, Willett WC, Manson JE, Buring JE (2004). A prospective study of fruit and vegetable intake and the risk of type 2 diabetes in women. *Diabetes Care* **27** (12): 2993-6.

Lobstein T and Leach R (2004). Diabetes may be undetected in many children in the UK. *BMJ* **328** (7450): 1261-2.

Loveman E, Frampton GK, Clegg AJ (2008). The clinical effectiveness of diabetes education models for Type 2 diabetes: a systematic review. Health Technology Assessment **12** (9). NHS R&D HTA Programme.

Lowe J, Linjawi S, Mensch M, James K, Attia J (2008). Flexible eating and flexible insulin dosing in patients with diabetes: Results of an intensive self-management course. *Diabetes Res Clin Pract* **80** (3): 439-443.

Livesey G, Taylor R, Hulshof T, Howlett J (2008). Glycemic response and health--a systematic review and meta-analysis: relations between dietary glycemic properties and health outcomes. *Am J Clin Nutr* **87** (1): 258S-268S.

Lumb AN and Gallen IW (2009). Diabetes management for intense exercise. *Curr Opin Endocrinol Diabetes Obes* **16** (2): 150-5.

Lunet N, Valbuena C, Vieira AL, Lopes C, David L, Carneiro F, Barros H (2007). Fruit and vegetable consumption and gastric cancer by location and histological type: case-control and meta-analysis. *Eur J Cancer Prev* **16** (4): 312-27.

Ma Y, Olendzki BC, Merriam PA, Chiriboga DE, Culver AL, Li W, Hebert JR, Ockene IS, Griffith JA, Pagoto SL (2008). A randomized clinical trial comparing low-glycemic index versus ADA dietary education among individuals with type 2 diabetes. *Nutrition* **24** (1): 45-56.

Malik VS, Schulze MB, Hu FB (2006). Intake of sugar-sweetened beverages and weight gain: a systematic review. *Am J Clin Nutr* **84** (2): 274-88.

Mann JI, Kinmonth AL, Todd E, Angus RM, Simpson HC, Hockaday TD (1981). High fibre diets and diabetes. *Lancet* **1** (8222): 731-2.

Mann J and McAuley K (2007). Carbohydrates: is the advice to eat less justified for diabetes and cardiovascular health? *Curr Opin Lipidol* **18** (1): 9-12.

Manley SE, Stratton IM, Cull CA, Frighi V, Eeley EA, Matthews DR, Holman RR, Turner RC, Neil H.A (2000). Effects of three months' diet after diagnosis of Type 2 diabetes on plasma lipids and lipoproteins (UKPDS 45). UK Prospective Diabetes Study Group. *Diabet Med* **17** (7): 518-23.

Manson JE, Rimm EB, Stampfer MJ, Colditz GA, Willett WC, Krolewski AS, Rosner B, Hennekens CH, Speizer FE (1991). Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* **338** (8770): 774-8.

Manson JE, Nathan DM, Krolewski AS, Stampfer MJ, Willett WC, Hennekens, CH (1992). A prospective study of exercise and incidence of diabetes among US male physicians. *JAMA* **268** (1): 63-7.

Meadows K, Steen N, McColl E, Eccles M, Shiels C, Hewison J, Hutchinson A. (1996). The Diabetes Health Profile (DHP): a new instrument for assessing the psychosocial profile of insulin requiring patients--development and psychometric evaluation. *Qual Life Res* **5** (2): 242-54.

Meckling KA, O'Sullivan C, Saari D (2004). Comparison of a low-fat diet to a low-carbohydrate diet on weight loss, body composition, and risk factors for diabetes and cardiovascular disease in free-living, overweight men and women. *J Clin Endocrinol Metab* **89** (6): 2717-23.

Meetoo D and Gopaul H (2004). Empowerment: giving power to people with diabetes. *Diabetes and Primary Care* **6** (4): 164-70.

Millett C, Car J, Eldred D, Khunti K, Mainous AG, Majeed A (2007). Diabetes prevalence, process of care and outcomes in relation to practice size, caseload and deprivation: national cross-sectional study in primary care. *J R Soc Med* **100** (6): 275-83.

Merriam-Webster (2006). Merriam-Webster's Collegiate Dictionary. Springfield.

Moore H, Summerbell C, Hooper L, Cruickshank K, Vyas A, Johnstone P, Ashton V, Kopelman P (2004). Dietary advice for treatment of type 2 diabetes mellitus in adults. *Cochrane Database Syst Rev* (2): CD004097.

Moran M (2004). The evolution of the nutritional management of diabetes. *Proc Nutr Soc* **63**(4): 615-20.

Muhlhauser I, Bruckner I, Berger M, Cheta D, Jorgens V, Ionescu-Tirqoviste C, Scholz V, Mineu I (1987). Evaluation of an intensified insulin treatment and teaching programme as routine management of type 1 (insulin-dependent) diabetes. The Bucharest-Düsseldorf Study. *Diabetologia* **30** (9): 681-90.

Nathan DM, Zinman B, Cleary PA, Backlund JY, Genuth S, Miller R, Orchard TJ (2009). Modern-day clinical course of type 1 diabetes mellitus after 30 years' duration: the diabetes control and complications trial/epidemiology of diabetes interventions and complications and Pittsburgh epidemiology of diabetes complications experience (1983-2005). *Arch Intern Med* **169** (14): 1307-16.

Nelson M, Lean ME, Connor H, Thomas BJ, Lord K, Hartland B, Waldron S, McGough N, Walker L, Ryan A, Start K (2000). Survey of dietetic provision for patients with diabetes. *Diabet Med* **17** (8): 565-71.

NICE (2002). Patient Education Models for Diabetes. London, National Institute for Clinical Excellence.

NICE (2003). Guidance on the use of patient-education models for diabetes. Technical Appraisal Guidance 60. London, National Institute of Clinical Excellence.

NICE (2004). Type 1 diabetes in adults: full guideline part 1. London, National Institute of Clinical Excellence.

NICE (2005). Clinical guidelines for type 2 diabetes. Management of blood glucose. London, National Institute of Clinical Excellence.

NICE (2008). Type 1 diabetes in adults: full guideline part 2. London, National Institute of Clinical Excellence.

NICE (2008). Type 2 diabetes: the management of type 2 diabetes (update). London, National Institute of Clinical Excellence.

Nicholas L, Pond D, Roberts DC (2005). The effectiveness of nutrition counselling by Australian General Practitioners. *Eur J Clin Nutr* **59** (Suppl 1): S140-6.

Nield L, Moore HJ, Hooper L, Cruickshank JK, Vyas A, Whittaker V, Summerbell CD (2007). Dietary advice for treatment of type 2 diabetes mellitus in adults. *Cochrane Database Syst Rev* (3): CD004097.

Nielsen JV and Joensson E (2006). Low-carbohydrate diet in type 2 diabetes. Stable improvement of bodyweight and glycemic control during 22 months follow-up. *Nutr Metab (Lond)* **3**: 22.

Nielsen JV and Joensson EA (2008). Low-carbohydrate diet in type 2 diabetes: stable improvement of bodyweight and glycemic control during 44 months follow-up. *Nutr Metab (Lond)* **5**: 14-9.

Nielsen JV, Jonsson E, Nilsson AK (2005). Lasting improvement of hyperglycaemia and bodyweight: low-carbohydrate diet in type 2 diabetes. A brief report. *Ups J Med Sci* **110** (2): 179-83.

Nielsen JV, Westerlund P, Bygren P (2006). A low-carbohydrate diet may prevent end-stage renal failure in type 2 diabetes. A case report. *Nutr Metab (Lond)* **3**: 23.

NIH (2005). Theory at a glance: A guide for health promotion practice. London, National Institute of Health.

Nordmann AJ, Nordmann A, Briel M, Keller U, Yancy WS, .Brehm BJ, Bucher HC (2006). Effects of low-carbohydrate vs low-fat diets on weight loss and cardiovascular risk factors: a meta-analysis of randomized controlled trials. *Arch Intern Med* **166** (3): 285-93.

Nothlings U, Schulze MB, Weikert C, Boeing H, van der Schouw YT, Bamia C *et al* (2008). Intake of vegetables, legumes, and fruit, and risk for all-cause, cardiovascular, and cancer mortality in a European diabetic population. *J Nutr* **138** (4): 775-81.

Nutrition sub-committee of the British Diabetic Association (1982). Dietary recommendations for the 1980s. *Human nutrition: applied nutrition* **36** (5): 378-82.

Nutrition sub-committee of the British Diabetic Association (1987). The provision of dietetic services to diabetics in the United Kingdom. *Human nutrition: applied nutrition* **41A**: 13-22.

Nutrition sub-committee of the British Diabetic Association's Professional Advisory Committee (1992). Dietary recommendations for people with diabetes. An update for the 1990s. *Diabet Med* **9**: 189-202.

Nutrition sub-committee of the Diabetes Care Advisory Committee of Diabetes UK (2003). The implementation of nutritional advice for people with diabetes. *Diabet Med* **20** (10): 786-807.

Nuttall FQ(1980). Dietary recommendations for individuals with diabetes mellitus, 1979: summary of report from the Food and Nutrition Committee of the American Diabetes Association. *Am J Clin Nutr* **33** (6): 1311-2.

Oliveira A, Gevirtz R, Hubbard D (2006). A psycho-educational video used in the emergency department provides effective treatment for whiplash injuries. *Spine* **31** (15): 1652-7.

Oxford English Dictionary (1989). The Oxford English Dictionary. Oxford, Oxford Univesity Press.

Panja S, Starr B, Colleran KM (2005). Patient knowledge improves glycemic control: is it time to go back to the classroom? *J Investig Med* **53** (5): 264-6.

Peyrot M, Rubin RR, Lauritzen T, Snoek FJ, Matthews DR, Skovlund SE (2005). Psychosocial problems and barriers to improved diabetes management: results of the Cross-National Diabetes Attitudes, Wishes and Needs (DAWN) Study. *Diabet Med* **22** (10): 1379-85.

Pierce M, Agarwal G, Ridout D (2000). A survey of diabetes care in general practice in England and Wales. *Br J Gen Pract* **50** (456): 542-5.

Pi-Sunyer X, Blackburn G, Brancati FL, Bray GA, Bright R, Clark JM *et al* (2007). Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the look AHEAD trial. *Diabetes Care* **30** (6): 1374-83.

Polonsky WH, Anderson BJ, Lohrer PA, Welch G, Jacobson AM, Aponte JE, Schwartz CE (1995). Assessment of diabetes-related distress. *Diabetes Care* **18** (6): 754-60.

Prentice AM, Black AE, Coward WA, Cole TJ (1996). Energy expenditure in overweight and obese adults in affluent societies: an analysis of 319 doubly-labelled water measurements. *Eur J Clin Nutr* **50** (2): 93-7.

Priebe MG, van Binsbergen JJ, de Vos R, Vonk RJ (2008). Whole grain foods for the prevention of type 2 diabetes mellitus. *Cochrane Database Syst Rev* (1): CD006061.

Purnell JQ, Hokanson JE, Marcovina SM, Steffes MW, Cleary PA, Brunzell JD (1998). Effect of excessive weight gain with intensive therapy of type 1 diabetes on lipid levels and blood pressure: results from the DCCT. Diabetes Control and Complications Trial. *JAMA* **280** (2): 140-6.

Rennie KL and Jebb SA (2005). Prevalence of obesity in Great Britain. *Obes Rev* **6** (1): 11-2.

Rizkalla SW, Taghrid L, Laromiguiere M, Huet D, Boillot J, Rigoir A, Elgrably F, Slama G (2004). Improved plasma glucose control, whole-body glucose utilization, and lipid profile on a low-glycemic index diet in type 2 diabetic men: a randomized controlled trial. *Diabetes Care* **27** (8): 1866-72.

Robertson A and Broom I (2002). Low carbohydrate diets in the treatment of resistant overweight patients with type 2 diabetes. *Diabetic Medicine* **19** (Suppl 2): 24.

Robertson L, Waugh N, Robertson A (2007). Protein restriction for diabetic renal disease. *Cochrane Database Syst Rev* (4): CD002181.

Rodbard HW, Blonde L, Braithwaite SS, Brett EM, Cobin RH, Handelsman Y (2007). American Association of Clinical Endocrinologists medical guidelines for clinical practice for the management of diabetes mellitus. *Endocr Pract* **13** Suppl 1: 1-68.

Rollnick S, Mason P, Butler C (1999). Health behavior change: a guide for practitioners. London, Churchill Livingstone.

Roth G. (1993). Why weight? A guide to ending compulsive eating. New York, Penguin Books.

Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC (1997). Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* **277** (6): 472-7.

Samaha FF, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams T, Williams M, Gracely EJ, Stern L (2003). A low-carbohydrate as compared with a low-fat diet in severe obesity. *N Engl J Med* **348** (21): 2074-81.

Sargrad K.R, Homko C, Mozzoli M, Boden G (2005). Effect of high protein vs high carbohydrate intake on insulin sensitivity, body weight, hemoglobin A1c, and blood pressure in patients with type 2 diabetes mellitus. *J Am Diet Assoc* **105** (4): 573-80.

Schulze MB, Manson JE, Ludwig DS, Colditz GA, Stampfer MJ, Willett WC, Hu FB (2004). Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. *JAMA* **292** (8): 927-34.

Selvin E, Marinopoulos S, Berkenblit G, Rami T, Brancati FL, Powe NR, Golden SH (2004). Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. *Ann Intern Med* **141** (6): 421-31.

Sheard NF, Clark NG, Brand-Miller JC, Franz MJ, Pi-Sunyer FX, Mayer-Davis E, Kulkarni K, Geil P (2004). Dietary carbohydrate (amount and type) in the prevention and management of diabetes: a statement by the American Diabetes Association. *Diabetes Care* **27** (9): 2266-71.

Slama G, Haardt MJ, Jean-Joseph P, Costagliola D, Goicolea I, Bornet F, Elgrably F, Tchobroutsky G (1984). Sucrose taken during mixed meal has no additional hyperglycaemic action over isocaloric amounts of starch in well-controlled diabetics. *Lancet* **2** (8395): 122-5.

Soljak MA, Majeed A, Eliahoo J, Dornhorst A (2007). Ethnic inequalities in the treatment and outcome of diabetes in three English Primary Care Trusts. *Int J Equity Health* **6**: 8.

Speight and Bradley C (2001). The ADKnowl: identifying knowledge deficits in diabetes care. *Diabet Med* **18** (8): 626-33.

Speight J, Reaney MD, Barnard KD (2009). Not all roads lead to Rome-a review of quality of life measurement in adults with diabetes. *Diabet Med* **26** (4): 315-27.

Steffenino G, Viada E, Marengo B, Canale R (2007). Effectiveness of video-based patient information before percutaneous cardiac interventions. *J Cardiovasc Med (Hagerstown)* **8** (5): 348-53.

Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF (2004). The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. *Ann Intern Med* **140** (10): 778-85.

Teoh WL, Anderson DM, Allwinkle J, Richards MG, Lamb A, Patrick AW (2010). Royal Infirmary of Edinburgh Carbohydrate Learning and Insulin Management (RECLAIM): carbohydrate counting course for both Type 1 and insulin-treated Type 2 diabetes. *Diabet Med* **27** (Suppl 1): 17.

Texas Department of State Health Services (2010). Insulin algorithm for Type 1 diabetes mellitus in children and adults. Texas.

Thomas DE, Elliott EJ, Baur L (2007). Low glycaemic index or low glycaemic load diets for overweight and obesity. *Cochrane Database Syst Rev* (3): CD005105.

Thomas D and Elliott EJ (2009). Low glycaemic index, or low glycaemic load, diets for diabetes mellitus. *Cochrane Database Syst Rev* (1): CD006296.

Toeller M, Buyken A, Heitkamp G, Milne R, Klischan A, Gries FA (1997). Repeatability of three-day dietary records in the EURODIAB IDDM Complications Study. *Eur J Clin Nutr* **51** (2): 74-80.

Tremblay GJ, Drouin D, Parker J, Monette C, Cote DF, Reid RD (2004). The Canadian Cardiovascular Society and knowledge translation: turning best evidence into best practice. *Can J Cardiol* **20** (12): 1195-8.

Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P *et al* (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* **344** (18): 1343-50.

Turner RC, Cull CA, Frighi V, Holman RR (1999). Glycemic control with diet, sulfonylurea, metformin, or insulin in patients with type 2 diabetes mellitus: progressive requirement for multiple therapies (UKPDS 49). UK Prospective Diabetes Study (UKPDS) Group. *JAMA* **281** (21): 2005-12.

Turner RC and Holman RR (1995). Lessons from UK prospective diabetes study. *Diabetes Res Clin Pract* **28** Suppl: S151-7.

UKPDS (1990). UK Prospective Diabetes Study 7: response of fasting plasma glucose to diet therapy in newly presenting type II diabetic patients, UKPDS Group. *Metabolism* **39** (9): 905-12.

UKPDS (1998). Effect of intensive blood-glucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). UK Prospective Diabetes Study (UKPDS) Group. *Lancet* **352** (9131): 854-65.

UKPDS (1998). Intensive blood-glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). UK Prospective Diabetes Study (UKPDS) Group. *Lancet* **352** (9131): 837-53.

Ulahannan TJ, Ross W, Davies FC (2007). Carbohydrate counting in type 1 diabetes: time to REACCT. *Prac Diab Int* **24** (3): 134-6.

VanItallie TB and Nufert TH (2003). Ketones: metabolism's ugly duckling. *Nutr Rev* **61** (10): 327-41.

Vartanian LR, Schwartz MB, Brownell KD (2007). Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. *Am J Public Health* **97** (4): 667-75.

Verhoeven F, van Gemert-Pijnen L, Dijkstra K, Nijland N, Seydel E, Steehouder M (2007). The contribution of teleconsultation and videoconferencing to diabetes care: a systematic literature review. *J Med Internet Res* **9** (5): e37.

Volek JS and Westman EC (2002). Very-low-carbohydrate weight-loss diets revisited. *Cleve Clin J Med* **69** (11): 849, 853, 856-8 passim.

Volek JS, Sharman MJ, Love DM, Avery NG, Gomez AL, Scheett TP, Kraemer WJ (2002). Body composition and hormonal responses to a carbohydrate-restricted diet. *Metabolism* **51**(7): 864-70.

Volek J, Sharman M, Gomez A, Judelson D, Rubin M, Watson G, Sokmen B, Silvestre R, French D (2004). Comparison of energy-restricted very low-carbohydrate and low-fat diets on weight loss and body composition in overweight men and women. *Nutr Metab (Lond)* **1** (1): 13.

Wadden TA, Butryn ML, Byrne KJ (2004). Efficacy of lifestyle modification for long-term weight control. *Obes Res* **12** Suppl: 151S-62S.

Wallace TM, Meston NM, Gardner SG, Matthews DR (2001). The hospital and home use of a 30-second hand-held blood ketone meter: guidelines for clinical practice. *Diabet Med* **18** (8): 640-5.

Weber C,Kocher S, Neeser K, Joshi SR (2009). Prevention of diabetic ketoacidosis and self-monitoring of ketone bodies: an overview. *Curr Med Res Opin* **25** (5): 1197-207.

Weltman NY, Saliba SA, Barrett EJ, Weltman A (2009). The use of exercise in the management of type 1 and type 2 diabetes. *Clin Sports Med* **28** (3): 423-39.

Westerterp-Plantenga MS, Nieuwenhuizen A, Tome D, Soenen S, Westerterp KR (2009). Dietary protein, weight loss, and weight maintenance. *Annu Rev Nutr* **29**: 21-41.

Wheeler LA, Wheeler ML, Ours P, Swider C (1985). Evaluation of computer-based diet education in persons with diabetes mellitus and limited educational background. *Diabetes Care* **8** (6): 537-44.

Wheeler ML and Pi-Sunyer FX (2008). Carbohydrate issues: type and amount. *J Am Diet Assoc* **108** (Suppl 1): S34-9.

Wiethop BV and Cryer PE (1993). Alanine and terbutaline in treatment of hypoglycemia in IDDM. *Diabetes Care* **16** (8): 1131-6.

Willi SM, Oexmann MJ, Wright NM, Collop NA, Key LL (1998). The effects of a high-protein, low-fat, ketogenic diet on adolescents with morbid obesity: body composition, blood chemistries, and sleep abnormalities. *Pediatrics* **101** (1 Pt 1): 61-7.

Williams B, Poulter NR, Brown MJ, Davis M, McInnes GT, Potter JF, Sever PS, Thom SM (2004). British Hypertension Society guidelines for hypertension management 2004 (BHS-IV): summary. *BMJ* **328** (7440): 634-40.

Williams G. and Pickup JC (2004). The handbook of diabetes. Oxford, Blackwell.

Winocour PH, Mearing C, Ainsworth A, Williams DR (2002). Association of British Clinical Diabetologists (ABCD): survey of specialist diabetes care services in the UK, 2000. 4. Dietetic services and nutritional issues. *Diabet Med* **19** Suppl 4: 39-43.

Wing RR, Marcus MD, Epstein LH, Salata R (1987). Type II diabetic subjects lose less weight than their overweight nondiabetic spouses. *Diabetes Care* **10** (5): 563-6.

Wing RR, Vazquez JA, Ryan CM (1995). Cognitive effects of ketogenic weight-reducing diets. *Int J Obes Relat Metab Disord* **19** (11): 811-6.

Wolever TM, Barbeau M, Charron S, Harrigan K, Leung S, Madrick B, Taillefer T, Seto C (1999). Guidelines for the nutritional management of diabetes in the new millenium: A position statement by the Canadian Diabetes Association. *Canadian Journal of Diabetes Care* **23** (3): 56-69.

Wolever TM, Gibbs AL, Mehling C, Chiasson JL, Connelly PW, Josse RG, Leiter LA, Maheux P, Rabasa-Lhoret R, Rodger NW, Ryan EA (2008). The Canadian Trial of Carbohydrates in Diabetes (CCD), a 1-y controlled trial of low-glycemic-index dietary carbohydrate in type 2 diabetes: no effect on glycated hemoglobin but reduction in C-reactive protein. *Am J Clin Nutr* **87** (1): 114-25.

Wolpert HA and Anderson BJ (2001). Management of diabetes: are doctors framing the benefits from the wrong perspective? *BMJ* **323** (7319): 994-6.

World Health Organisation (1995). Physical status: The use and interpretation of anthropometry. *Technical report series* Geneva, WHO.

World Health Organisation (1998). World Health Organisation (WHO) Five Well-Being Index. Geneva, WHO.

World Health Organisation (2000). Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications. Geneva, WHO.

Wright A, Burden AC, Paisey RB, Cull CA, Holman RR (2002). Sulfonylurea inadequacy: efficacy of addition of insulin over 6 years in patients with type 2 diabetes in the U.K. Prospective Diabetes Study (UKPDS 57). *Diabetes Care* **25** (2): 330-6.

Wu CL and Williams C (2006). A low glycemic index meal before exercise improves endurance running capacity in men. *Int J Sport Nutr Exerc Metab* **16** (5): 510-27.

Yancy WS, .Olsen MK, Guyton JR, Bakst R, .Westman EC (2004). A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. *Ann Intern Med* **140** (10): 769-77.

Yancy WS, Foy M, Chalecki AM, Vernon MC, Westman EC (2005). A low-carbohydrate, ketogenic diet to treat type 2 diabetes. *Nutr Metab (Lond)* **2**: 34.

Yang YL, Wang KJ, Chen WH, Chuang KC, Tseng CC, Liu C (2007). Improved satisfaction of preoperative patients after group video-teaching during interview at preanesthetic evaluation clinic: the experience of a medical center in Taiwan. *Acta Anaesthesiol Taiwan* **45** (3): 149-54.

Zhou BF, Stamler J, Dennis B, Moag-Stahlberg A, Okuda N, Robertson C, Zhao L, Chan Q, Elliott P (2003). Nutrient intakes of middle-aged men and women in China, Japan, United Kingdom, and United States in the late 1990s: the INTERMAP study. *J Hum Hypertens* **17** (9): 623-30.

Zimmet P, Alberti KG, Shaw J (2001). Global and societal implications of the diabetes epidemic. *Nature* **414** (6865): 782-7.

Zimmet P, Magliano D, Matsuzawa Y, Alberti G, Shaw J (2005). The metabolic syndrome: a global public health problem and a new definition. *J Atheroscler Thromb* **12** (6): 295-300.

Zinman B (1998). Glucose control in type 1 diabetes: from conventional to intensive therapy. *Clin Cornerstone* **1** (3): 29-38.

Appendix 1

PAGES NOT SCANNED AT THE REQUEST OF THE UNIVERSITY

SEE ORIGINAL COPY OF THE THESIS FOR THIS MATERIAL

Dietary advice for people with diabetes: the role of carbohydrate in dietary treatment and an assessment of video education

Pamela Ann Dyson

A thesis submitted in partial fulfilment of the requirements of Oxford Brookes University for the degree of Doctor of Philosophy

Volume II

July 2010

Table of Contents

Appendix 1

Copies of ethical approval for studies

BS/JW/04/Q1606/39

Oxford Clinical Research Ethics Committee
Room 13, Manor House
The John Radcliffe Hospital
Headley Way
Headington
Oxford OX3 9DZ

Tel: 01865 222548
Fax: 01865 222699

Ms Pamela Dyson
Research Dietitian
OCDEM
Churchill Hospital
Oxford OX3 7LJ

23 June 2004

Pam

Dear ~~Ms Dyson~~

Re: OxREC 04/Q1606/39 – Low Carbohydrate Ketogenic Diet Study

Thank you for your letter of June 1st 2004 enclosing copies of the documentation for the above study. The Committee met on Friday 18th June and after discussion at this meeting, the Committee agreed approval of this study.

In accordance with the authority set out in the Terms of Reference, I am happy to confirm ethical approval and wish you every success with this study.

Please note:

- Ethical approval is valid for three years, subject to submission of a yearly progress report (a reminder letter will be sent when this is due).
- No changes to the research protocol should be made without appropriate research ethics approval. Any deviations from or changes to the protocol which increase the risk to subjects, or affect the conduct of the research, or are made to eliminate hazards to the research subjects should be made to OxREC.
- OxREC should be made aware of any serious adverse events.

Please ensure that a copy of any publication arising from this study is sent to OxREC.

Yours sincerely

Brian

Dr Brian Shine
Chairman
Oxfordshire Research Ethics Committee A

NHS

BS/JW/C03.097

Oxfordshire Clinical Research Ethics Committee
Room 13, Manor House
The John Radcliffe Hospital
Headley Way
Headington
Oxford
OX3 9DZ

Tel: 01865 222548
Fax: 01865 222699
Email: joanne.westhead@orh.nhs.uk

Miss Susan Beatty
Research Nurse
OCDEM
Churchill Hospital
Oxford
OX3 7LJ

Dear Miss Beatty

20 January 2004

Re: C03.097 - Oxford Healthy Living for Type 2 Diabetes - A Media Based Learning Programme. Protocol Version 1.2

Thank you for attending the meeting on Friday 9th January 2004 to discuss the above named study, addressing the concerns raised by the Committee at their meeting on 5th December 2003. After discussion at the meeting on 9th January and the Committee expressed the following views:

1. The power of the trial was discussed and because of the nature of the study, large numbers of patients are required. The study might produce a negative conclusion if the trial numbers were too small.
2. The video would enable patients to review information in their own home and in their own time. The videos captures the essence of what the Dietician would say in person. The aim is that the patients can either meet with the Dietician or take a video home.
3. It was decided that the upper age limit of 75 years was to be removed.
4. The matter of telephoning patients was explained, highlighting that it was simply to keep track of the videos that have been distributed and the call would only last 30 seconds. It was confirmed that the patient would not be asked for any further information.
5. The matter of creating the videos was discussed. The study needs funding to make the videos and they need ethical approval before they are able to get the funding.

In accordance with the authority set out in the Terms of Reference, I am happy to confirm ethical approval and wish you every success with the study.

Please note:

- Ethical approval is valid for three years, subject to the submission of a yearly progress report (a reminder letter will be sent when this is due).

- No changes to the research protocol should be made without appropriate research ethics committee/chairman's approval. Any deviations from or changes to the protocol which increase the risk to subjects, or affect the conduct of the research, or are made to eliminate hazards to the research subjects, should be made known to OxA.

- OxA should be made aware of any serious adverse events.

- Whilst the study has received approval on ethical grounds, it is necessary for you to obtain management approval from the relevant Clinical Directors and/or Chief Executive of the Trusts (or Health Boards/StHAs) in which the work will be done.

I should be very grateful if you could send me a copy of any publication which may arise from this study.

The committee would also need to see a copy of the video before it is given to patients. Once the video has been made, a copy should be forwarded to the committee for review.

Yours sincerely,

Brian

Dr Brian Shine
Chairman
Oxfordshire Research Ethics Committee A

This committee replaces OxREC (Oxfordshire Clinical Research Ethics Committee)
All correspondence relating to this study should be directed to Oxfordshire Research Ethics Committee A

S. Beatty

Oxford Radcliffe Hospitals NHS

NHS Trust

JM/JS/C03.097

05 NOV 2003

Professor David Matthews
OCDEM
Churchill Hospital
Old Road
Headington
Oxford OX3 7LJ

From the Medical Director
Dr James Morris, FRCPath
c/o R&D Office, Manor House
The John Radcliffe Hospital
Headley Way, Headington
Oxford OX3 9DZ

Tel: (01865) 222672
Fax: (01865) 222699
Email: julie.shankland@orh.nhs.uk

Dear Professor Matthews

28 October 2003

Re: C03.097 Oxford Healthy Living for Type 2 Diabetes – A media based learning programme

I can confirm that the Oxford Radcliffe Hospitals NHS Trust will provide indemnity for the above study, as described in your application to the Research Ethics Committee. This confirmation is dependent on the formal approval of the Research Ethics Committee and on the understanding that you have a contract of employment with this Trust.

I wish you every success with the study.

Yours sincerely,

Dr James Morris, FRCPath
Medical Director

CC Joanne Westhead, Ethics Administrator

Appendix 2

Subject information sheets

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7LJ
Tel: 01865 857333
Fax: 01865 857311

OxREC no: 04/Q1606/39
Version 2.
May 2004

Low Carbohydrate Ketogenic Diet Study

Patient Information Sheet

You are being invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

Q. What is this study about?

A. The aim of this 12-week study is to examine the effects of a very low carbohydrate diet in people who do and do not have diabetes.
It is designed to measure the amount of weight lost and level of ketones in the blood while on this type of diet. In order to do this we will investigate ketone levels in two groups of people; one group maintaining a standard healthy eating weight reducing diet and one group following a low carbohydrate diet

Q. What are ketones?

A. When eating a normal diet the body digests the carbohydrate we eat and it turns into glucose that provides us with energy. When following a low carbohydrate diet the body uses our stores of fat instead of glucose and ketones are produced as a result of the breakdown of this fat.

Q. What is meant by a low carbohydrate diet?

A. This type of diet is one where you would eat a very small amount of carbohydrate each day. This means that you would restrict the amount of starchy and sugary foods such as cake, sweets, biscuits bread, potatoes; but you are able to eat most other types of food. We will give you an opportunity to discuss the diet in full and give you a leaflet outlining the diet when you come for your first visit.

Q. What is a standard healthy weight reducing diet?

A. This type of diet concentrates on reducing the amount of fatty and sugary foods eaten and increasing the amount of fruit and vegetables. You will be given specific advice about the quantity of food you need to eat.

Q. Why have I been chosen?

A. You may have said in the past that you are interested in helping with our research or you may have been approached by your doctor or dietician and have expressed a wish to take part.

Q. Do I have to take part?

A. It is up to you to decide whether or not to take part. If you do decide to take part you would be given this information sheet to keep and be asked to sign a consent form. If you do decide to take part, you are still free to withdraw at any time and without giving any reason. This would not affect the standard of medical care you receive.

Q. What will happen to me if I take part?

A. This is a short study over 12 weeks and would mean 5 visits in total to the Oxford Diabetes Centre at the Churchill Hospital

- The first visit, which would last about 2 hours, would be to discuss the study, and if you agree to participate you would be asked to sign a consent form. We will do a short test called a glucose tolerance test. This involves giving you a glucose drink and measuring your blood glucose levels four times over the next two hours. We would also record some baseline measurements such as height and weight and take a blood sample. We will also ask you to complete 2 questionnaires.

- The next visit would be to allocate (randomise) you into one of two groups, one following a low carbohydrate diet and the other following a standard healthy eating weight reducing diet. The diet will be fully explained to you and you will have the opportunity to discuss this with the dietician.
  We randomise people into different groups because sometimes we do not know which way of treating patients is best and this allows us to make comparisons. The groups are selected by a computer, which has no information about the individual so it works by chance. Patients in each group then have a different diet and the effects of these are compared.

- You will be contacted by telephone once a week by the research nurse to offer support and to monitor your blood glucose and ketone levels.

- The next two visits would take about ½ hour. During these visits we will record your weight, record your blood pressure and take a small blood sample. You will also have the opportunity to discuss any queries you have about the study at this time.

- The final visit will take about 2 hours. We will again record your weight, record your blood pressure, take a small blood sample, repeat the glucose tolerance test and complete 2 questionnaires. If you have been allocated to the group that have maintained their normal diet, you will have the opportunity to discuss the low carbohydrate diet in more detail and be offered support if you wish to try the diet.

Q. What do I have to do during the study?

A. The study lasts for 12 weeks, during which we would expect you to maintain the diet you have been allocated. We would also ask that you complete a food diary for 3 days four times during the study and that you monitor your own blood glucose and ketone levels up to four times a day, in the early stages of the study although this will be reduced after the first week.

Q. How do I measure my own blood ketone and glucose levels?

A. We would also ask you to monitor your own blood ketone and glucose levels using a small meter. This would mean that you would use a finger-pricking device to get a small drop of blood that you place on a special strip that the meter can read. When you come for your first visit we will show you the meter and how to use it.

Q. What would happen to the blood samples taken during the study?

A. Some of the blood samples will be sent to the laboratory at the John Radcliffe Hospital for analysis. These samples will have your name on them and be destroyed immediately. A small part of the blood sample will be sent to the laboratories at the Diabetes Centre. These samples will only be identified by a number and will be stored for up to one year following completion of the study. These samples may be tested for two hormones (insulin and c-peptide) as part of other studies, however it is important to note that these samples will not have your name on them.

Q. Are there any side effects of the diets used in the study?

A. There have been some mild side effects reported when following a low carbohydrate diet, usually headaches, constipation and lethargy. If this happens to you, you will be given advice to reduce these side effects.

Q. What are the advantages of taking part in the study?

A. Both of these diets have been shown to be an effective way to lose weight. During the study you may have the opportunity to try the low carbohydrate diet under close

supervision and have weekly contact with a nurse or dietician to discuss any aspect of the diet. If you are asked to follow the healthy eating diet, you will be given the opportunity to try the low carbohydrate diet at the end of the study if you wish.

Q. What are the disadvantages of taking part in the study?

A. You will be asked to visit the Oxford Diabetes Centre five times and asked to give a small blood samples during the study. You will also be asked to use a finger-pricking device to enable you to monitor your own blood glucose and ketone levels on a regular basis. You may have some of the mild side effects we have described.

Q. Is my GP informed that I am taking part in the study?

A. Yes, if you agree, your GP will be informed by letter once you agree to take part in the study

Q. What happens if I do not want to continue in the study?

A. You can withdraw from the study at any time, without giving any reason and without your medical care or legal rights being affected in any way.

Q. Would I be paid for taking part in the study?

A. You will not be paid for taking part in the study, however all travelling expenses will be refunded.

Q. What if new information becomes available?

A. Sometimes during the course of a research project, new information becomes available about the diets that are being studied. If this happens the research doctor will tell you about it and discuss with you whether you want to continue in the study. If you decide to withdraw your medical care will not be affected in any way, and if you decide to continue in the study you will be asked to sign an updated consent form.

Q. What if something goes wrong?

A. Indemnity for the study is provided by the Oxford Radcliffe Hospitals NHS Trust. There are also established procedures if you wish to make a complaint at any time and normal NHS complaints mechanisms are available to you.

Q. Would my taking part in this study be kept confidential?

A. All information that is collected about you during the course of the study would be kept strictly confidential. All information will be stored under the guidelines of the Data Protection Act 1999, and under University of Oxford Data Protection Policy.

Q. What will happen to the results of the research study?

A. The results will be published in a professional journal and sent for display to professional meetings. You will not be identified in any way in any publication that arises as a result of this study. You will also be informed by letter of the results of the study.

Q. Who is organising and funding the research?

A. The research is organised by a team at the Oxford Centre for Diabetes, Endocrinology and Metabolism, Churchill Hospital, Oxford and is funded by a grant from Abbott Laboratories (Medisense) UK.

Q. Who can I talk to about taking part in the study?

A. You can contact Sue Beatty, Research Nurse at the Oxford Centre for Diabetes, Endocrinology and Metabolism. Telephone 01865 857333

Thank you

The Oxford Centre for Diabetes, Endocrinology and Metabolism

OxREC no: C03.097
December 2004
Version 1.5

HEALTHY LIVING STUDY

A MEDIA BASED LEARNING PROGRAMME

PATIENT INFORMATION SHEET

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take the time to read the following information carefully and ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this

Q. What is this study about?

A. We are developing a home-based education programme for people with type 2 diabetes. We have produced 3 videos, each lasting 10 minutes, covering different aspects of diabetes that you could watch at home. This will address issues such as: 'What should I eat?' Physical activity and fitness', and 'Weight Management'. We are interested in what you think about this way of giving people information and whether it has any effect on the way you manage your diabetes.

Q. How long is the study?

A. Your participation in the study will be for 6 months although the study itself will last for 2 years.

Q. Do I have to take part?

A. It is up to you to decide whether or not to take part. If you do decide to take part you would be given this information sheet to keep and be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving any reason. This would not affect the standard of care you receive.

Q. How often do I have to come to the hospital?

A. Once you have agreed to take part in the study, we would ask that you visit the Oxford Diabetes Centre twice in a period of six months. We may be able to arrange to visit you at your local surgery.

Q. What will I have to do during the study?

A. The main part of the study involves you watching the videos mentioned above. We would also ask that you keep a food diary for 3 days and monitor your levels of physical activity using a pedometer (a small thing that you wear which counts the steps you take) at the beginning and end of the study. We would also ask you to complete questionnaires that will indicate your knowledge of diabetes, your quality of life and evaluation of the videos.

Q. What will happen at each study visit?

A. You will have the opportunity to discuss the study and we will record weight and blood pressure and take a small blood sample. This sample will allow us to measure and monitor any changes in your blood sugar and cholesterol levels. These samples will be destroyed at the end of the study and will not be used for any other purposes.

Q. Would my medical notes need to be looked at?

A. We would only access your medical notes if you give permission to do so and your notes will only be accessed by health professionals. We may need to look at your notes to clarify information and to document any relevant medical history.

Q. Would my taking part in the study be kept confidential?

A. All information that is collected about you during the course of the study would be kept strictly confidential. All information will be stored under the guidelines of the Data Protection Act 1999.

Q. What happens if I do not want to continue in the study?

A. You can withdraw from the study at any time, without giving any reason and without your medical care or legal rights being affected in any way.

Q. What are the advantages of the study?

A. The videos will give you an opportunity to find out more about diabetes and give you some advice about managing life with diabetes. You will also have contact with a specialist research nurse.

Q. What are the disadvantages of the study?

A. You may need to come to the Oxford Diabetes Centre twice in 6 months and you will have to give a small blood sample at each visit.

Q. Will I be paid for taking part?

A. You will not be paid for taking part. However, all travel expenses will be refunded.

Q. What if something goes wrong?

A. Indemnity for the study is provided by the Oxford Radcliffe Hospitals NHS Trust. There are also established procedures if you wish to make a complaint at any time normal NHS complaints mechanisms are available to you.

Q. Who can I speak to about taking part in the study?

A. You can contact Sue Beatty, Research Nurse at the Oxford Centre for Diabetes, Endocrinology and Metabolism. Telephone 01865 857333

Appendix 3

Consent forms

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7LJ
Tel: 01865 857333
Fax: 01865 857311

OxREC no: 04/Q1606/39
Version 2
July2004

CONSENT FORM

Title of Project: Low Carbohydrate Ketogenic Diet Study

Name of Researcher: Professor D Matthews

Please initial box

1. I confirm that I have read and understand the information sheet Dated July 2004,(version 3) for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I understand that sections of any of my medical notes may be looked at by responsible individuals from the Oxford Diabetes Centre and I give permission for these individuals to have access to my records. ☐
4. I agree that part of the blood samples taken during the study will be stored, with full anonymity, for one year after the completion of the study. ☐
5. I agree that these blood samples may be used for further study after the samples are anonymised and it will not be possible to withdraw this consent in the future. ☐
6. I agree that my GP will be informed about my participation in the study. ☐
7. I agree to take part in the above study. ☐

Name of Patient	Signature	Date
Name of Person taking consent (if different from researcher)	Signature	Date
Researcher	Signature	Date

1 for patient; 1 for researcher; 1 to be kept with hospital note

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7LJ
Tel: 01865 857333
Fax: 01865 857311

Ox REC no: C0.097
Version 1.4
December 2004

CONSENT FORM

Title of Project: Oxford Healthy Living for Type 2 Diabetes
A Media Based Learning Programme

Name of Researcher: Professor D Matthews

Please initial box

7. I confirm that I have read and understand the information sheet dated December 2004,(version 1.5) for the above study and have had the opportunity to ask questions. ☐

8. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

9. I understand that sections of any of my medical notes may be looked at by responsible individuals from the Oxford Diabetes Centre and I give permission for these individuals to have access to my records. ☐

4. I agree to take part in the above study. ☐

Name of Patient	Signature	Date
Name of Person taking consent (if different from researcher)	Signature	Date
Researcher	Signature	Date

1 for patient; 1 for researcher; 1 to be kept with hospital notes

Appendix 4

Low carbohydrate diet sheet

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

LOW CARBOHYDRATE KETOGENIC DIET STUDY

INFORMATION

Low Carbohydrate Diet

Introduction

This diet is high in protein (found in meat, fish, eggs, cheese) and very low in carbohydrate (starchy and sugary foods). This will make your body use its fat stores for energy and will help you to lose weight and to control your blood glucose levels. To make this diet work you will have to follow it very carefully.

How will I know this diet is working?

When you use your body's fat stores for energy your body produces substances known as **ketones**. You will learn how to measure the levels of ketones in your blood using a special meter. If you already measure your blood glucose levels, then you will be familiar with this process.

What can I eat?

You can include the following protein foods daily:

Fresh, lean meat	-	beef, pork, lamb
Processed meats	-	lean bacon, gammon, ham, corned beef, brawn
Poultry	-	chicken, turkey, duck, goose
Offal	-	liver, kidney, heart, oxtail, tongue, tripe
Game	-	rabbit, hare, venison, wild boar, pheasant, partridge
Fish	-	white fish - cod, plaice, haddock, sole, coley oily fish - salmon, trout, tuna, sardines, mackerel
Shellfish	-	prawns, crab, shrimp, cockles, mussels
Eggs	-	boiled, poached, scrambled, omelette
Cheese	-	no more than 50g (2oz) each day of hard cheese **or** 100g (4oz)cottage cheese

Remember - some of these foods are very high in fat. To speed up weight loss and for general health you can reduce your fat intake by cutting all the visible fat off the meat that you eat, removing the skin from poultry and roasting, grilling, stewing or dry-frying rather than frying these foods.

Are there any foods I should avoid?

This diet restricts carbohydrate foods to 40g each day. Carbohydrates are found in all starchy and sugary foods:

Sugar, sweets, chocolate
Jam, marmalade, honey, syrup
Puddings, desserts, ice-cream
Bread, biscuits (sweet and savoury), cakes, pastries
Breakfast cereals, porridge
Potatoes, pasta, noodles, rice
Savoury snacks – crisps, twiglets, corn snacks, Bombay mix

There are other foods that contain carbohydrate:

Milk and yogurt
Fruit and vegetables

Try to avoid all starchy and sugary foods and take your daily portion of carbohydrate in the form of milk, fruit and vegetables.

What about fat?

Fat does not contain any carbohydrate but it is not good for your health to eat too much fat and it could slow down the rate of weight loss. Try to avoid too much of the following fatty foods:

Butter and margarine
Vegetable oils - blended oils, olive, sunflower, corn and rapeseed oil
Cream
Fried foods

Is there a daily food plan I can use?

Each day you should include the following foods and this will give you **40g** of carbohydrate (**CHO**) daily.

- 200ml (⅓pt) skimmed or semi-skimmed milk (enough for tea and coffee) **or** 150g (6oz) natural or diet yogurt **= 10g CHO**
- **either** 2 portions of fruit **(20g CHO)**and 2 portions vegetables **(10g CHO)** - see lists below
- **or** 1 portion of fruit **(10g CHO)**and 4 portions of vegetables **(20g CHO)** - see lists below
- At least 2 litres (4 pints) of fluid daily (see list)

Fruit

1 portion of fruit will give **10g CHO**. Choose your portion sizes from this list:

100g (4oz)	150g (6oz)	200g (8oz)	400g (1lb)
Apples	Apricots, fresh	Blackberries	Gooseberries
Cherries	Blackcurrants	Guava	Rhubarb
Clementines	Fruit cocktail -	Melon	
Kiwi fruit	canned in juice	Raspberries	
Nectarines	Grapefruit		
Oranges	Passion fruit		
Paw-paw	Strawberries		
(papaya)			
Peaches			
Pears			
Pineapple			
Plums, damsons			
Satsumas			
Tangerines			

The following fruit is high in carbohydrate and should be avoided:

- bananas, grapes, lychees, mangoes,
- all dried fruit (currants, raisins, sultanas, prunes, figs, dates, apricots)
- all fruit canned in syrup (check labels for fruit canned in juice)
- glace cherries, mixed peel

Vegetables

1 portion of vegetables will give you **5g CHO**. Choose your portion sizes from this list:

100g (4oz)	200g (8oz)	400g (1lb)
Beansprouts	Aubergine	Asparagus
Beetroot (pickled)	Baby corn	Broccoli
Brussels Sprouts	Cabbage (boiled)	Celery
Cabbage (raw)	Cauliflower (boiled)	Cucumber
Carrots (boiled)	Chicory	Curly kale (boiled)
French beans	Courgette (boiled)	Fennel
Green beans	Gherkin (pickled)	Gourd (karela)
Mangetout peas	Green pepper	Lettuce
Onions(boiled, pickled)	Leeks	Mushrooms
Runner beans	Marrow	Mustard and cress
Swede, turnip (raw)	Okra	Spinach (boiled)
Tomatoes (raw, canned)	Pumpkin	Spring greens (boiled)
	Radish	Watercress
	Spring onion	
	Swede, turnip (boiled)	

The following vegetables are high in carbohydrate and should be avoided:

- all starchy root vegetables - potatoes, parsnips, sweet potatoes, yams, beetroot (except pickled)
- all types of peas and sweetcorn (except baby corn)
- dried peas and beans - baked beans, lentils, kidney beans, butter beans, processed peas, chick peas
- raw carrots, raw onion
- mixed vegetables
- red and yellow peppers

Fluids

Try to drink at least 2 litres (4 pints) fluid daily. Choose from these drinks

- Water - tap water and bottled mineral water (still or sparkling)
- Tea and coffee - use milk from allowance
- Oxo, Bovril, Marmite, Vegemite
- Diet fizzy drinks and squashes - check the label as some reduced sugar drinks still contain carbohydrate

Meal suggestions

Remember that you should eat no more than **40g CHO** each day.

Breakfast

These meals all contain little or no carbohydrate (**0g CHO**):

Eggs - boiled, poached or scrambled (cooked with milk from allowance)
Bacon and egg
Bacon, egg and mushrooms
Grilled or poached kippers
Smoked haddock and poached egg
Grilled kidneys, bacon and mushrooms
Cheese omelette

Add 100g (4oz) fresh or canned tomatoes to the above suggestions and the meals then contain **5g CHO**

Add a glass (125ml, 4fl oz) of unsweetened fruit juice and you add **10g CHO**

Mid-day or evening meal

Choose any lean meat, fish, game, offal or eggs – these provide **0g CHO**

Each portion of vegetables you eat will add **5g CHO** – see suggestions below:

Cold meat eg ham, turkey, chicken, beef, pork with a large salad made from lettuce, cucumber, celery, watercress, mustard and cress (see vegetable lists list for portion sizes)
Roast meat eg lamb, beef, pork, chicken, turkey or game with boiled or steamed vegetables - broccoli, cauliflower, courgettes, spinach, spring greens, cabbage or swede (see vegetables lists for portion sizes)
Fish - any type steamed, grilled, fried, tinned (in either brine or oil, not tomato sauce) with salad or vegetables as above
Omelette - cheese, ham or mushroom with salad and vegetables as above

Each portion of fruit that you add provides **10g CHO**

To add some variety to your meals ask about the low carbohydrate recipe book.

What about snacks between meals?

You may take two or three weeks to get used to this diet. If you feel hungry between meals during this time then try the following as snacks:

Cold meats – ham, beef, lamb, pork, chicken portions, hard boiled eggs, cheese, olives, handful of peanuts, celery sticks, cucumber sticks

Can I drink alcohol?

You do not need to avoid alcohol entirely but it is recommended that you avoid all alcohol containing carbohydrate and that you drink within healthy limits. Alcohol can be measured in units (1 unit = a small glass of dry wine or a pub measure of spirits), and it is recommended that men should have no more than 3 units a day and women no more than 2 units a day. You should also try to have at least 2 days each week without alcohol.

These drinks contain carbohydrate and must be avoided – beer, lager, cider, sweet and medium wines, port, sherry, liqueurs and all types of alcopops.

You may drink all types of spirits (gin, vodka, whisky, rum, brandy) with or without sugar-free mixers and dry red or white wines.

Can I eat manufactured or convenience foods?

As long as you do not eat more than 40g of carbohydrate each day, you can include manufactured or convenience foods. Unfortunately, many of these foods contain large quantities of carbohydrate and you will not be able to include them in your diet. It is recommended that you always check the nutrition information on the label before you buy these foods.

Some commonly asked questions about this low carbohydrate diet

How quickly will I lose weight?

During the first week you may find that you lose weight quite quickly and this is related to fluid loss, so you may notice that you pass more urine than normal. After this time you will find that you lose weight at the rate of about 0.5 – 1.0kg (1-2lb) per week. You may have times when weight loss is either faster or slower than this.

Are there any side-effects?

There are two main reported side-effects. First, you may feel light-headed or have headaches or palpitations for the first 7-10 days of this diet. Most people report that they feel better if they drink plenty of water during these first few days. If you have palpitations for the first time or if they get worse, you should see your doctor.

Secondly, you may find that you have fewer bowel movements than before. On this diet, it is quite common to have only three bowel movements each week. You are not constipated unless you are straining to pass a motion. If you do think that you are constipated, you may find that drinking more fluid can help. Do not take laxatives unless they are recommended or prescribed by your doctor.

What about my medication?

You can continue with your usual medication. If you take insulin or tablets to treat diabetes, then you will be told how to change these to make sure your blood glucose stays at a safe level.

Do I need to take any vitamin or mineral supplements?

This diet may not give you all the vitamins or minerals that you need. You should take the supplements prescribed by the doctor to keep you healthy on this diet.

My friend needs to lose weight – can I give her this diet?

This diet has been prescribed for you by the doctor and should only be used under medical supervision. It is unsafe to give it to anyone else.

Appendix 5

Video design questionnaires

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7LJ
Tel: 01865 857300
Fax: 01865 857305

Oxford Healthy Living Media-Based Learning Programme

We are designing and evaluating videos to be used in education in people with type 2 diabetes and would be very grateful if you could take a few minutes to complete this form. We are interested in what you think should be included in an education programme giving advice about lifestyle (food and physical activity).

1. Please tick any of the following subjects that you think may be of importance and should be included in the education programme:

Foods that affect blood glucose levels	
Foods that are related to heart disease	
Sugar	
Fruit and vegetables	
Fatty foods	
Salty foods	
Glycaemic index	
Alcohol	
Losing weight	
Physical activity (exercise)	

2. Please list below what you consider to be the 3 most important topics from the list above:

3. Is there anything else that has not been mentioned that you think should be included in the video?

Appendix 6

Voice-over script

Food

Title : So what can you eat?

VO: Managing blood glucose levels effectively means getting the balance right with food, medication and physical activity. Food raises blood glucose level, but not all food has a similar effect.

Title : Which foods have the most effect on blood glucose?

VO: Foods which raise blood glucose are foods which contain carbohydrate. Carbohydrates are found in four groups of food.

The first group is sugary foods, these food are rich in carbohydrate and will raise blood glucose quite quickly. They also often contain fat and calories, and large amounts can cause weight gain and high blood glucose levels.

These foods do not need to be avoided entirely, and small amounts can be included.

The second group is starchy foods. These foods contain vitamins and minerals and are a healthy source of carbohydrate. They should form the basis of the dietary carbohydrate and are found in all starchy foods; potatoes, bread, pasta rice and breakfast cereals.

The third group is fruit and fruit juices. Fruit and vegetables are important for good health and at least five servings a day should be eaten, but large amounts of fruits will raise blood glucose levels.

The fourth group is milk and yoghurt.

Title : What about fat?

VO: People with type 2 diabetes are at an increased risk of coronary heart disease and should be advised to reduce the amount of saturated or animal fat they eat, and use mono-saturated fat like olive oil, or rapeseed oil, or polyunsaturated fat like corn or sunflower oil.

Title : So what is a healthy diet for diabetes?

A healthy diet should consist of, low fat foods, small amounts of saturated fat, like butter, cheese and fatty meat, and using more fish, poultry and lean meat. Substitute olive or corn oil for lard or butter.

Reduce sugar intake, use artificial sweeteners rather than sugar and include small amounts of sugary foods.

Moderate amounts of starchy foods, remember they do have an effect on blood glucose levels, and large amounts will raise levels.

At least five servings of fruit and vegetables each day. Most green leafy vegetables and salads will have little effect on blood glucose, so these can be included freely.

Physical Activity

Title : Type 2 Diabetes / Physical Activity

VO: Physical activity is important for general health and helps control blood glucose levels in people with diabetes

Title : So how do you do it?

VO: An easy way to start is to think of ways to increase your daily physical activity. You could try walking more, always use the stairs, or take up gardening.

If you want to take up something more strenuous like jogging or aerobics then its best to start slowly and work up gradually.

Title : Is it safe to start exercising?

VO: If your going to increase your general daily physical activity then it is quite safe to do. If you want to do something more strenuous like going to the gym, jogging or aerobics, then it's best to check with your doctor first. You should always wear supportive footwear and never exercise if you feel unwell.

If you're taking some medication for diabetes, and these drugs are called sulphonylureas, then you may find your blood glucose can go too low during exercise. If you think this is the case for you always keep some food or drink, like sports drinks, bananas or biscuits handy, so if your blood glucose levels go too low you can have something to eat or drink.

Weight Management

Title : What's the best way to lose weight?

VO: The only way to lose weight successfully is to eat less but there is not just one way to achieve this. The best way to lose weight is the way that suits you and fits in with your lifestyle. Here are some strategies to think about.

First of all, you could try adopting a healthy diet.

Another strategy is a calorie controlled diet. These diets do control the amount of food that you're eating, but they can be prescriptive, but they do suit people who like a set routine.

Or you could try joining a slimming club. Some people find the support they get from members of slimming clubs like weight watchers or Rosemary Conley can help them lose weight.

Appendix 7

Feedback forms

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7 LJ
Tel: 01865 857399
Fax: 01865 857368

The Healthy Living Programme – Evaluation form

Thank you for taking the time to look at these videos and for completing this evaluation form. Please mark the line at the side of each question with a cross at the point that matches your personal opinion.

Overall evaluation

	completely useless	***neither useful nor useless***	***very useful***
How useful were these videos?	|----------	----------|----------	----------|

What do you think of the:

	poor	***average***	***excellent***
idea of using videos to give this type of information?	|----------	----------|----------	----------|
presentation?	|----------	----------|----------	----------|
ease-of-use?	|----------	----------|----------	----------|

	too little	***about right***	***too much***
amount of information?	|----------	----------|----------	----------|

Do you have any general remarks about the three videos?

Please choose one section – food, physical activity or weight management and complete the evaluation overleaf

Section .....(please write food, activity or weight)

What do you think of the:

	poor	*average*	*excellent*
idea of using videos to give this type of information?	I----------I	----------I	----------I
presentation?	I----------I	----------I	----------I
ease-of-use?	I----------I	----------I	----------I
	too little	*about right*	*too much*
amount of information?	I----------I	----------I	----------I

Are there any changes you could suggest to improve this video?

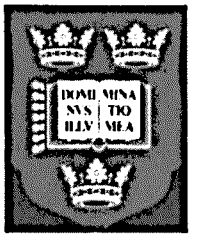
Is there anything you would leave out?

Are there any topics would you like to see included in another video?

Please add any other comments below

Appendix 8

3-day food diary



Low Carbohydrate Ketogenic Diet Study

FOOD DIARY

Name .....

Subject No ☐☐☐

Date ☐☐ ☐☐ ☐☐
D D M M Y Y

This diary is designed to obtain accurate information about the type and quantity of food you eat

Please answer the General Question section and then go onto the Food Diary

Which type of bread do you usually eat?

White ☐ Brown / Hovis ☐

Granary ☐ Wholemeal ☐

None ☐ Other ☐

Please specify .....

What size / type of bread do you buy?

Large ☐ Sliced ☐

Small ☐ Unsliced ☐

If you eat any type of biscuit regularly, please specify which brands.

.....

Which type of milk do you usually use?

Full cream milk ☐ Semi-skimmed milk ☐

Skimmed milk ☐ Channel Islands ☐

Evaporated ☐ None ☐

Other ☐

How much milk do you usually drink?

1-2 pints daily ☐ 1/2—1 pint daily ☐

1/4—1/2 pint daily ☐ None ☐

How many tablespoons of milk do you take in tea and coffee?
(please enter amount in box e..g. 1/2, 1 tablespoon)

☐ Tablespoons milk in tea None ☐

☐ Tablespoons milk in coffee

How much sugar do you take in your tea and coffee?

☐ Teaspoon(s) in tea None ☐

☐ Teaspoon(s) in coffee

Which kind of spread do you use on bread, crispbread etc?

Butter ☐ Margarine ☐

Low Fat Spread ☐ None ☐

Which brand do you normally use?
.....

What do you do with visible fat on your meat?

Eat most of it ☐ Eat as little as possible ☐

Eat some of it ☐ Don't eat meat ☐

How often do you eat food that is fried?

Daily ☐ 4-6 times a week ☐

1-3 times a week ☐ Less than once a week ☐

Filling in your Food Diary

We would like to know what you eat and drink for **THREE DAYS.**
One of these days should be either a Saturday or Sunday , so ideally, your diary should cover *Thursday, Friday and Saturday or Sunday* or *Sunday, Monday, and Tuesday*. This is because people tend to eat differently at weekends.

Try to keep this diary with you and fill it in after each meal or snack while the information is fresh in your mind.

Please do not change what you normally eat just because you are filling in this diary—**be honest.**

Please write down everything you eat and everything you drink, including alcohol, snacks and nibbles between meals.

Please read these instructions carefully before you start

Write down as accurately as you can, the type of food or drink

- What type of cereal was it?
- Was it butter, margarine or low fat spread?
- What type of cheese was it?
- What was the name of the biscuits?
- Did you cut the fat off your meat?
- Was it a level, rounded or heaped tablespoon?
- Was it a large thick sausage or a small chipolata?
- Was it a half pint glass or a small glass?
- Was it a medium sliced large loaf or a small slice from a round granary/

How was the food prepared: casseroled, grilled, boiled or fried?

You do no need to weigh everything as long as you give a really good description

- For many foods such as vegetable and cereals, state the number of tablespoons and whether level, rounded or heaped.

- For bread, fruit loaves etc. indicate the size of the loaf and the thickness of the slice.

- For meat, fish and cheese, describe as well as you can, e.g. 2 *large* slices of ham or 2 *small* lamb chops (no fat eaten) or small fillet of cod grilled with 1 teaspoon of Flora or square of Cheshire cheese the size of a matchbox.

- State whether the drinks are in a glass / cup / mug.

- Remember to look at wrappers and labels and write down the brand name and weights eg. 125g St Ivel Real Low Fat Yoghurt or 225g Heinz Baked Beans

Include everything you eat and drink, including nibbles

Try to be as clear as you can

Look at the example page. You might not eat any of the foods written down but it will show you how to fill in your diary

Example

Day: Monday
Date : 23.08.2004

Before Breakfast	
Portion size / quantity	Details of food and drink
1 cup 1 tablespoon	Tea with Semi-skimmed milk
Breakfast	
Portion size / quantity	Details of food and drink
3 heaped tablespoons 1/4 pint 1 medium slice 1 teaspoon (level) 2 mugs	Branflakes (Kellogs) Semi-skimmed milk for cereals and drinks Wholemeal bread (large loaf) Flora extra light margarine Coffee
During the morning	
Portion size / quantity	Details of food and drink
250 ml carton 1 large 1 teaspoon (level) 1 small slice 1 teaspoon (rounded)	Low sugar Ribena Light Beefburger bun with Flora extra light margarine Corned beef Branston pickle
Midday Meal	
Portion size / quantity	Details of food and drink
2 large serving spoons 2 large serving spoons 1 large 1 large 1 can	Canteen Meal Lamb curry Pilau rice Tomato (sliced) Banana Diet Tango

During the afternoon	
Portion size / quantity	Details of food and drink
1 glass (1 tablespoon squash) 1/2 a 26g packet	Tescos's Low calorie orange squash make with concentrate KP plain crisps
Evening Meal	
Portion size / quantity	Details of food and drink
3 heaped tablespoons 1 large Cereal bowl full 1 150g pot 1 275 ml can	Sainsbury's baked beans Jacket potato Chopped celery, carrot and tomato Shape raspberry yoghurt Skol lager
During the evening and night	
Portion size / quantity	Details of food and drink
1 mug 1 tablespoon 2	Tea make with Semi-skimmed milk Rich tea biscuits (McVities)

Day

Date

Before Breakfast	
Portion size / quantity	Details of food and drink
Breakfast	
Portion size / quantity	Details of food and drink
During the morning	
Portion size / quantity	Details of food and drink
Midday Meal	
Portion size / quantity	Details of food and drink

During the afternoon	
Portion size / quantity	Details of food and drink
Evening Meal	
Portion size / quantity	Details of food and drink
During the evening and night	
Portion size / quantity	Details of food and drink

Appendix 9

WHO-5 Well-Being questionaire

Psychiatric Research Unit
WHO Collaborating Centre in Mental Health

WHO (Five) Well-Being Index (1998 version)

Please indicate for each of the five statements which is closest to how you have been feeling over the last two weeks. Notice that higher numbers mean better well-being.

Example: If you have felt cheerful and in good spirits more than half of the time during the past two weeks, put a tick in the box with the number 3 in the upper right corner.

	Over the last two weeks	All of the time	Most of the time	More than half of the time	Less than half of the time	Some of the time	At no time
1	**I have felt cheerful and in good spirits**	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1	☐ 0
2	**I have felt calm and relaxed**	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1	☐ 0
3	**I have felt active and vigorous**	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1	☐ 0
4	**I woke up feeling fresh and rested**	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1	☐ 0
5	**My daily life has been filled with things that interest me**	☐ 5	☐ 4	☐ 3	☐ 2	☐ 1	☐ 0

Scoring:

The raw score is calculated by totalling the figures of the five answers. The raw score ranges form 0 to 25, 0 representing worst possible and 25 representing best possible quality of life.

To obtain a percentage score ranging from 0 to 100, the raw score is multiplied by 4. A percentage score of 0 represents the worst possible, whereas a score of 100 represents the best possible quality of life.

Appendix 10

EQ-5D questionnaire

PAGE NUMBERING AS ORIGINAL

Health Questionnaire

English version for the UK
(validated for Ireland)

By placing a tick in one box in each group below, please indicate which statements best describe your own health state today:

Mobility

I have no problems in walking about ❑

I have some problems in walking about ❑

I am confined to bed ❑

Self-care

I have no problems with self-care ❑

I have some problems washing or dressing myself ❑

I am unable to wash or dress myself ❑

Usual activities (e.g. work, study, housework, family or leisure activities)

I have no problems with performing my usual activities ❑

I have some problems with performing my usual activities ❑

I am unable to perform my usual activities ❑

Pain/Discomfort

I have no pain or discomfort ❑

I have moderate pain or discomfort ❑

I have extreme pain or discomfort ❑

Anxiety/Depression

I am not anxious or depressed ❑

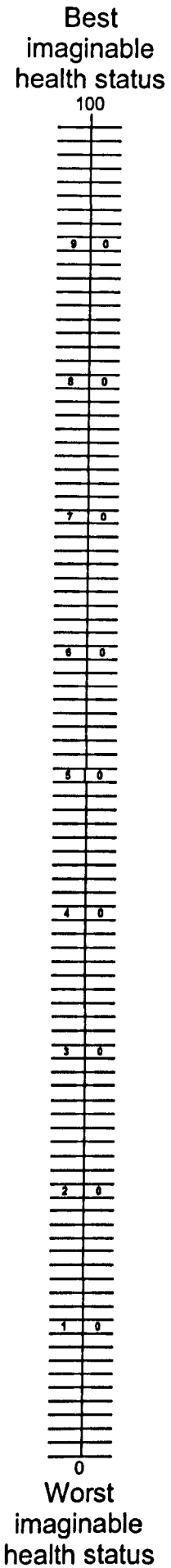
I am moderately anxious or depressed ❑

I am extremely anxious or depressed ❑

To help people say how good or bad a health state is, we have drawn a scale (rather like a thermometer) on which the best state you can imagine is marked 100 and the worst state you can imagine is marked 0.

We would like you to indicate on this scale how good or bad your own health is today, in your opinion. Please do this by drawing a line form the box below to whichever point on the scale indicates how good or bad your health state is today.

Your own health state today



Appendix 11

PAID and hypoglycaemia questionnaire

Which of the following diabetes issues are currently a problem for you?

Circle the number that gives the best answer for you.
Please provide an answer for each question.

	Not a Problem	Minor Problem	Moderate Problem	Somewhat Serious Problem	Serious Problem
Not having clear and concrete goals for your diabetes care?	0	1	2	3	4
Feeling discouraged with your diabetes treatment plan?	0	1	2	3	4
Feeling scared when you think about living with diabetes?	0	1	2	3	4
Uncomfortable social situations related to your diabetes care (eg. People telling you what to eat)?	0	1	2	3	4
Feelings of deprivation regarding food and meals?	0	1	2	3	4
Feeling depressed when you think about living with diabetes?	0	1	2	3	4
Not knowing if your mood or feelings are related to your diabetes?	0	1	2	3	4
Feeling overwhelmed by your diabetes?	0	1	2	3	4
Worrying about low blood sugar reactions?	0	1	2	3	4
Feeling angry when you think about living with diabetes?	0	1	2	3	4
Feeling constantly concerned about food and eating?	0	1	2	3	4
Worrying about the future and the possibility of serious complications?	0	1	2	3	4
Feelings of guilt and anxiety when you get off track with your diabetes management?	0	1	2	3	4
Not "accepting" your diabetes?	0	1	2	3	4
Feeling unsatisfied with your diabetes physician?	0	1	2	3	4
Feeling that diabetes is taking up too much of your mental and physical energy every day?	0	1	2	3	4
Feeling alone with your diabetes?	0	1	2	3	4
Feeling that your friends and family are not supportive of your diabetes management efforts?	0	1	2	3	4
Coping with complications of diabetes?	0	1	2	3	4
Feeling 'burned out' by the constant effort needed to manage your diabetes?	0	1	2	3	4

Hypoglycaemia Questionnaire

For each question, please tick the relevant box

1. Which best describes you?

   I always have symptoms when my blood sugar is low ☐

   I sometimes have symptoms when my blood sugar is low ☐

   I no longer have symptoms when my blood sugar is low ☐

2. Have you lost some of the symptoms that used to occur when your blood sugar was low?

   Yes ☐ No ☐

3. In the past six months, how often have you had a moderate hypoglycaemic episode? (where you have felt confused, disorientated or lethargic and you were unable to treat yourself)

   Never ☐ Once or twice ☐ More ☐

4. In the past year, have you had a severe hypoglycaemic episode? (where you were unconscious and needed paramedic assistance)

   Never ☐ Once or twice ☐ More ☐

5. How often in the last month have you had readings less than 4.0mmol/l **with symptoms**?

   Never ☐ 1-3 times ☐ Once a week ☐ 2-3 times a week ☐ Daily ☐

6. How often during the last month have you had a reading less than 4.0mmol/l **without symptoms?**

   Never ☐ 1-3 times ☐ Once a week ☐ 2-3 times a week ☐ Daily ☐

7. How low does your blood sugar need to go before you feel symptoms?

   3.5-4.0mmol/l ☐ 3.0-3.5mmol/l ☐ less than 3.0mmol/l ☐

8. To what extent can you tell by your symptoms that your blood sugar is low?

   Rarely ☐ Sometimes ☐ Often ☐ Always ☐

Appendix 12

ADKnowl questionnaire

The Oxford Centre
for Diabetes, Endocrinology and Metabolism

Churchill Hospital
Headington
Oxford OX3 7LJ
Tel: 01865 857333
Fax: 01865 857368

PATIENT NUMBER __/__/__

PATIENT INITIALS __/__/__

DATE OF CONTACT __ __/__ __/__ __

OxREC no: C03.097
Version 1.1

October 2003

OXFORD HEALTHY LIVING

DIABETES KNOWLEDGE QUESTIONNAIRE

Below are some statements about diabetes.
For each statement, please tick the box to indicate whether you believe it to be true or false.
There may be any number of true statements in each set.

1

Please consider each of the following statements about diabetes	**True**	**False**	**Don't Know**
a. Diabetes can be controlled with treatment			
b. A little glucose in the urine is a good thing			
c. Diabetes is likely to go away after a while			
d. Stressful experiences can affect blood glucose levels			
e. Blood glucose levels do not affect your chances of developing complications			

2

The usual effect of physical exercise is to...	True	False	Don't Know
a. Lower blood glucose levels			
b. Raise blood glucose level			
c. Increase blood glucose levels in urine			
d. Leave blood glucose levels unchanged			

3

Please consider each of the following statements about the effects of food on blood glucose levels	True	False	Don't Know
a. Sugary foods affect blood glucose levels			
b. Starch foods (e.g. potato, bread etc.) affect blood glucose levels			
c. Protein foods (e.g. meat, cheese etc.) affect blood glucose levels			
d. Alcohol- free wines and lagers will have no effect on blood glucose levels			
e. Full-fat foods will affect blood glucose levels more than low-fat foods			
f. Sugary foods require more insulin than starch foods, even if they contain the same amount of carbohydrate			
g. Any amount of fresh fruit can be eaten with little effect on blood glucose levels			

4

Please consider the following statements about food	True	False	Don't Know
a. People with diabetes need to avoid foods containing any sugar			
b. It is not possible to eat too much protein			
c. Fried foods are usually low in fat			
d. Pastry and cakes are high in fat			
e. Cheese and biscuits are usually less fattening than puddings			
f. All margarines and spreads have fewer calories than butter			
g. Restricting the use of salt can help to reduce high blood pressure			
h. High fat foods can increase the risk of complications			
I. Special diabetic products can be eaten freely without leading to weight gain			

5

Alcoholic drinks (particularly beer, ciders and liquers) generally ...	True	False	Don't Know
a. Lower blood glucose levels after a few hours			
b. Raise blood glucose levels initially			
c. Have no calories			

6

Keeping diabetes well controlled over the years can lower your risk of damage to ...	**True**	**False**	**Don't Know**
a. Nerves in your feet			
b. Your kidneys			
c. Your eyes			

7.

Regular examinations are recommended to check	**True**	**False**	**Don't Know**
a. For nerve damage to your feet			
b. Your blood pressure			
c. Your eyes			
d. Your cholesterol level			
e. Only things you have been having a problem with			

Appendix 13

Curriculum for InSight

Week 1

Process:

- The InSight facilitator will make introductions and attempt to put all participants at their ease. The facilitator will encourage participants to set ground rules for the group and these will be written on a flip-chart and displayed during the course. The facilitator will describe the Insight programme and will provide a copy of the timetable to all participants.

- Participants will be asked specific questions about their personal aims and objectives for the course, write these down on thought bubbles and discuss their expectations. They will share experiences of diabetes, their thought and beliefs about self-care management and the role of other people using pre-prepared statements. Information and answers will be written down on a flip-chart.

- The physiology of the fed and fasting state in individuals with and without diabetes and the role of carbohydrates will be explored using the Bodylink tool. The facilitator will encourage every individual to participate and contribute and all comments will be actively listened to.

- The facilitator will engage the participants in discussion about self-monitoring and will introduce the diary log and discuss the importance of sharing information and recording usual practice.

- All participants will be actively engaged throughout the session by being encouraged to reflect, share their experiences and to ask questions. Any questions not addressed immediately will be recorded on a flip-chart for the future. All questions will be answered in an honest, respectful, open and non-judgemental way.

Week 1: Session 1
Introduction, aims and objectives, setting ground rules and clarifying the programme
Time allocation: 45 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
	Introduction and welcome	Providing welcoming atmosphere and refreshments Providing course materials		OHP/computer Refreshments Pens Timetable of course Workbook
Emotion management, elicitation of personal experience	To learn peoples' names and shared experiences of diabetes To establish ground rules for course	Asking for names and length of diagnosis Discussing ground rules and recording on flip-chart	Giving name and recalling length of diagnosis Identifying and agreeing ground rules	Flip-chart and pens
Emotion management, identification and elicitation of expectations	To identify personal aims and objectives for the programme	Providing thought bubbles and ask participants to identify main aims Reviewing each comment and discuss realistic expectations	Writing main aims on thought bubbles Describing and discussing the aims of the group	Thought bubbles Felt pens Blue-tack
Delivering information	To learn about the content and duration of the programme	Providing slide of programme content	Clarifying programme	3 slides of programme content (slides 1-3)
Elucidating philosophy	To understand the principles of non-judgemental, shared experience	Promoting discussion about the roles of people with diabetes and their relationships with other people	Discussing and sharing experience of the 'Diabetes Police'	Policeman's hat

Week 1: Session 2
Diabetes myths
Time allocation: 45 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Emotion management, elicitation of beliefs and knowledge	To recognise that personal beliefs influence self-management	Providing myth cards and encouraging each participant to read a statement Promoting discussion about personal beliefs Facilitating responses from all individuals	Reading statements and sharing their beliefs and knowledge Listening to other individuals point of view	Top hat and 6 myth cards

Week 1: Session 3
Learning to think like a pancreas
Time allocation: 45 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of knowledge, role modelling, delivering information by utilising different learning styles	To understand: • Normal physiology in the fed and fasting state • Diabetes physiology in the fed and fasting state • Physiology of insulin action To determine each individual's basal and prandial insulin	Utilising Bodylink tool to encourage participants to demonstrate normal physiology and diabetes physiology Encouraging participants to demonstrate active involvement Demonstrating insulin action using slide Recording each individual's insulin regimen on flip chart and evaluating proportion of basal insulin	Physically and verbally engaging in using Bodylink to explore physiology in diabetic and non-diabetic state Reflecting on insulin action and physiology Providing details of personal insulin use Discussing and formulating any changes to basal insulin	Slide 4 Bodylink tool and accessories Hand-out of bodylink diagram Slides of insulin action (slides 5-7) Template of record of insulin for flip-chart Flip-chart and pens Workbook pages 6 – 9 and appendix 1 (p 46)

Week 1: Session 4
Self-monitoring – introducing the diary log
Time allocation: 35 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of knowledge, delivering information by utilising different learning styles	To identify and record carbohydrate containing foods and differing effects on blood glucose	Utilising Bodylink tool and food models to explore the different effects of different foods Discussing identification of individual's carbohydrate foods Explaining usefulness of recording weights of carbohydrate foods	Identifying carbohydrate containing foods Recognising usefulness of weighing and recording personal portion size of foods eaten	Bodylink tool and accessories Food models: apple, slice of bread, chocolate bar Slides 8-10
Experiential learning, discussing concrete experience and encouraging reflection	To appreciate the principles of accurate information gathering and self-monitoring	Introducing diary log and explaining completion using an example Discussing use of scales for weighing portions of carbohydrate foods Reviewing principles of information gathering including food, exercise stress in relation to blood glucose monitoring Discussing importance of sharing information and maintaining usual lifestyle	Understanding principles of log as a tool for self-management Agreeing to diary completion and monitoring for the coming week Practising using scales with food models	Testing slide (slide 11) Demonstration scales Scales and batteries Diary logs – 28 paper and 2 acetate logs Acetate pens Slides of diary log and example (slides 12-13) Form for weights of commonly eaten carbohydrates

Week 1: Session 5
Any other questions and evaluation
Time allocation: 10 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
		Asking for any questions or clarification of issues Recording any future topics on the flip-chart for discussion in week 4	Asking questions	Flip chart and pens
		Providing and collecting PAID and hypo questionnaires (unless completed before course)	Completing forms	PAID and hypo questionnaires
		Weighing patients and organising blood sample for A1c and lipid levels (unless completed before course)	Agreeing to biomedical assessment	Scales Process for blood collection
		Providing evaluation forms for the session (at least once annually)	Providing completed evaluation form	Evaluation form

Week 2

Process:

- The InSight facilitator will welcome back all participants. The facilitator will encourage individual participants to share and discuss their 2 day diary logs and will display them to the group using the overhead projector. The facilitator will promote group support, ask relevant questions and enable all individuals to offer advice and support based upon their own experiences

- The facilitator will promote discussion about various means of calculating carbohydrates and will use food models, scales, food tables, nutritional labels and DAFNE plate models to encourage every individual to participate and contribute in the session

- The facilitator will explain the formulae used to calculate both insulin:carbohydrate ratios and correction doses and encourage the participants to calculate their own ratios and practice this approach by referring to diary logs

- Facilitators and participants will eat lunch together and practice carbohydrate counting and applying the new ratios. More practice will take place by completion of a carbohydrate quiz

- Injections technique will be explored and addressed

- All participants will be actively engaged throughout the session by being encouraged to reflect, share their experiences and to ask questions. Any questions not addressed immediately will be recorded on a flip-chart for the future. All questions will be answered in an honest, respectful, open and non-judgmental way

Week 2: Session 1: Introduction to carbohydrate counting
Time allocation: 60 minutes

Learning outcomes	Facilitator activity	Participant activity	Resources	Key questions/statements
To accurately assess carbohydrate-containing foods	Asking participants to divide food models into 2 groups – those containing carbohydrates and those not Summarising that fat and protein have little or no effect on blood glucose levels	Physically and verbally engaging with food models to identify carbohydrate containing foods	Food models	Do all foods raise blood glucose levels after eating? Can you identify those which have little or no effect on glucose levels? Only food containing carbohydrate have an effect on blood glucose levels after eating
To identify different methods of assessing carbohydrate content of foods	Asking participants to rank food models by carbohydrate content Discussing methods of accurately assessing carbohydrate content of foods and listing on flip-chart	Physically and verbally engaging with food models to rank carbohydrate containing foods Identifying different methods of assessing carbohydrate	Food models	Do all foods contain the same amount of carbohydrate? Different foods contain different amounts of carbohydrate. How could you estimate the carbohydrate content of the food you eat?
To establish weighing foods and using food tables to calculate carbohydrate content	Discussing and demonstrating using weights of food and food tables to assess carbohydrate content	Using personal information to calculate carbohydrate content of foods eaten	Scales Food tables (books) Calculators	How do you calculate the carbohydrate content of food from the weight? Which foods would this method be most useful for?
To investigate us of nutritional labels to count carbohydrates	Providing and explaining nutritional labels	Practising carbohydrate counting using nutritional labels	Nutritional labels	How is the information presented? How useful is information per 100g? What are the issues with information per portion?

Week 2: Session 1: Introduction to carbohydrate counting *contd*

Learning outcomes	Facilitator activity	Participant activity	Resources	Key questions/statements
To identify and use visual calculation of carbohydrate	Providing DAFNE plates Asking for estimation of carbohydrate content	Observing and estimating carbohydrate content of meals	DAFNE plates	Which foods on each plate would you need to count? How much carbohydrate do you think each food contains? What is the total carbohydrate of the meal?

Week 2: Session 2: Calculation of individual insulin:carbohydrate ratios
Time allocation: 15 minutes

Learning outcomes	Facilitator activity	Participant activity	Resources	Key questions/statements
To calculate personal insulin:carbohydrate ratio	Discussing general guidelines of I unit of insulin for every 10-15g carbohydrate Writing formula on flip chart (total daily dose/50) Explaining that this figure gives the amount of units of insulin for each 10g carbohydrate Inviting participants to calculate individual ratio	Calculating total daily dose Calculating personal doses	Calculators	Most people need I unit of insulin for every 10-15g carbohydrate, but there is individual variation. We can use a formula to calculate this more accurately
To assess the safety of this new approach	Encouraging participants to compare new approach with usual dose at meals	Calculating and comparing new approach		We can practice this to see if it works by comparing what you usually do

Week 2: Session 3
Safety rules, correction doses and summarising individual ratios
Time allocation: 45 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Delivering information by utilisation of different learning styles, exploring personal beliefs, goal setting	To identify amount of insulin needed to correct out of target blood glucose levels	Discussing causes of high or low blood glucose levels Encouraging participants to identify personal targets for blood glucose levels and recording on flip-chart Discussing formula for calculating correction doses and recording on flip-chart Inviting participant to calculate personal correction dose	Identifying causes of high or low glucose Identifying personal targets Calculating personal ratio	Slide of formula (slide 15) Flip-chart Pens Workbook page 18
	To identify and adopt individual ratios for the coming week	Summarising and reviewing each participants ratios Dispensing personal calculator cards if required	Documenting personal ratios in workbook	Flip-chart with completed ratios Correction dose and carb counting personal calculator card Plan for week (slide 16)
	To identify situations when insulin is not given with carbohydrates	Promoting discussion about situations when eating carbohydrate should not be accompanied by insulin	Sharing experiences and discussing	Safety rules (slide 17)

Week 2: Session 4
Carbohydrate quiz
Time allocation: 45 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Experiential learning, utilisation of different learning styles	To calculate carbohydrate of foods using different strategies	Explaining and distributing carbohydrate quiz forms Discuss correct answers Encourage further practice at home	Engaging and calculating carbohydrate content of different foods in different scenarios Agreeing to further practice at home	Carbohydrate quiz forms Food models DAFNE plates Take-away handouts

Week 2: Session 5
Injection technique
Time allocation: 20 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, delivering information	To identify and use correct needle size, technique and site for insulin injections	Discussing factors affecting insulin absorption Eliciting discussion on current practice	Engaging and discussing personal injection procedure	Needles Injection technique slides (slides 18-21) Workbook Appendix 3 (p 51)

Week 2: Session 6
Any other questions and evaluation
Time allocation: 20 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
		Asking for any questions or clarification of issues Recording any future questions on the flip-chart Providing acetate diary logs	Asking questions	Flip chart and pens 2 acetate diary logs
		Providing evaluation forms for the session (if applicable)	Providing completed evaluation form	Evaluation form

Week 3

Process: The InSight facilitator will welcome back all participants. The facilitator will encourage individual participants to share and discuss their 2 day diary logs and will display them to the group using the overhead projector. The facilitator will promote group support, ask relevant questions and enable all individuals to offer advice and support based upon their own experiences. The facilitator will promote discussion about hypoglycaemia by encouraging participants to share and reflect on their own experiences. A practical session will take place to categorise warning signs and symptoms. The Bodylink tool will be utilised to demonstrate physiology of hypoglycaemia and suitable treatments will be discussed. The tool will then be used to explore the effects of exercise and alcohol. More carbohydrate counting practice will take place if time allows by introducing a take-away game. All participants will be actively engaged throughout the session by being encouraged to reflect, share their experiences and to ask questions. Any questions not addressed immediately will be recorded on a flip-chart for the future. All questions will be answered in an honest, respectful, open and non-judgmental way.

Week 3: Session 1
Welcome and reflection of individual diary logs
Time allocation: 60 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
	Welcome	Providing welcoming atmosphere and refreshments		Refreshments
Reflection from concrete experience, utilisation of different learning styles, emotion management, vicarious learning	To learn about personal and other group member's blood glucose pattern To relate patterns to insulin, food, stress and activity To appreciate relevance of accurate written records to establish baseline information for future management	Displaying individual overheads of diary logs Asking relevant questions to elicit further details Promoting group support and sharing of information Enabling all group participants to offer non-judgemental advice and support based on personal experience	Providing 2 completed diary logs on acetate overheads Sharing and explaining their 2 day record Offering advice and support to other group members	OHP/computer Completed acetate diary logs

Week 3: Session 2
Hypoglycaemia
Time allocation: 45 - 60 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, elicitation of knowledge and beliefs,		Asking relevant questions to elicit individual experiences	Sharing experiences and discussing	
	To identify symptoms of hypoglycaemia	Distributing hypo card game Reviewing symptoms using slides	Engaging in categorising symptoms into sub-headings	Hypo cards Hypo symptom slides (slides 22-24) Workbook pages 21 – 24)

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Delivering information by utilisation of different learning styles, vicarious learning	To understand physiology in relation to low blood glucose	Encouraging discussion and engagement using Bodylink tool	Physically and verbally engaged with discussion and tool	Bodylink (used for hypos, exercise and alcohol)
	To identify blood glucose levels which equate to hypo	Eliciting viewpoints Clarifying safety levels	Volunteering personal views	Slide (25)
	To understand treatment in relation to low blood glucose	Asking relevant questions to elicit individual treatment Advising most effective treatment Discussing effects of specific amounts of carbohydrate	Sharing and discussing suitable treatment	Samples of hypo treatment 2 handouts - treatment and overview Rule of 15 and effect of 10g CHO slides (26 - 27)

Week 3: Session 3
Exercise
Time allocation: 20 - 30 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, elicitation of knowledge and beliefs, delivering information by utilisation of different learning styles, vicarious learning		Asking relevant questions to elicit individual experiences	Sharing experiences and discussing	
	To understand the physiology of exercise	Utilising Bodylink tool to explore and review effects of exercise	Physically and verbally engaging with tool to explore effects of exercise	Bodylink Workbook pages 36 – 37 and Appendix 4 (p 52)
	To formulate strategies for personal exercise	Discussing and explaining individual response to planned or unplanned exercise	Exploring and discussing individual responses Formulating strategies	Slides (28 – 32)

Week 3: Session 4
Alcohol
Time allocation: 10 - 20 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, elicitation of knowledge and beliefs, delivering information by utilisation of different learning styles, vicarious learning		Asking relevant questions to elicit individual experiences	Sharing experiences and discussing	
	To understand the physiological effects of alcohol	Utilising Bodylink tool to explore and review effects of alcohol	Physically and verbally engaging with tool to explore effects of alcohol	Bodylink Workbook pages 33 - 35 Bottle of alcohol
	To calculate carbohydrate content of alcoholic drinks	Encouraging use of workbook to calculate carbohydrate content of drinks	Reviewing section in workbook	
	To identify safety factors	Asking relevant questions Explaining government safe levels of alcohol Discussing insulin adjustment	Sharing experiences and discussing	Slide 33

Week 3: Session 5
Any other questions and evaluation
Time allocation: 10 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
		Asking for any questions or clarification of issues Recording any future questions on the flip-chart Distributing acetate diary logs for week 4	Asking questions	Flip chart and pens 2 acetate diary logs

Week 3: Supplementary sessions
Practising carbohydrate counting
Time allocation: 10 – 20 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Experiential learning, utilisation of different learning styles	To calculate carbohydrate content of take-away meals	Distributing take-away game Facilitating calculations and stimulating discussion	Practising calculating carbohydrate content of different meals	Take-away game DAFNE plates Menu cards
	To investigate the effect of carbohydrate on blood glucose	Advising the effect of 10g carbohydrate on blood glucose levels Encouraging experimentation during the week with known amount of 10g carbohydrate	Exploring the effect of a known amount of carbohydrate	Food containing 10g carbohydrate eg 1 Jaffa cake

Week 4

Process: The InSight facilitator will welcome back all participants. The facilitator will encourage individual participants to share and discuss their 2 day diary logs and will display them to the group using the overhead projector. The facilitator will promote group support, ask relevant questions and enable all individuals to offer advice and support based upon their own experiences.
The facilitator will promote discussion about hyperglycaemia by encouraging participants to share and reflect on their own experiences. A practical session will take place to categorise warning signs and symptoms. The Bodylink tool will be utilised to demonstrate physiology of hyperglycaemia and appropriate management will be discussed. A general review of participants aims amd objective from the first session will take place. All group agenda items not previously explored will be addressed. All participants will be actively engaged throughout the session by being encouraged to reflect, share their experiences and to ask questions. Any questions not addressed immediately will be recorded on a flip-chart for the future. All questions will be answered in an honest, respectful, open and non-judgmental way.

Week 4: Session 1
Welcome and reflection of individual diary logs
Time allocation: 60 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
	Welcome	Providing welcoming atmosphere and refreshments		Refreshments Handout of description of diabetes physiology using bodylink
Reflection from concrete experience, utilisation of different learning styles, emotion management, vicarious learning	To learn about personal and other group member's blood glucose pattern To relate patterns to insulin, food, stress and activity To appreciate relevance of accurate written records to establish baseline information for future management	Displaying individual overheads of diary logs Asking relevant questions to elicit further details Promoting group support and sharing of information Enabling all group participants to offer non-judgemental advice and support based on personal experience	Providing 2 completed diary logs on acetate overheads Sharing and explaining their 2 day record Offering advice and support to other group members	OHP/computer Completed acetate diary logs

Week 4: Session 2
Hyperglycaemia
Time allocation: 30 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, elicitation of knowledge and beliefs, delivering information by utilisation of different learning styles, vicarious learning	To identify symptoms of hyperglycaemia	Reviewing symptoms using slides	Engaging in discussion and sharing experience	Slides of symptoms (34 – 36)
	To understand physiology in relation to high blood glucose	Encouraging discussion and engagement using Bodylink tool	Physically and verbally engaged with discussion and tool	Bodylink Workbook pages 25 - 29
	To identify blood glucose levels and ketone levels which equate to hyperglycaemia	Eliciting viewpoints Clarifying safety levels	Volunteering personal views Identifying glucose and ketone levels	Slide 37 - 39 Ketone meter – demonstration meter and meters for participants Urine ketone sticks Handout of ketone guidelines
	To understand carbohydrate content of foods and drinks to use when ill	Asking relevant questions to elicit individual use Advising use of alternative foods	Sharing and discussing suitable foods	Handout of common CHO foods (given week 2)

Week 4: Session 3
Review of programme
Time allocation: 60 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
Elicitation of personal experience, elicitation of knowledge and beliefs, delivering information by utilisation of different learning styles, vicarious learning, goal setting	To gain information about agenda items	Providing information about individual topics not covered in core curriculum and identified by participants Identifying and providing other options to provide further knowledge/support	Gaining necessary information to support individual needs	Flip-chart from previous weeks
		Facilitating discussion of thought bubbles and aims and objectives identified in week 1	Engaging in discussion	Completed thought bubbles
	To identify further support	Eliciting information about future peer and professional support	Identifying future needs	

Week 4: Session 4
Evaluation
Time allocation: 20 minutes

Specific aspects of theory	Learning outcomes	Facilitator activity	Participant activity	Resources
		Providing evaluation forms for the session	Providing completed evaluation form	Evaluation form

Low carbohydrate diet study - Demographics of all subjects at baseline

Subject no	Gender (M/F)	Age (years)	Diabetic status	Randomisation
01	F	47	diabetes	healthy eating
02	F	60	control	low carbohydrate
03	M	51	diabetes	low carbohydrate
04	M	58	diabetes	low carbohydrate
05	F	57	diabetes	low carbohydrate
06	F	35	diabetes	healthy eating
07	M	51	diabetes	healthy eating
08	M	64	diabetes	healthy eating
09	F	41	control	healthy eating
10	F	54	control	low carbohydrate
11	F	60	diabetes	low carbohydrate
12	M	44	control	healthy eating
13	F	40	control	healthy eating
14	F	60	diabetes	low carbohydrate
15	F	70	diabetes	healthy eating
17	F	42	control	healthy eating
18	F	60	control	low carbohydrate
19	F	52	control	low carbohydrate
20	M	58	diabetes	healthy eating
21	F	41	control	healthy eating
22	F	46	diabetes	low carbohydrate
23	M	48	diabetes	healthy eating
24	F	53	control	low carbohydrate
25	F	48	control	healthy eating
26	F	51	control	low carbohydrate
27	F	72	control	healthy eating

Low carbohydrate diet study - Biomedical results for all subjects at baseline

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)	Ketones (mmol/l)
01	79.8	29.7	0.93	43.0	116	80	7.6	5.0	1.10	3.1	1.7	0.0
02	81.0	30.1	0.79	44.1	112	73	6.1	6.8	1.66	4.5	1.3	0.0
03	104.8	32.7	1.03	32.4	148	93	8.1	2.9	0.69	1.3	1.9	0.1
04	89.2	29.5	0.96	34.0	116	72	5.8	6.0	1.37	3.6	2.2	0.1
05	93.5	33.1	0.84	46.8	142	66	6.0	7.3	2.36	4.5	0.9	0.0
06	101.3	37.7	0.84	48.8	115	83	8.4	4.8	1.30	2.9	1.3	0.0
07	112.9	36.0	1.02	38.5	142	84	8.6	4.1	0.98	2.5	1.3	0.1
08	85.4	29.6	1.02	31.7	184	92	7.4	5.0	2.30	2.4	0.6	0.1
09	67.6	25.4	0.88	37.7	111	72	5.5	4.7	1.56	2.4	1.7	0.1
10	82.4	30.6	0.82	47.2	118	65	5.9	5.0	1.04	3.5	1.1	0.1
11	96.7	40.8	0.88	56.3	138	72	6.4	4.5	1.07	2.0	3.1	0.0
12	93.2	30.8	0.86	31.9	139	80	6.0	5.1	1.08	3.2	1.7	0.0
13	136.5	56.8	UTM	57.7	126	98	6.0	5.4	1.24	3.7	1.0	0.0
14	87.4	36.4	0.89	52.3	166	64	6.9	4.4	0.96	3.0	0.9	0.0
15	93.9	34.1	0.85	53.1	138	87	6.1	4.1	1.62	2.3	0.5	0.1
17	104.6	38.4	0.78	51.1	145	84	5.8	6.2	1.08	4.2	2.0	0.0
18	81.3	33.0	0.88	49.0	120	83	5.7	6.6	1.21	4.0	3.0	0.0
19	126.7	48.9	0.85	58.8	174	108	6.1	4.7	1.31	2.8	1.2	0.0
20	107.2	34.2	1.02	35.5	152	94	6.7	6.1	1.18	3.8	2.4	0.0
21	81.2	30.2	0.88	45.1	116	73	5.7	4.8	1.34	3.1	0.7	0.0
22	126.8	46.6	0.93	58.9	161	87	10.2	3.4	0.96	1.6	1.8	0.1
23	108.1	32.6	0.92	36.0	133	92	6.7	4.9	1.53	2.9	1.1	0.0
24	97.7	31.9	0.89	45.0	130	90	6.3	5.2	1.11	3.5	1.4	0.0
25	100.6	38.8	0.90	51.2	154	71	5.7	3.9	1.43	1.8	1.4	0.0
26	77.5	27.8	0.98	43.1	138	78	6.3	4.6	1.56	2.4	1.4	0.2
27	85.6	36.1	0.90	53.6	157	87	6.4	6.9	1.39	5.0	1.2	0.1

UTM = unable to measure

Low carbohydrate diet study - Biomedical results for all subjects at 3 months.

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)	Ketones (mmol/l)
01	79.4	29.5	0.90	44.6	120	79	7.6	4.5	0.98	2.9	1.3	0.0
02	71.7	26.6	0.79	43.2	124	84	5.8	6.3	1.47	4.4	0.9	0.0
03	91.0	28.4	0.96	29.5	135	78	6.4	3.3	0.84	1.5	2.1	0.3
04	85.8	27.4	0.94	32.4	127	78	5.8	5.7	1.38	3.7	1.4	0.1
05	86.8	30.7	0.82	45.9	128	80	6.1	7.0	2.43	4.3	0.6	0.1
06	101.2	37.7	0.83	NR	106	59	8.2	4.8	1.30	2.9	1.3	NR
07	111.6	35.6	0.98	38.8	128	88	7.9	4.7	1.06	2.8	1.8	0.1
08	82.6	28.6	0.96	32.1	176	96	7.2	4.6	1.74	2.4	1.1	0.1
09	65.5	24.7	0.80	38.2	124	80	5.0	4.8	1.46	2.4	2.1	0.1
10	74.3	27.6	0.81	45.1	122	66	5.4	5.7	1.26	4.0	0.9	1.0
11	94.2	39.7	0.91	56.9	140	80	6.5	4.5	1.26	2.8	0.9	0.0
12	88.0	29.1	0.85	30.1	125	76	6.0	4.7	1.22	2.9	1.2	0.1
13	133.1	55.4	UTM	60.1	115	81	5.9	5.1	1.09	3.7	0.7	0.0
14	78.6	32.3	0.92	48.7	165	72	6.3	4.6	1.09	3.0	1.1	0.1
15	91.1	33.0	0.97	53.2	132	81	5.8	4.4	1.78	2.4	0.5	0.0
17	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
18	77.5	31.4	0.95	50.6	128	76	5.9	7.7	1.38	4.7	3.6	0.0
19	118.8	45.8	0.83	57.8	168	103	5.9	4.8	1.38	3.1	0.8	0.0
20	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
21	81.2	30.2	0.78	NR	116	73	5.7	4.8	1.34	3.1	0.7	0.0
22	113.7	41.8	0.88	52.8	141	81	9.7	3.7	0.9	2.3	1.1	0.7
23	110.4	33.3	0.93	35.7	137	96	7.0	4.7	1.16	3.1	1.0	0.0
24	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
26	73.1	26.2	0.97	42.5	144	89	6.2	4.3	1.82	2.0	1.1	0.0
27	80.2	33.8	0.95	53.0	152	85	6.2	6.9	1.73	4.6	1.3	0.2

UTM = unable to measure, NR = not recorded, LFU = lost to follow-up

Low carbohydrate diet study - Dietary intake for all subjects at baseline

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	% Energy from:			
					Protein	Fat	Carbohydrate	Alcohol
01	1903	107.8	88.2	181.6	22.6	41.6	35.7	0.0
02	1792	93.4	69.0	175.1	21.7	36.1	38.2	3.9
03	2637	104.4	126.0	228.6	15.8	42.9	32.5	8.8
04	2762	99.3	135.1	250.8	14.4	44.0	34.1	7.5
05	1295	95.5	38.8	152.2	29.3	26.8	43.8	0.0
06	2909	108.7	145.0	309.6	15.0	45.0	40.0	0.0
07	1958	101.7	88.9	171.0	20.8	40.9	32.7	5.6
08	1537	85.1	33.3	167.9	22.1	19.5	40.9	17.6
09	1858	54.5	87.6	226.3	11.8	42.5	45.7	0.0
10	1858	74.8	76.8	226.2	16.1	37.3	45.7	0.8
11	2738	117.4	136.9	274.2	17.2	45.1	37.7	0.0
12	2664	139.4	114.6	269.6	20.8	38.6	37.8	2.8
13	1951	100.6	92.5	186.9	20.8	43.0	36.2	0.0
14	2782	98.2	90.3	420.7	14.1	29.2	56.7	0.0
15	1900	72.1	56.5	238.4	15.1	26.7	47.0	11.2
17	2034	114.4	84.4	218.3	22.5	37.3	40.2	0.0
18	2413	98.4	121.4	248.0	16.3	45.2	38.5	0.0
19	1966	101.1	78.4	229.1	20.5	35.8	43.6	0.0
20	2015	109.4	108.1	114.9	21.5	47.8	21.1	9.6
21	1396	71.1	60.2	145.4	20.3	38.8	39.0	1.9
22	2701	110.4	147.0	227.3	16.9	50.6	32.6	0.0
23	2599	105.8	89.8	293.7	16.2	30.9	42.1	10.7
24	1997	72.8	95.1	226.4	14.6	42.9	42.5	0.0
25	1976	77.5	77.4	251.6	15.8	35.4	48.0	0.8
26	1946	81.7	86.4	168.8	16.8	39.9	32.5	10.8
27	1798	79.6	76.9	199.5	18.1	39.4	42.5	0.0

Low carbohydrate diet study - Dietary intake for all subjects at 3 months

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	% Energy from:			
					Protein	Fat	Carbohydrate	Alcohol
01	1701	82.8	65.0	209.3	19.5	34.4	46.1	0.0
02	967	75.4	36.9	81.6	31.2	34.4	31.7	0.0
03	1318	100.6	61.5	21.6	30.6	42.1	6.2	21.2
04	1284	86.0	72.0	53.7	36.9	50.7	15.8	6.6
05	1146	100.4	43.6	92.4	34.8	34.0	30.0	1.2
06	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC
07	1926	109.1	102.0	95.7	22.7	47.8	18.7	10.8
08	1334	72.3	35.3	137.3	21.4	23.5	38.1	17.0
09	1609	65.4	46.3	249.0	16.2	25.9	57.9	0.0
10	1646	98.4	120.3	33.2	24.0	65.9	7.6	2.6
11	1498	107.4	103.2	36.7	28.7	62.1	9.2	0.0
12	1970	77.4	86.0	200.4	16.1	40.3	39.2	4.4
13	1239	78.5	53.8	95.3	25.3	39.1	28.8	6.8
14	1334	141.1	63.5	52.9	42.3	42.8	14.9	0.0
15	1354	64.5	39.3	152.4	19.1	26.2	42.3	12.4
17	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
18	1104	98.3	55.0	58.9	35.5	44.6	19.9	0.0
19	1184	104.8	46.0	93.8	35.4	34.9	29.7	0.0
20	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
21	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC
22	1422	69.5	75.8	23.2	19.6	48.1	6.2	26.1
23	1872	105.3	86.3	109.9	22.4	41.3	22.0	14.3
24	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
26	1545	87.7	84.2	76.7	22.6	48.9	18.6	9.8
27	1286	58.3	48.5	164.1	18.1	34.0	47.9	0.0

FDNC = food diary not completed, LFU - lost to follow-up

Low carbohydrate diet study - quality of life and hunger score for all subjects at baseline

Subject no	WHO-5 (%)	Hunger score
01	44	4
02	76	3
03	88	5
04	92	4
05	92	4
06	24	4
07	60	3
08	88	4
09	72	5
10	52	4
11	56	3
12	68	5
13	36	4
14	44	5
15	44	5
17	24	4
18	24	3
19	60	3
20	80	5
21	32	3
22	48	3
23	48	4
24	48	5
25	52	5
26	48	3
27	20	8

Low carbohydrate diet study - quality of life and hunger score for all subjects at 3 months

Subject no	WHO-5 (%)	Hunger score
01	32	3
02	100	3
03	80	3
04	60	4
05	80	5
06	24	4
07	68	4
08	100	3
09	68	3
10	64	3
11	36	4
12	88	5
13	56	3
14	76	3
15	68	4
17	LFU	LFU
18	72	6
19	80	6
20	LFU	LFU
21	32	3
22	72	6
23	44	6
24	LFU	LFU
25	LFU	LFU
26	32	4
27	48	5

LFU = lost to follow-up

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 1

Subject no	Ketone readings (mmol/l)												
01	0.0	0.0	0.0	0.0	0.0	0.0	NR	NR	NR	NR	NR	NR	NR
02	0.0	0.1	0.2	0.0	0.3	0.3	0.3	0.3	NR	NR	NR	NR	NR
03	0.1	0.0	0.0	0.1	0.1	0.1	0.1	0.3	0.4	0.3	0.6	0.3	0.7
04	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1	0.0	0.1	0.1	0.1
05	0.0	0.1	0.2	0.0	0.0	0.2	0.0	0.0	0.0	0.1	0.1	0.0	0.0
06	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
07	0.0	0.0	0.1	0.2	0.2	0.1	0.3	0.2	0.0	0.0	0.0	0.0	0.0
08	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0
09	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.0	0.2	0.0	0.1
10	0.0	0.0	0.0	0.0	0.0	0.1	0.5	0.4	0.3	0.8	0.7	0.2	0.2
11	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
12	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
13	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0
15	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
18	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.4
19	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.1
20	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
22	0.3	0.2	0.4	0.5	0.2	0.8	0.4	0.4	0.4	0.5	0.1	0.2	0.8
23	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
24	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.1	0.0	NR	NR	NR	NR
25	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
26	0.1	0.0	0.1	0.1	0.0	0.1	NR	NR	NR	NR	NR	NR	NR
27	0.1	0.0	0.1	0.1	0.0	0.1	0.1	0.0	NR	NR	NR	NR	NR

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 1 *(contd)*

Subject no	Ketone readings (mmol/l)												
01	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
02	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
03	0.4	0.3	0.2	0.6	0.6	0.6	0.5	1.0	0.5	1.2	0.5	0.9	0.4
04	0.1	0.1	0.1	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0	0.0	0.1
05	0.3	0.2	0.1	0.1	0.1	0.1	0.0	0.0	0.1	0.2	0.0	0.0	0.4
06	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1	0.0
07	0.0	0.0	0.0	0.0	0.2	0.0	0.1	0.1	0.2	0.0	0.0	NR	NR
08	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.1	0.1	0.1	0.1
09	0.0	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.0	NR	NR
10	0.4	0.1	0.0	0.3	0.6	0.6	0.7	0.6	0.9	1.2	0.2	0.3	0.2
11	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
12	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.1
13	0.0	0.1	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
14	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.2	0.0	0.0	0.2	0.1	0.0	0.1	0.0	0.1	0.0
19	0.0	0.1	0.1	0.0	0.1	0.1	0.1	0.0	0.1	0.1	0.1	0.1	0.0
20	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
22	0.6	0.4	0.9	0.3	0.2	0.4	0.5	0.4	0.4	0.2	1.2	0.7	0.7
23	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
24	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
25	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
26	0.2	0.0	0.1	0.0	0.1	0.1	0.0	0.1	NR	NR	NR	NR	NR
27	0.1	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1	0.0	0.1	0.1	0.0

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 2

Subject no	Ketone readings (mmol/l)			
01	NR	NR	NR	NR
02	0.0	0.0	0.1	0.1
03	0.8	0.4	0.2	0.9
04	0.1	0.1	0.0	0.1
05	0.1	0.3	0.2	0.0
06	0.0	0.0	0.0	0.0
07	0.0	0.0	0.0	0.1
08	0.0	0.0	0.0	0.1
09	0.0	0.0	0.1	0.0
10	0.2	0.1	0.6	0.2
11	0.0	0.0	0.0	0.0
12	0.0	0.3	0.0	0.0
13	0.0	0.0	0.0	0.0
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.1	0.1
17	LFU	LFU	LFU	LFU
18	0.0	0.2	0.1	0.0
19	0.0	0.1	0.1	0.1
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	1.7	0.8	0.8	0.7
23	NR	NR	NR	NR
24	NR	NR	NR	NR
25	LFU	LFU	LFU	LFU
26	0.1	0.2	NR	NR
27	NR	NR	NR	NR

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 3

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.1	0.0	0.0	0.2
03	0.8	0.8	0.8	0.4
04	0.0	0.0	0.0	0.0
05	0.0	0.1	0.3	0.1
06	0.1	0.0	0.0	0.0
07	0.0	0.0	0.0	0.0
08	0.0	0.0	0.0	0.0
09	0.0	0.0	0.0	NR
10	0.4	0.2	0.4	0.3
11	0.1	0.1	0.0	0.0
12	0.0	0.1	0.0	0.0
13	0.0	0.0	0.1	0.0
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.1	0.0	0.1	0.1
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	NR	NR	NR	NR
23	NR	NR	NR	NR
24	NR	NR	NR	NR
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.1	
27	NR	NR	NR	NR

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 4

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.1	0.2	0.3	0.3
03	1.1	0.6	0.6	0.4
04	0.0	0.0	0.0	0.1
05	0.0	0.0	0.1	0.0
06	0.0	0.0	0.0	0.0
07	0.0	0.0	0.0	0.0
08	0.0	0.2	0.3	0.1
09	0.0	0.2	0.1	NR
10	0.1	0.2	0.7	0.7
11	0.0	0.0	0.0	NR
12	0.0	0.1	0.0	0.0
13	0.0	0.1	0.1	0.0
14	0.0	0.0	0.1	0.1
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.1	0.1	0.0	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	NR	NR	NR	NR
23	NR	NR	NR	NR
24	NR	NR	NR	NR
25	LFU	LFU	LFU	LFU
26	0.0	0.0	NR	NR
27	0.0	0.0	0.0	0.0

LFU = lost to follow-up, NR = no recordings, DNA = did not attend this visit

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 5

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.1	0.2
03	0.8	0.5	0.5	0.2
04	0.0	0.0	0.0	0.1
05	0.0	0.0	0.0	0.0
06	NR	NR	NR	NR
07	0.0	0.0	0.2	0.2
08	0.2	0.0	0.0	0.4
09	NR	NR	NR	NR
10	0.3	0.2	0.7	0.1
11	0.0	0.0	0.1	0.1
12	0.0	0.0	0.0	0.0
13	0.0	0.0	0.1	0.0
14	0.0	0.0	0.0	NR
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.0	0.1	0.1	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	NR	NR	NR	NR
23	DNA	DNA	DNA	DNA
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.0	NR
27	0.0	0.0	0.0	0.0

LFU = lost to follow-up, NR = no recordings, DNA = did not attend this visit

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 6

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.2	0.0	0.1
03	0.5	0.4	0.4	0.3
04	0.0	0.0	0.0	0.1
05	0.0	0.0	0.0	0.0
06	NR	NR	NR	NR
07	0.1	0.1	0.1	0.1
08	0.1	0.1	0.1	0.1
09	0.0	0.0	0.0	NR
10	0.2	0.1	0.4	0.2
11	0.0	0.0	0.0	0.1
12	0.0	0.0	0.0	0.0
13	0.0	0.0	0.1	0.0
14	0.0	0.0	0.0	0.1
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.0	0.0	0.0	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	NR	NR	NR	NR
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.2	0.0	NR
27	0.0	0.0	0.0	0.0

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 7

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.0	0.0
03	0.2	0.2	0.3	0.2
04	0.0	0.1	0.1	0.0
05	0.0	0.0	0.1	0.0
06	NR	NR	NR	NR
07	0.1	0.1	0.1	0.0
08	0.0	0.0	0.1	0.0
09	0.0	0.2	0.0	NR
10	0.4	0.5	0.5	0.2
11	0.1	0.0	0.0	0.0
12	0.0	0.1	0.0	0.0
13	0.0	0.0	0.1	0.0
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	NR	NR	NR	NR
19	0.0	0.0	0.1	0.1
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	NR	NR	NR	NR
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	NR	NR	NR
27	0.0	0.1	0.0	0.0

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 8

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.2	0.0
03	0.3	1.0	0.4	NR
04	0.0	0.0	0.0	0.1
05	0.0	0.0	0.0	0.1
06	NR	NR	NR	NR
07	0.0	0.1	0.1	0.1
08	0.0	0.3	0.0	0.0
09	0.0	0.0	0.0	NR
10	0.5	0.4	0.7	0.5
11	0.0	0.0	0.0	0.1
12	0.0	0.0	0.0	0.0
13	0.1	0.1	0.0	0.1
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.5
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.0	0.1	0.1	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	1.0	0.8	0.7	0.8
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.1	0.3	0.0
27	0.0	0.2	0.0	0.2

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 9

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.1	0.0
03	0.3	1.0	0.4	NR
04	0.0	NR	NR	NR
05	0.0	0.2	0.1	0.0
06	NR	NR	NR	NR
07	0.0	0.0	0.0	0.0
08	0.1	0.0	0.1	0.0
09	0.0	0.0	0.0	0.0
10	0.3	0.3	0.4	0.6
11	0.0	0.0	0.0	0.0
12	0.0	0.0	0.0	0.0
13	0.0	0.0	0.0	0.0
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.2	0.1
19	0.1	0.0	0.0	0.1
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	1.2	0.7	0.4	0.7
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.0	NR
27	0.0	0.0	0.1	0.1

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 10

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.0	0.0
03	0.4	0.3	0.3	0.2
04	0.0	0.0	0.0	0.0
05	0.0	0.0	0.0	0.1
06	NR	NR	NR	NR
07	0.0	0.0	0.0	0.0
08	0.0	0.0	0.1	0.1
09	0.0	0.1	0.1	NR
10	0.4	0.8	0.5	0.1
11	0.0	0.0	0.0	0.1
12	0.0	0.0	0.1	0.0
13	0.0	0.0	0.0	0.0
14	0.0	0.0	0.2	0.1
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.0
19	0.0	0.0	NR	NR
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	0.9	0.7	0.5	0.5
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.0	NR
27	0.1	0.1	0.1	NR

LFU = lost to follow-up, NR = no recordings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 11

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.0	0.0	0.0	0.0
03	0.2	0.0	0.4	0.3
04	0.0	0.0	0.0	0.1
05	0.0	0.1	0.1	0.1
06	NR	NR	NR	NR
07	0.0	0.0	0.1	0.2
08	0.0	0.1	0.1	0.1
09	0.0	0.0	0.0	NR
10	0.0	0.2	0.2	0.1
11	0.0	0.0	0.0	0.0
12	0.0	0.2	0.0	0.0
13	0.0	0.0	0.1	0.0
14	0.0	0.0	0.2	0.1
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	0.1
19	0.0	0.0	0.1	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	1.2	0.7	0.8	0.5
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.2	NR
27	0.1	0.1	0.0	0.1

LFU = lost to follow-up, NR = no recorrdings

Low carbohydrate study - Self-monitored ketone levels for all subjects during Week 12

Subject no	Ketone readings (mmol/l)			
01	0.0	0.0	0.0	0.0
02	0.1	0.0	0.2	0.0
03	0.1	0.1	0.1	0.1
04	0.0	0.0	0.0	0.1
05	0.0	0.1	0.1	0.0
06	NR	NR	NR	NR
07	0.0	0.0	0.0	0.0
08	0.0	0.1	0.0	0.2
09	0.0	0.0	0.0	0.0
10	0.1	0.4	0.4	0.7
11	0.0	0.0	0.0	0.0
12	0.0	0.0	0.0	0.0
13	0.0	0.0	0.0	0.0
14	0.0	0.0	0.0	0.0
15	0.0	0.0	0.0	0.0
17	LFU	LFU	LFU	LFU
18	0.0	0.0	0.0	NR
19	0.0	0.0	0.0	0.0
20	LFU	LFU	LFU	LFU
21	NR	NR	NR	NR
22	0.8	0.7	0.4	0.5
23	NR	NR	NR	NR
24	LFU	LFU	LFU	LFU
25	LFU	LFU	LFU	LFU
26	0.0	0.0	0.0	0.1
27	NR	NR	NR	NR

LFU = lost to follow-up, NR = no recordings

Video-based education study - Demographics of all subjects at baseline

Subject no	Gender (M/F)	Age (years)	Duration of DM (months)	Randomisation
001	F	67.9	2	video
002	F	57.7	1	delay
003	F	55.2	1	delay
004	M	54.2	4	video
005	M	60.6	2	video
006	F	75.4	1	video
007	M	49.9	4	delay
008	M	64.5	4	delay
009	F	64.2	3	delay
010	M	70.9	6	video
011	M	48.1	2	video
012	F	63.0	7	delay
013	M	72.1	2	delay
015	F	56.5	7	video
016	F	43.9	6	video
017	F	47.6	7	video
018	F	71.7	2	delay
019	F	71.6	2	delay
020	F	59.2	2	video
021	F	66.6	2	video
022	F	64.7	1	delay
023	F	49.2	4	video
024	F	60.5	1	video
025	M	65.8	9	delay
026	M	75.5	1	delay
027	M	45.1	3	video
028	M	68.2	1	delay
029	M	65.8	1	video
030	M	73.9	1	delay
031	F	67.6	4	delay
032	F	67.3	5	video
034	M	45.7	2	delay
035	M	63.3	1	video
037	F	59.8	10	video
038	M	70.5	6	delay
039	M	67.3	3	video
040	F	63.9	2	delay
041	F	62.5	6	delay
042	F	53.2	8	video
043	M	53.5	4	delay
045	F	47.8	4	video
046	F	39.8	6	delay

Video-based education study - Biomedical results for all subjects at baseline

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)
001	116.5	39.4	0.89	56.7	136	84	9.0	4.9	1.05	2.71	2.5
002	82.3	29.5	0.95	47.3	133	78	8.3	7.3	1.13	4.35	4.0
003	88.5	33.3	0.88	50.3	134	79	6.6	6.0	1.47	3.62	2.0
004	96.4	32.2	0.97	34.2	126	92	8.2	5.5	0.75	4.02	1.6
005	87.1	27.4	0.99	28.7	180	84	8.8	6.0	1.39	2.29	5.1
006	74.6	27.1	0.96	49.1	133	76	7.3	4.8	1.64	2.57	1.3
007	113.0	30.3	0.95	31.4	167	99	6.3	5.7	1.25	3.95	1.1
008	90.7	30.3	1.02	34.0	124	72	6.3	3.4	0.71	1.83	1.9
009	93.2	35.0	0.89	51.6	123	82	6.5	3.8	0.94	2.00	1.9
010	79.1	26.7	0.95	28.2	161	80	6.6	2.4	0.66	1.33	0.9
011	88.4	31.7	0.94	35.6	136	94	8.0	6.1	1.54	3.29	2.8
012	106.4	37.7	1.00	56.1	132	84	7.4	no result	no result	no result	no result
013	84.7	28.2	1.00	32.2	179	81	7.3	4.1	1.01	2.73	0.8
015	99.0	35.1	1.00	49.9	158	68	7.2	5.3	1.09	3.21	2.2
016	90.4	31.7	0.91	46.9	108	79	7.1	5.2	1.01	3.51	1.5
017	92.8	31.7	1.02	46.6	125	70	8.4	5.1	1.18	3.33	1.3
018	108.0	40.6	0.91	58.4	160	84	7.0	3.8	0.85	2.13	1.8
019	83.0	31.6	0.95	51.0	156	78	6.7	4.3	0.96	2.39	2.1
020	73.0	28.2	0.85	44.9	147	90	6.7	6.3	1.22	4.63	1.0
021	82.6	27.9	0.89	45.6	143	64	6.1	4.9	1.1	3.00	1.8
022	75.6	26.5	0.83	42.2	142	76	14.0	6.0	2.4	3.26	0.8
023	85.5	34.7	0.83	49.6	147	98	7.1	6.1	1.3	3.92	1.9
024	111.5	42.5	0.95	no result	130	89	8.4	4.3	1.6	2.20	1.1
025	71.0	24.9	0.97	32.3	144	60	5.8	2.7	1	1.37	0.7
026	110	29.8	1.03	36.3	174	95	7.2	4.2	1.5	2.08	1.4
027	78.3	24.7	0.89	26.3	123	76	6.1	2.5	0.8	0.62	2.4
028	83.5	30.3	0.98	37.8	162	86	6.4	5.4	0.9	3.05	3.2
029	92.1	30.8	0.94	31.6	140	74	11.6	3.6	0.8	1.65	2.5
030	86.7	27.1	0.95	31.0	158	87	6.1	4.7	1.1	no result	no result

Video-based education study - Biomedical results for all subjects at baseline (contd)

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)
031	66.5	24.4	0.83	40.3	108	68	6.3	5.5	1.3	3.20	2.2
032	58.1	22.1	0.87	42.9	134	68	6.5	3.3	1	1.69	1.3
034	110.1	36.8	1.04	39.1	140	100	11.7	6.7	1.1	4.31	2.8
035	127.0	39.2	1.02	39.6	182	117	6.4	5.8	1.3	4.10	0.8
037	108.2	36.0	0.91	50.6	130	80	5.8	5.1	1.1	3.50	1.1
038	85.0	31.2	1.02	30.7	130	68	6.7	4.3	0.8	2.60	1.9
039	96.1	29.7	0.91	30.0	116	67	7.3	3.1	0.8	1.60	1.5
040	59.4	23.2	0.91	39.1	176	60	5.9	3.8	1.8	1.60	0.9
041	100.5	38.3	0.98	51.0	164	77	6.4	5.5	1.3	3.70	1.0
042	73.9	32.0	0.95	47.8	169	83	8.4	4.1	1.0	1.30	3.9
043	75.5	23.6	0.95	23.8	116	62	6.9	3.9	1.0	2.50	0.9
045	94.4	39.8	0.96	45.7	103	62	7.6	3.9	1.3	2.30	0.6
046	80.2	30.2	0.95	43.5	159	91	6.3	4.3	0.9	2.70	1.5

Video-based education study - Biomedical results for all subjects at 6 months follow-up

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)
001	106.7	36.1	0.90	53.4	130	88	7.6	3.7	0.97	3.46	1.7
002	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
003	84.3	31.7	0.82	48.6	137	73	6.5	6.8	1.7	4.2	2.0
004	99.6	33.3	0.97	35.3	145	84	6.6	4.2	0.9	2.6	1.6
005	87.4	29.9	1.00	31.1	158	80	9.7	5.6	1.8	3	1.8
006	74.9	27.1	0.92	48.1	146	84	7.2	5.0	1.6	2.8	1.4
007	116.6	31.6	0.98	34.8	171	100	6.8	4.6	1.3	2.9	0.8
008	92.9	31.0	0.98	34	137	82	6.6	3.7	0.6	1.5	3.6
009	92.7	34.9	0.90	52.5	136	83	6.1	4.1	no result		2.1
010	81.2	27.4	0.95	34.2	173	88	6.6	2.1	0.7	0.8	1.3
011	88.2	31.6	0.92	31.3	156	94	6.6	5.5	1.2	2.34	4.3
012	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
013	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
015	99.2	35.1	1.00	51.1	168	86	7.5	5.1	1.1	2.5	3.2
016	92.5	32.4	0.88	43.1	125	73	6.6	5.6	no result	no result	1.6
017	91.3	31.2	1.00	40.6	102	63	5.1	4.3	1.5	2.5	0.7
018	107.8	40.6	0.92	56.8	147	74	7.1	3.5	0.9	1.8	1.7
019	81.4	31.0	0.98	50.5	183	100	5.8	4.5	1.5	2.2	1.8
020	62.4	24.1	0.93	39.9	121	81	6.2	4.2	1.5	2.3	0.9
021	78.8	26.6	0.89	44	127	75	6.1	4.4	1.2	2.5	1.5
022	71.6	25.1	0.81	36	101	57	7.6	3.9	2.4	1.2	0.6
023	84	34.1	0.82	47.3	157	102	6.4	5.9	1.3	3.6	2.2
024	105.5	40.2	0.88	no result	125	74	6.7	4.3	1.6	2.0	1.6
025	70.4	24.6	0.98	32.5	136	64	5.8	2.7	0.9	1.4	0.9
026	109.4	29.7	1.01	36.2	163	100	7.5	4.8	1.5	1.95	2.0
027	77.4	24.4	0.88	19.9	111	76	6	2.5	0.9	1.0	1.4
028	85.1	30.9	0.98	38.2	166	94	6.7	7.9	1.0	5.5	3.0
029	87.7	29.3	0.96	28.8	140	80	6.3	3.1	1.1	1.2	1.7
030	90.8	28.3	0.97	32.4	204	104	6.4	4.2	1.0	2.6	1.3

LFU lost to follow up

Video-based education study - Biomedical results for all subjects at 6 months follow-up (contd)

Subject no	Weight (kg)	BMI (kg/m2)	Waist/hip ratio	%body fat	Systolic BP (mmHg)	Diastolic BP (mmHg)	A1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	LDL cholesterol (mmol/l)	Triglycerides (mmol/l)
031	65.4	24.0	0.82	40.8	135	80	6	4.9	1.2	2.9	1.8
032	57.3	21.8	0.91	40.1	133	58	6.6	3.2	0.9	1.5	1.8
034	120.1	40.1	1.06	38.1	132	79	7.3	4	0.9	2	2.36
035	133.2	41.0	1.08	43.6	136	77	6.5	3.3	1.4	1.6	0.66
037	no result	no result	no result	no result	no result	no result	5.7	4.4	no result	no result	no result
038	81	29.8	no result	no result	no result	no result	6.3	3.7	no result	no result	1.74
039	95.2	29.3	0.91	31.3	124	73	7.7	2.8	0.9	1.2	1.42
040	57.3	22.4	0.89	41.4	173	72	5.9	4.6	2.1	2.2	0.75
041	104.7	39.7	0.95	55.6	159	85	7	4.9	1.3	3.2	0.82
042	78.2	33.8	0.94	54.1	155	77	7.5	4.1	1.1	no result	5.14
043	76.7	23.9	0.90	27.2	126	71	5.9	4.2	1.1	2.7	0.95
045	87.8	37.0	0.94	47.7	111	60	7.7	5.2	1.2	3.2	1.68
046	76.2	28.6	0.90	39.6	142	91	6.2	4	1.2	2.1	1.54

LFU lost to follow up

Video-based education study - Absolute dietary intake for all subjects at baseline

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Total sugars (g)	Saturated fat (g)	Monounsaturated fat (g)	Polyunsaturated fat (g)	Dietary fibre (g)
001	1563	65.8	62.5	197.1	79.2	22.6	23.3	11.1	14.5
002	1401	60.8	60.0	165.5	55.2	24.4	17.6	10.1	9.6
003	1307	83.3	48.7	142.9	69.3	16.4	15.4	10.7	11.3
004	1871	95.7	68.4	234.5	89.1	21.7	23.4	14.5	17.0
005	1442	85.6	51.4	98.1	29.4	18.5	13.2	17.2	12.9
006	1701	71.3	76.2	195.1	99.6	33.8	23.1	11.7	16.2
007	2046	101.9	76.1	154.7	69.4	17.7	22.5	17.9	11.7
008	2046	70.7	75.0	294.0	94.6	18.0	25.0	19.0	18.5
009	1646	69.3	67.4	202.0	73.1	21.3	25.6	16.4	11.8
010	2300	89.0	76.5	304.2	87.6	17.9	25.7	25.5	21.9
011	2135	101.6	66.5	235.6	30.8	14.4	21.9	18.9	12.9
012	1266	64.9	43.7	162.7	48.8	13.9	14.1	12.2	14.0
013	1333	76.7	44.6	163.6	62.2	17.0	17.1	6.4	14.3
015	1647	70.0	50.6	258.3	95.0	13.4	14.7	12.0	16.2
016	2051	78.6	79.3	251.3	101.4	32.1	24.8	14.9	16.9
017	3142	99.6	128.7	353.8	103.6	51.9	31.0	11.9	17.8
018	1484	52.1	58.3	182.9	69.4	25.4	19.7	7.9	10.6
019	1553	54.2	58.3	213.0	119.2	16.1	21.4	15.6	24.4
020	1189	67.3	32.3	154.5	60.2	11.4	12.3	6.0	13.0
021	1688	79.6	55.7	225.3	78.5	16.0	12.7	13.5	18.7
022	2169	87.1	98.5	242.9	114	32.9	36.3	21.3	21.1
023	1795	73.4	70.4	181.4	75.0	24.6	23.8	15.7	15.2
024	1406	66.1	75.7	122.6	32.9	32.2	26.7	11.5	8.0
025	1819	83.3	54.7	261.2	113.5	15.8	17.1	14.5	28.2
026	1698	98.1	82.6	125.6	39.1	22.5	36.9	15.5	11.1
027	1546	81.1	44.0	210.3	90.5	7.2	14.7	11.6	19.9
028	2257	128.2	77.1	284.7	65.1	29.4	20.8	17.9	26.8
029	2312	108.1	99.9	235.7	36.9	27.4	29.2	27.5	14.9
030	1886	61.8	68.1	271.2	141.3	23.8	20.2	17.9	18.9

Video-based education study - Absolute dietary intake for all subjects at baseline (contd)

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Total sugars (g)	Saturated fat (g)	Monounsaturated fat (g)	Polyunsaturated fat (g)	Dietary fibre (g)
031	1589	58.9	67.5	197.4	76.5	32.4	19.2	7.1	13.5
032	2031	80.2	95.4	227.3	47.4	29.9	33.8	19.9	19.1
034	2414	99	110.9	240.1	72.1	38.2	36.1	28.5	17.4
035	1794	82.3	89.3	151.0	36.9	38.0	32.3	12.2	10.5
037	1280	66.8	36.8	181.6	101.6	9.7	11.2	3.5	9.7
038	2326	107	96.1	273	138.8	41.5	34.7	12.8	14.1
039	1917	77.0	84.5	226.9	58.5	24.1	23.6	28.1	15.1
040	1826	59.7	65.2	263.9	96.8	23.2	19.1	14.3	19.0
041	2466	131.4	70.4	374.6	183.1	20.3	21.2	16.0	17.0
042	1829	84.0	69.3	206.0	99.4	17.1	23.1	21.1	20.0
043	2995	122.4	167.2	256.3	86.1	49.8	59.5	42.3	25.0
045	1691	77.9	54.4	239	133.2	20.7	19.4	9.6	15.0
046	2287	70.9	103.8	234.2	88.0	38.0	37.4	22.7	18.0

Video-based education study - Absolute dietary intake for all subjects at six months

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Total sugars (g)	Saturated fat (g)	Monounsatured fat (g)	Polyunsaturated fat (g)	Dietary fibre (g)
001	1492	74.1	50.1	200.2	83.1	12.3	20.2	12.7	8.2
002	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
003	1576	71.7	64.3	190.3	75.4	22.5	22	13	7.3
004	2306	95.3	89.3	298.5	116.1	34.3	30	16.9	17.1
005	1851	87	38	126.9	48.7	17.7	11.7	4.2	11.8
006	1907	87.3	80.2	214.6	109.7	34.9	22.7	7.4	19.1
007	2199	101.2	79.8	201.4	86.6	37.5	25.3	10.3	13.5
008	2209	80.8	80.7	315.5	102	15.1	20.1	19.3	25.3
009	1283	71.7	54.9	132.6	48	22.1	19.2	8.4	12
010	1814	79.3	59.9	232.7	67	21.7	21.1	8.1	23.8
011	2036	90.8	60.1	272.3	35.6	13.8	17.9	17.1	9.5
012	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
013	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU	LFU
015	1927	92.7	62	276.9	86.7	18	16.2	13	13
016	2016	75.1	120.1	174	91.1	52.2	37.9	17.7	11.9
017	1675	99.4	43.9	200.6	75.7	12.4	15.9	6.3	20.4
018	1232	63	47.5	142.2	55.9	19.2	14.9	8.7	4.4
019	1565	61.6	47.6	233.6	119.9	13	15.6	13.8	23.6
020	1793	101.9	66.3	196.9	99.8	19.7	20.1	21.4	20.3
021	1435	68.5	47.1	179.3	74.1	12.2	17.7	12.7	14.6
022	1838	80.6	70.8	233.4	73.2	24.4	23.8	17	23
023	2393	88.6	106.5	247	67.8	41.1	35	17	13.7
024	1387	76.5	51.2	165	42	18.5	16.5	11.3	11.3
025	1972	78.2	65.9	277.7	110.5	12.9	27.9	20.2	22
026	1447	75.7	75.5	95.8	23.8	28.5	28.7	11.3	6.4
027	1903	77.1	58.4	259.5	93.6	9.6	15.5	20.2	27
028	1901	69.1	82	237.1	93.9	25.3	28	23	16.4
029	1409	57.2	53.2	139.2	49.1	20.3	17.8	7.2	12.8
030	2453	91.2	109.4	277	107.5	36.1	26	20.1	12.2

LFU lost to follow up FDNC food diary not completed

Video-based healthy living study - Absolute dietary intake for all subjects at six months. LFU = lost to follow up

Subject no	Energy (kcal)	Protein (g)	Fat (g)	Carbohydrate (g)	Total sugars (g)	Saturated fat (g)	Monounsatured fat (g)	Polyunsaturated fat (g)	Dietary fibre (g)
031	1620	69.9	50	237.1	113.5	20.8	10.7	8.5	10.2
032	1430	53	64.4	168.1	65	25.6	18	14.1	8
034	2144	92.8	74.7	216.8	83.1	24.3	21.1	19.4	13.1
035	1607	95.3	63.1	136.4	31.4	21.8	22.8	7.9	12.2
037	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC
038	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC
039	2098	103.4	75.7	268.2	84.0	21.3	28.3	16.4	19.6
040	1830	49.1	74.3	231.9	100.4	23.4	18.4	13.6	14.1
041	1922	103.4	63.5	249.1	143.3	24.1	20.6	12.0	24.4
042	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC	FDNC
043	2644	128.2	95.8	327.4	114.7	31.8	36.5	20.0	31.1
045	1974	71.8	70.0	259.7	84.5	25.0	26.4	9.6	13.9
046	1980	66.4	61.8	276.9	103.6	22.8	19.4	12.5	14

LFU lost to follow up FDNC food diary not completed

Video-based education study - % energy from macronutrients for all subjects at baseline

Subject no	%Energy from Protein	%Energy from Fat	%Energy from Carbohydrate	%Energy from Alcohol
001	16.8	35.9	47.2	0.0
002	17.3	38.5	44.2	0.0
003	25.0	32.8	40.2	2.0
004	20.4	32.8	46.8	0.0
005	23.9	32.2	25.6	18.2
006	16.8	40.3	43.0	0.0
007	19.9	33.4	28.3	18.3
008	13.7	32.8	53.5	0.0
009	16.9	37.0	46.2	0.0
010	15.5	30.0	49.7	4.9
011	19.0	28.0	41.3	11.8
012	20.6	31.2	48.3	0.0
013	23.2	30.4	46.4	0.0
015	16.4	26.7	56.8	0.0
016	15.4	35.0	46.3	3.3
017	12.7	36.9	42.3	8.0
018	14.0	35.2	46.0	4.8
019	13.8	33.5	51.0	1.7
020	22.6	24.4	48.6	4.7
021	18.8	29.6	49.9	1.7
022	16.1	41.0	42.1	0.8
023	16.2	35.1	37.6	11.0
024	18.8	48.5	32.7	0.0
025	18.2	26.9	53.5	1.4
026	23.1	43.7	27.7	5.5
027	21.0	25.6	50.9	2.5
028	22.6	30.5	47.0	0.0
029	18.8	39.0	38.4	3.9
030	13.0	32.2	53.4	1.5
031	14.9	38.4	46.8	0.0
032	15.8	42.3	42.0	0.0
034	16.4	41.4	37.4	4.8
035	18.5	45.3	31.9	4.3
037	20.9	25.9	53.2	0.0
038	18.5	37.3	44.1	0.1
039	16.0	39.6	44.3	0.0
040	13.2	32.3	54.5	0.0
041	21.3	25.7	52.7	0.0
042	18.4	34.1	42.3	5.2
043	16.6	50.9	32.5	0.0
045	18.4	28.9	52.8	0.0
046	12.5	41.0	38.6	8.0

Video-based education study - % energy from macronutrients for all subjects at 6 months

Subject no	%Energy from Protein	%Energy from Fat	%Energy from Carbohydrate	%Energy from Alcohol
001	19.8	30.1	50.1	0.0
002	LFU	LFU	LFU	LFU
003	18.2	36.6	45.2	0.0
004	16.5	34.9	48.6	0.0
005	18.8	18.5	25.7	36.9
006	18.4	37.9	42.3	1.4
007	18.5	32.8	34.5	14.3
008	14.5	32.5	53.0	0.0
009	22.4	38.7	38.9	0.0
010	17.5	29.8	48.3	4.3
011	17.8	26.5	50	5.7
012	LFU	LFU	LFU	LFU
013	LFU	LFU	LFU	LFU
015	18.9	28.9	52.8	0.0
016	14.8	53.2	32.1	0.0
017	23.7	23.5	44.8	8.0
018	20.3	34.5	43.0	2.1
019	15.6	27.2	55.5	1.7
020	22.7	33.3	41.2	2.8
021	19.1	29.5	46.8	4.7
022	17.6	34.7	47.7	0.0
023	14.9	40.3	39.0	5.8
024	22.0	33.1	44.5	0
025	15.8	30.0	52.7	1.4
026	20.9	47.0	24.8	7.2
027	16.4	27.7	51.3	4.6
028	14.5	30.8	46.7	0.0
029	16.1	33.8	36.9	13.2
030	14.8	39.9	42.1	3.3
031	17.3	27.8	54.9	0.0
032	14.9	40.8	44.3	0.0
034	17.3	31.4	38.0	13.3
035	23.7	35.3	31.8	9.2
037	FDNC	FDNC	FDNC	FDNC
038	FDNC	FDNC	FDNC	FDNC
039	19.7	32.4	47.9	0.0
040	10.9	37.2	48.8	3.5
041	21.6	29.8	48.6	0.0
042	FDNC	FDNC	FDNC	FDNC
043	19.3	32.5	48.2	2.0
045	14.4	31.5	48.7	5.4
046	13.4	28.1	52.6	5.9

LFU = lost to follow-up FDNC = food diary not completed

Video-based education study – number of steps per day measured by pedometer

Baseline

Subject no	Steps per day
001	2819
002	9120
003	5871
004	3046
005	12785
006	6082
007	11944
008	3952
009	1865
010	6214
011	4452
012	2194
013	3922
015	3500
016	9418
017	7189
018	358
019	1546
020	8081
021	1208
022	9904
023	3321
024	7671
025	3614
026	4580
027	9524
028	DNC
029	3289
030	6239
031	6825
032	7788
034	11895
035	643
037	DNC
038	6383
039	3609
040	945
041	5454
042	9493
043	8014
045	11810
046	2292

6 months

Subject no	Steps per day
001	3530
002	LFU
003	DNC
004	4669
005	9364
006	3499
007	9276
008	8136
009	703
010	11948
011	6500
012	LFU
013	LFU
015	1857
016	6793
017	7020
018	586
019	3000
020	6947
021	7367
022	7867
023	3369
024	4576
025	423
026	1543
027	7694
028	2146
029	3728
030	5779
031	7987
032	4686
034	13441
035	2430
037	DNC
038	DNC
039	2091
040	2526
041	3015
042	DNC
043	2870
045	8756
046	8698

LFU lost to follow up DNC diary not completed

Video-based education study - Quality of life for all subjects at baseline

Subject no	WHO-5 (%)	EQ-5D VAS	EQ-5D Dimensions - reported problems				
			1 Mobility	2 Self-care	3 Usual activity	4 Pain/discomfort	5 Anxiety/depression
001	24	60	some	none	some	moderate	moderate
002	80	NR	none	none	none	none	none
003	48	70	NR	NR	NR	NR	none
004	80	80	some	none	none	some	none
005	48	80	none	none	none	none	none
006	80	90	none	none	none	none	none
007	72	85	none	none	none	none	moderate
008	68	79	none	none	none	moderate	none
009	56	NR	none	none	none	none	none
010	80	75	none	none	none	none	none
011	52	80	none	none	none	none	none
012	72	75	none	none	none	none	none
013	68	65	none	none	none	moderate	none
015	76	80	none	none	none	none	moderate
016	28	45	some	none	none	moderate	moderate
017	36	50	some	none	none	none	moderate
018	48	80	some	none	some	moderate	moderate
019	72	60	none	none	none	none	moderate
020	84	NR	none	none	none	none	none
021	72	85	some	none	none	moderate	none
022	96	95	none	none	none	none	none
023	80	90	none	none	none	none	none
024	68	71	none	none	none	none	none
025	60	60	some	none	some	moderate	moderate
026	80	90	none	none	none	none	none
027	76	80	none	none	none	not done	none
028	96	80	NR	NR	NR	NR	NR
029	60	67	some	none	none	moderate	moderate
030	60	60	some	none	some	moderate	moderate

NR = not recorded

Video-based education study - Quality of life for all subjects at baseline (contd)

Subject no	WHO-5 (%)	EQ-5D VAS	EQ-5D Dimensions - reported problems				
			1 Mobility	2 Self-care	3 Usual activity	4 Pain/discomfort	5 Anxiety/depression
031	24	50	some	some	some	extreme	extreme
032	56	85	none	none	none	none	none
034	72	85	none	none	none	none	none
035	84	69	none	none	none	none	none
037	76	95	none	none	none	moderate	none
038	88	85	some	none	some	moderate	none
039	80	85	some	none	some	moderate	none
040	72	88	none	none	none	none	none
041	80	90	none	none	none	none	NR
042	60	90	none	none	none	none	none
043	64	80	none	none	none	none	none
045	56	75	none	none	none	moderate	none
046	32	40	none	none	none	none	moderate

NR = not recorded

Video-based education study - Quality of life for all subjects at 6 months.

Subject no	WHO-5 (%)	EQ-5D VAS	EQ-5D Dimensions - reported problems				
			1 Mobility	2 Self-care	3 Usual activity	4 Pain/discomfort	5 Anxiety/depression
001	20	59	some	none	some	moderate	moderate
002	LFU	LFU	LFU	LFU	LFU	LFU	LFU
003	36	50	none	none	some	moderate	moderate
004	92	90	none	none	none	none	none
005	32	60	none	none	none	none	none
006	84	89	none	none	none	none	none
007	76	85	none	none	none	none	moderate
008	76	85	none	none	none	moderate	none
009	72	75	none	none	none	none	none
010	80	85	none	none	none	none	none
011	68	75	none	none	none	none	none
012	LFU	LFU	LFU	LFU	LFU	LFU	LFU
013	LFU	LFU	LFU	LFU	LFU	LFU	LFU
015	48	70	none	none	none	none	moderate
016	32	30	some	none	some	moderate	moderate
017	48	NR	none	none	none	none	moderate
018	40	50	some	none	some	moderate	moderate
019	76	NR	none	none	none	none	none
020	88	90	none	none	none	none	none
021	60	65	none	none	none	moderate	moderate
022	100	100	none	none	none	none	none
023	72	90	none	none	none	none	none
024	72	81	none	none	none	moderate	none
025	72	85	some	none	none	moderate	none
026	80	98	none	none	NR	none	none
027	76	80	none	none	none	moderate	none
028	80	80	none	none	none	moderate	none
029	80	70	some	none	none	moderate	none
030	80	65	some	none	some	moderate	none

LFU = lost to follow up, NR = not recorded

Video-based education study - Quality of life for all subjects at 6 months (contd)

Subject no	WHO-5 (%)	EQ-5D VAS	EQ-5D Dimensions - reported problems				
			1 Mobility	2 Self-care	3 Usual activity	4 Pain/discomfort	5 Anxiety/depression
031	80	50	some	some	some	extreme	extreme
032	76	85	none	none	none	moderate	none
034	68	85	none	none	none	none	none
035	72	80	none	none	none	none	none
037	NR	NR	NR	NR	NR	NR	NR
038	NR	NR	NR	NR	NR	NR	NR
039	56	90	some	none	some	some	none
040	64	90	none	none	none	none	none
041	84	80	none	none	none	none	none
042	60	91	some	none	none	moderate	none
043	64	70	none	none	none	none	none
045	84	90	none	none	none	moderate	none
046	84	85	none	none	none	none	none

LFU = lost to follow up, NR = not recorded

InSight study - Demographics of all subjects at baseline

Subject No	Age (years)	Duration of DM (years)	Gender (M/F)
001	48	17	F
002	47	33	F
003	44	41	M
004	44	26	M
005	58	45	F
006	41	1	F
007	33	26	F
008	37	6	F
009	26	16	F
010	36	10	M
011	40	1	F
012	63	33	F
013	37	31	F
014	27	18	F
015	59	46	F
016	31	3	M
017	31	19	M
018	42	21	F
019	43	31	M
020	59	43	F
021	39	37	F
022	55	29	F
023	63	29	F
024	39	6	M
025	38	10	M
026	60	16	F
027	32	10	M
028	72	37	M
029	51	42	M
030	46	12	F
031	43	37	M
032	23	18	F
033	54	10	M
034	46	29	M
035	31	10	F
036	46	19	F
037	42	7	F
038	36	13	M
039	56	52	M
040	33	8	F
041	29	14	M
042	42	31	F
043	35	24	F
044	33	2	F
045	46	12	F
046	51	24	F
047	46	5	F
048	51	unknown	M
049	31	3	F
050	28	22	F
051	25	17	F

InSight study - Biomedical results of all subjects at baseline

	Weight (kg)	BMI (kg/m^2)	HbA1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	Triglycerides (mmol/l)	LDL cholesterol (mmol/l)
001	103.1	31.5	10.6	4.5	0.9	1.3	3.01
002	83.1	27.8	7.1	4.0	2.2	0.7	1.48
003	92.0	31.5	8.8	4.0	1.07	2.0	2.02
004	87.3	26.1	7.9	4.8	1.4	1.4	2.76
005	85.1	30.2	8.3	5.2	2.1	1.1	2.60
006	64.4	22.5	6.7	4.5	1.84	0.6	2.39
007	83.5	25.5	6.9	4.4	2.2	0.6	1.93
008	73.6	31.0	5.9	5.6	0.9	5.0	2.43
009	76.5	29.5	7.1	4.4	1.6	1.1	2.30
010	79.9	23.6	7.0	4.5	1.1	1.0	2.95
011	61.4	22.3	8.4	4.5	2.2	0.7	1.98
012	65.6	25.3	8.7	4.6	3.1	0.5	1.27
013	60.7	22.6	7.9	4.2	2	0.3	2.06
014	63.5	25.8	7.4	5.0	2.4	0.8	2.24
015	67.3	25.0	8.8	3.5	1.1	0.5	2.17
016	75.8	23.1	7.4	5.5	2.2	0.4	3.12
017	77.9	23.0	6.1	no data	no data	no data	no data
018	74.4	24.0	8.6	4.3	no data	0.3	no data
019	85.6	30.0	7.6	2.8	no data	0.6	no data
020	64.7	21.9	9.6	no data	no data	no data	no data
021	74.5	26.4	7.4	4.9	1.9	0.8	2.64
022	85.9	32.7	7.0	5.4	1.8	1.2	3.05
023	64.2	23.6	7.7	4.0	1.9	0.7	1.78
024	86.8	29.0	10.6	6.7	1.0	3	4.34
025	61.2	21.7	8.8	3.7	1.2	1.1	2.00
026	52.9	22.9	10.3	4.7	2.6	0.6	1.83
027	83.1	23.0	10.6	3.3	1.2	0.8	1.74
028	112.6	35.5	8.1	4	1.4	1.1	2.10
029	77.6	23.4	8.3	5.5	1.9	0.7	3.28
030	86.8	30.8	9.0	4.8	1.7	0.7	2.78
031	81.4	26.3	7.7	5.6	2.3	0.6	3.03
032	62.8	21.5	10.8	4.9	2.1	1.2	2.25
033	85.9	25.7	8.7	4.7	1.6	0.9	2.69
034	73.1	22.3	8.8	4.4	1.1	0.6	3.03
035	58.4	23.1	8.5	5.4	2.0	0.8	3.04
036	76.5	27.8	8.1	5.2	2.2	0.4	2.82
037	65.2	23.7	9.7	5.3	1.9	0.7	3.08
038	104.0	30.7	10.0	3.4	1.5	0.4	1.72
039	79.9	25.5	no data	no data	no data	no data	no data
040	68.7	23.0	9.3	4.1	1.6	0.9	2.09
041	71.3	21.3	8.9	3.7	1.8	0.5	1.67
042	75.1	29.3	7.6	3.8	1.7	0.5	1.87
043	77.6	27.5	8.2	5.0	1.8	0.6	2.93
044	52.2	18.9	8.9	3.9	1.8	0.5	1.87
045	79.2	29.8	5.6	4.3	1.9	0.4	2.22
046	82.8	30.4	8.4	5.3	1.9	0.8	3.04
047	67.3	23.8	10.0	5.7	1.5	1.4	3.56
048	77.7	27.9	9.8	no data	no data	no data	no data
049	60.0	22.3	10.5	4.8	1.7	0.6	2.83
050	91.1	31.5	9.2	3.6	1.2	0.7	2.08
051	95.7	32.3	12.8	5.4	1.6	1.4	3.16

InSight study - Biomedical results of all subjects at 6 months

	Weight (kg)	BMI (kg/m^2)	HbA1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	Triglycerides (mmol/l)	LDL cholesterol (mmol/l)
001	no data	no data	9.4	4.5	0.9	1.3	2.95
002	84.4	28.2	8.0	4.1	2.5	0.7	1.25
003	no data	no data	7.5	3.9	1.2	1.5	1.95
004	no data	no data	8	no data	no data	no data	no data
005	no data	no data	8.4	no data	no data	no data	no data
006	no data	no data	6.3	4.2	1.8	0.5	2.17
007	no data	no data	8.3	no data	no data	no data	no data
008	71.4	30.1	no data	4.9	0.8	3.7	2.42
009	74.6	28.8	6.3	4.4	1.6	1.1	2.30
010	82.3	24.3	no data	no data	no data	no data	no data
011	63.7	23.1	7.5	4.9	2.0	0.4	2.72
012	no data	no data	8.5	no data	no data	no data	no data
013	59.3	22.0	8.0	3.5	1.5	0.4	1.82
014	63.8	25.9	7.9	no data	no data	no data	no data
015	no data	no data	8.8	5.4	1.5	0.8	3.54
016	no data	no data	no data	5.3	1.9	0.5	3.17
017	no data	no data	6.2	4.3	1.4	0.8	2.54
018	no data	no data	no data	4.4	2.2	0.5	1.97
019	no data	no data	7.4	2.8	1.4	0.5	1.17
020	no data	no data	8.4	no data	no data	no data	no data
021	no data	no data	7.3	4.7	1.8	0.6	2.63
022	85.6	32.6	6.7	4.4	1.5	1.9	2.04
023	65.4	25.5	6.7	4.3	2.1	0.6	1.93
024	83.7	28	9.2	6.2	1.0	4.1	3.34
025	61.2	21.7	8.1	3.7	1.2	1.1	2.00
026	51.5	22.3	9.1	4.7	2.6	0.6	1.83
027	83.0	23.0	9.5	3.3	1.2	0.8	1.74
028	110.6	34.9	7.9	4.0	1.4	1.1	2.10
029	75.1	22.7	8.1	5.2	1.9	1	2.85
030	87.2	30.9	9.3	4.8	1.7	0.7	2.78
031	80.0	25.8	8.1	5.6	2.3	0.6	3.03
032	60.4	20.7	9.3	4.9	2.1	1.2	2.25
033	90.4	27	8.5	4.5	1.43	1	2.62
034	no data	no data	8.8	4.6	1.1	0.5	3.27
035	58.7	23.3	8.2	5.5	2.1	0.7	3.08
036	77.3	28.1	9.3	4.5	2.1	0.6	2.13
037	65.5	23.8	10.3	5.1	1.9	0.7	2.88
038	104.6	30.9	9.3	2.9	1.3	0.4	1.42
039	80	25.8	no data	no data	no data	no data	no data
040	67	22.4	8.7	3.7	1.5	0.6	1.93
041	72.5	21.6	9.4	4.1	1.6	0.8	2.14
042	73.6	28.7	8.1	5.1	1.6	0.6	3.23
043	77.4	27.4	7.7	4.8	1.9	0.7	2.58
044	52.2	18.9	7.8	3.7	1.6	0.5	1.87
045	79.2	29.8	no data	no data	no data	no data	no data
046	79.5	29.2	8.8	5.4	1.87	0.8	3.17
047	no data	no data	9.1	5.7	1.5	0.9	3.79
048	79.5	28.5	9.5	no data	no data	no data	no data
049	58.5	21.8	8.6	4.2	1.4	0.7	2.48
050	92.2	31.9	9	4.4	1.2	0.9	2.79
051	92.9	31.4	10.2	5.3	1.7	1.7	2.83

InSight study - Biomedical results of all subjects at 1 year

	Weight (kg)	BMI (kg/m^2)	HbA1c (%)	Total cholesterol (mmol/l)	HDL cholesterol (mmol/l)	Triglycerides (mmol/l)	LDL cholesterol (mmol/l)
001	no data	no data	9.8	6.7	1.1	1.5	4.85
002	81	27.1	7.9	3.7	1.9	0.6	1.50
003	94.9	32.5	7.5	4.2	1.3	1.2	2.30
004	no data	no data	8	4.3	1.3	0.9	2.55
005	85	30.1	8.4	4.1	1.4	0.9	2.25
006	85	30.1	7.9	4.3	1.9	0.4	2.20
007	no data	no data	7.9	no data	no data	no data	no data
008	70.3	29.6	7.1	4.3	0.9	2.5	2.26
009	75.2	29	6.2	4.7	1.5	0.7	2.88
010	82.3	24.3	6.4	5.6	1.3	1.1	3.80
011	67.4	24.5	8.7	4.8	2.2	1.0	2.15
012	62.8	24.2	8.0	4.5	2.7	0.5	1.57
013	58.4	21.7	8.5	4.0	1.6	0.4	2.22
014	66.7	27.1	7.4	4.5	2.1	1.2	1.85
015	67.7	25.2	8.7	3.9	1.4	0.7	2.18
016	74.6	22.8	7.2	4.3	2	0.7	1.98
017	no data	no data	6.3	no data	no data	no data	no data
018	no data	no data	7.5	no data	no data	no data	no data
019	79.7	27.6	7.5	no data	no data	no data	no data
020	no data	no data	7	no data	no data	no data	no data
021	71.2	25.2	7.8	5.0	1.9	0.8	2.74
022	85.6	32.6	7.2	6	1.9	1.3	3.51
023	65.4	25.5	7.2	4.3	2.1	0.6	1.93
024	83.7	28	9.6	4.3	1	1.5	2.62
025	63.5	22.5	9	4	1.2	0.9	2.39
026	53.3	23.1	8	5.2	2.7	0.6	2.23
027	83.2	23	8.9	4.2	1.2	1	2.55
028	112.1	35.4	7.8	4.4	1.4	1.2	2.45
029	75.8	22.9	8.6	5.9	2	0.8	3.54
030	82.3	29.2	10.2	5.4	1.8	0.8	3.24
031	79.5	25.7	7.8	5.5	2.1	0.8	3.04
032	60.4	20.7	9.3	4.9	2.1	1.2	2.25
033	no data	no data	8.4	5	1.57	1	2.98
034	no data	no data	8.9	4.6	1.1	0.5	3.27
035	57.9	22.9	no data	no data	no data	no data	no data
036	77.2	28	9.5	4.6	1.9	0.2	2.61
037	no data	no data	no data	no data	no data	no data	no data
038	103.9	30.7	9.5	3.4	1.3	0.6	1.83
039	80.5	26	9.8	4.2	2	1.1	1.70
040	69.2	23.1	8.6	4.7	1.6	0.9	2.69
041	75.5	22.5	8.9	3.4	1.3	0.7	1.78
042	73.6	28.7	8.1	5.1	1.6	no data	no data
043	76.9	27.2	7.9	4.7	1.8	0.6	2.63
044	54.8	19.9	8.5	3.9	1.7	0.5	1.97
045	79.2	29.7	5.5	4.1	1.9	0.6	1.93
046	82.2	30.2	7.9	4.7	1.9	0.6	2.53
047	66.3	23.4	9.5	5.6	1.5	1.2	3.55
048	80.4	28.8	9	no data	no data	no data	no data
049	62.5	23.2	9.2	5.1	1.7	0.6	3.13
050	90.8	31.1	8.7	3.8	1.2	0.6	2.33
051	93.7	31.7	10	5.4	1.5	0.6	3.63

InSight study - PAID results of all subjects at baseline

	Question number (PAID questionnaire)																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
001	2	2	3	3	2	3	4	2	3	2	1	2	3	3	2	2	3	1	3	2
002	0	0	1	0	0	2	3	2	1	2	1	3	2	0	0	1	2	1	1	2
003	1	1	2	0	0	2	1	1	2	3	1	3	0	1	4	1	3	0	2	1
004	1	1	0	0	1	0	1	0	1	0	1	2	2	0	0	1	0	0	1	1
005	2	4	0	0	0	1	0	0	3	0	4	2	0	1	2	3	0	0	2	3
006	1	1	1	0	0	1	1	0	1	0	1	3	3	0	1	0	2	0	1	0
007	1	1	1	3	1	1	4	0	4	0	0	2	2	1	1	2	1	3	3	1
008	2	1	0	0	1	0	2	0	0	0	2	1	2	0	0	0	0	0	0	0
009	0	2	0	0	0	0	0	0	0	0	2	2	2	0	3	0	0	0	0	0
010	3	0	2	1	1	1	4	1	3	1	0	2	3	1	2	1	1	0	0	0
011	3	2	3	0	0	4	2	2	4	3	0	4	0	0	2	2	1	0	0	0
012	0	1	1	0	1	1	1	0	1	0	2	3	3	0	0	3	1	0	2	3
013	2	2	0	1	0	2	1	0	1	0	3	3	1	0	0	1	3	1	0	2
014	1	2	0	1	0	1	1	0	2	0	0	2	1	0	0	1	0	0	1	1
015	0	0	3	0	3	2	0	3	2	3	3	3	3	3	0	0	3	3	3	3
016	0	1	2	2	1	1	2	1	3	2	1	2	3	0	0	2	1	0	2	1
017	2	0	2	2	0	2	2	0	1	2	1	2	0	1	0	2	2	0	0	2
018	2	3	3	0	0	0	2	2	0	0	1	2	2	2	4	1	3	3	0	1
019	1	1	0	0	0	1	2	0	2	0	2	3	2	0	0	1	0	0	3	2
020	0	4	4	4	4	4	4	0	0	4	2	4	4	4	0	3	0	0	4	4
021	0	4	4	0	0	0	2	4	4	0	0	2	0	0	3	4	0	0	4	4
022	2	2	2	0	0	2	2	0	1	1	3	3	3	0	1	0	2	0	1	0
023	1	1	0	0	0	0	1	0	1	0	2	2	3	0	0	0	0	0	1	0
024	1	0	1	0	3	3	0	2	3	3	3	4	4	0	0	3	0	0	3	3
025	2	2	2	0	2	3	1	2	1	3	2	4	3	2	2	3	2	0	1	3
026	0	1	0	0	0	2	1	1	3	1	2	1	0	0	0	1	1	0	0	1
027	1	1	0	2	1	2	3	1	1	1	1	3	2	0	1	1	1	2	3	1
028	1	1	0	1	0	1	2	0	1	0	1	3	2	2	0	3	1	0	3	1
029	2	0	0	0	0	0	2	0	0	0	0	0	0	0	1	0	0	1	0	0
030	2	1	2	2	1	3	2	2	1	2	2	2	2	1	0	2	2	2	1	1

InSight study - PAID results of all subjects at baseline *contd*

	Question number (PAID questionnaire)																			
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
032	4	3	4	2	2	4	4	4	2	3	2	4	4	4	0	4	4	4	2	3
033	0	0	1	1	0	1	1	1	2	2	1	2	2	0	0	0	1	0	0	1
034	2	3	1	2	3	2	2	2	1	2	3	2	2	1	2	2	2	1	2	2
035	3	2	1	3	1	2	1	1	4	1	3	1	3	1	2	2	2	1	1	1
036	1	1	0	0	1	1	1	0	1	0	0	1	1	0	1	1	1	1	1	1
037	3	4	4	2	2	3	3	4	1	4	4	4	3	4	3	3	4	2	3	3
038	3	4	0	1	3	1	2	0	1	2	2	1	2	0	2	1	1	0	1	2
039	0	0	1	0	0	0	0	0	3	0	0	1	0	0	0	0	0	0	0	0
040	1	1	0	1	0	0	0	0	2	0	1	2	2	0	2	1	1	0	2	0
041	2	1	2	1	1	1	3	1	2	0	2	3	2	0	1	1	1	1	1	2
042	1	1	1	2	1	1	1	1	2	1	0	2	1	0	0	2	2	0	1	1
043	1	2	3	0	0	3	2	3	1	0	3	4	3	1	1	1	2	0	2	1
044	0	1	1	1	1	0	0	1	2	0	1	1	1	1	1	1	0	0	1	1
045	1	0	0	1	0	1	1	0	1	0	1	2	1	0	2	1	1	0	0	1
046	4	3	1	2	0	1	0	0	0	0	0	2	0	0	2	4	2	2	4	4
047	3	2	4	2	1	3	3	3	4	2	3	3	3	4	1	1	3	3	3	3
048	3	4	2	0	0	1	1	0	2	0	1	4	3	0	4	1	0	0	3	1
049	1	2	0	0	0	1	1	1	2	0	2	0	1	0	2	0	1	1	1	0
050	0	0	3	0	0	3	3	2	1	3	1	3	2	3	0	2	2	0	0	3
051	0	0	1	1	2	0	0	0	1	0	1	1	0	0	0	0	0	0	0	0

Insight study - PAID results of all subjects at 6 months

	Question number (PAID questionnaire)																			
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
001	1	1	2	1	1	2	2	2	1	1	2	1	1	1	1	2	1	1	2	1
002	0	0	0	0	1	0	1	0	0	2	2	1	0	1	0	0	0	1	1	0
003	0	0	1	0	0	1	1	1	1	0	0	0	0	1	2	0	0	1	0	1
004	0	1	0	0	1	0	0	0	0	0	0	1	2	0	0	1	0	0	1	1
005	1	2	0	0	0	1	0	0	0	0	3	0	0	1	0	1	0	0	2	2
006	0	0	0	0	0	0	0	0	1	0	0	1	1	0	0	0	0	0	1	0
007	1	0	1	4	1	3	4	2	4	0	0	3	4	3	0	1	1	0	3	4
008	1	1	0	0	1	0	2	0	0	0	1	1	1	0	0	0	0	0	0	0
009	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
010	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
011	0	0	2	0	2	3	3	2	2	1	3	3	3	0	0	1	0	1	0	1
012	0	0	0	0	0	0	2	0	1	0	1	2	3	0	0	2	0	0	2	2
013	2	2	1	0	0	1	2	1	0	0	3	2	3	0	0	1	2	1	0	1
014	1	2	0	1	0	1	1	0	2	0	0	2	1	0	0	1	0	0	1	1
015	1	1	3	2	2	4	4	4	0	2	3	4	4	3	2	3	3	0	4	3
016	1	1	2	2	0	2	0	1	3	0	1	2	2	0	0	1	2	1	1	1
017	1	1	1	2	1	2	2	1	2	2	1	3	3	2	0	1	2	1	1	1
018	2	1	2	0	0	0	2	1	0	0	1	2	2	2	4	1	3	3	0	1
019	1	0	1	0	0	0	2	0	2	0	1	2	1	0	0	1	0	0	2	1
020	0	2	4	0	2	4	1	2	0	4	1	4	2	3	0	2	0	0	2	3
021	1	1	1	1	0	1	1	1	2	1	0	2	2	0	0	1	0	0	4	2
022	1	1	2	1	0	1	0	1	3	0	3	4	4	1	0	0	2	2	1	0
023	0	0	1	0	0	0	2	0	1	0	0	1	1	0	1	1	0	0	1	0
024	1	0	1	0	2	3	0	2	2	3	3	2	3	0	0	3	0	0	3	3
025	0	1	2	0	1	2	1	0	1	2	1	4	2	0	2	1	1	0	2	1
026	0	0	2	0	2	0	2	1	2	0	2	3	2	0	0	1	0	1	1	1
027	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
028	1	2	0	0	1	1	2	0	2	0	1	3	2	0	0	1	0	0	1	1
029	2	0	0	0	0	0	2	0	0	0	0	0	0	0	1	0	0	1	0	0
030	2	1	2	2	1	3	2	2	1	2	2	2	2	1	0	2	2	2	1	1

QNC = questionnaire not completed

Insight study - PAID results of all subjects at 6 months *contd*

	Question number (PAID questionnaire)																			
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
031	2	2	2	0	0	3	2	1	1	3	1	4	0	1	3	3	3	0	3	3
032	3	3	4	2	2	3	4	3	2	2	2	3	4	3	1	2	3	3	2	3
033	0	0	1	0	0	1	1	0	0	0	0	1	1	0	0	0	0	0	0	0
034	0	2	1	2	0	2	1	2	0	2	2	1	1	0	1	2	0	1	0	1
035	0	0	1	0	0	1	0	0	1	1	2	2	0	0	0	1	0	0	0	1
036	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
037	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
038	3	2	3	1	1	1	2	0	1	2	3	4	4	1	2	1	2	1	1	2
039	1	1	0	0	0	1	0	0	3	0	2	1	0	0	0	0	0	0	1	0
040	0	0	0	1	0	0	0	0	1	0	0	1	2	0	0	0	0	1	0	0
041	1	1	1	0	1	2	3	0	2	1	1	3	2	0	0	1	1	1	0	0
042	0	0	0	2	2	2	1	0	2	1	1	1	1	0	0	2	1	0	1	1
043	1	0	2	0	0	2	1	1	0	0	1	2	2	0	2	1	0	0	0	1
044	0	1	1	1	1	0	0	1	2	0	1	1	1	1	1	1	0	0	1	1
045	0	0	1	0	0	1	0	0	1	0	0	2	1	0	1	1	1	1	0	1
046	2	3	1	2	0	1	0	0	0	0	0	2	0	0	2	3	2	2	3	2
047	2	0	2	2	2	2	2	2	2	2	2	2	3	2	1	1	1	2	2	2
048	0	0	0	0	1	0	1	0	1	0	1	2	1	0	2	0	0	0	1	0
049	0	0	0	1	0	1	0	0	1	0	0	1	0	0	0	0	0	1	0	0
050	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
051	0	0	1	0	1	1	1	0	1	1	0	1	0	0	0	0	0	0	0	0

QNC = questionnaire not completed

Insight study - PAID results of all subjects at 1 year

	Question number (PAID questionnaire)																			
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
001	1	1	2	1	1	2	2	2	1	1	2	1	1	1	1	2	1	1	2	1
002	0	0	0	0	1	0	1	0	0	2	2	1	0	1	0	0	0	1	1	0
003	0	0	1	0	0	1	1	1	1	0	0	0	0	1	2	0	0	1	0	1
004	1	2	0	0	0	1	0	0	0	0	3	0	0	1	0	1	0	0	2	2
005	1	2	0	0	0	1	0	0	0	0	3	0	0	1	0	1	0	0	2	2
006	0	0	0	0	0	0	0	0	1	0	0	1	1	0	0	0	0	0	1	0
007	2	0	2	4	1	3	4	2	4	0	0	3	4	3	0	1	1	0	3	4
008	1	0	0	0	1	1	1	0	0	1	1	1	1	0	0	0	0	0	0	0
009	0	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0
010	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
011	0	0	2	0	2	3	3	2	2	1	3	3	3	0	0	1	0	1	0	1
012	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
013	2	2	1	0	0	1	2	1	0	0	3	2	3	0	0	1	2	1	2	1
014	1	0	1	1	0	1	1	0	1	0	0	1	1	0	0	1	0	0	1	1
015	1	1	3	2	2	4	4	4	0	2	3	4	4	3	2	3	3	0	4	3
016	1	1	2	2	0	2	0	1	3	0	1	2	2	0	0	1	2	1	1	1
017	1	1	1	2	1	2	2	1	2	2	1	3	3	2	0	1	2	1	1	1
018	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
019	1	0	1	0	0	0	2	0	2	0	1	2	1	0	0	1	0	0	2	1
020	0	2	4	0	2	4	1	2	0	4	1	4	2	3	0	2	0	0	2	3
021	1	1	1	1	0	1	1	1	2	1	0	2	2	0	0	1	0	0	4	2
022	1	1	2	1	0	1	0	1	3	0	3	4	4	1	0	0	2	2	1	0
023	0	0	1	0	0	0	2	0	1	0	0	1	1	0	1	1	0	0	1	0
024	1	0	1	0	2	2	0	2	3	2	2	3	3	0	0	2	0	0	3	3
025	0	1	2	0	1	2	1	0	1	2	1	4	2	0	2	1	1	0	2	1
026	0	0	2	0	2	0	2	1	2	0	2	3	2	0	0	1	0	1	1	1
027	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
028	1	2	0	0	1	1	2	0	2	0	1	3	2	0	0	1	0	0	1	1
029	2	0	0	0	0	0	2	0	0	0	0	0	0	0	1	0	0	1	0	0
030	2	1	2	2	1	2	2	2	1	2	2	2	2	1	0	2	2	2	1	1

QNC = questionnaire not completed

Insight study - PAID results of all subjects at 1 year
contd

	Question number (PAID questionnaire)																			
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**16**	**17**	**18**	**19**	**20**
031	2	2	2	0	0	1	2	1	1	3	1	4	0	1	3	3	3	0	3	3
032	4	3	4	2	2	2	3	2	2	3	2	2	3	3	1	3	2	3	2	3
033	0	0	1	0	0	1	1	0	0	0	0	1	1	0	0	0	0	0	0	0
034	0	2	1	2	0	2	1	2	0	2	2	1	1	0	1	2	0	1	0	1
035	0	0	1	0	0	1	0	0	1	1	2	2	0	0	0	1	0	0	0	1
036	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
037	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
038	3	2	3	1	1	1	2	0	1	2	3	4	4	1	2	1	2	1	1	2
039	1	1	0	0	0	1	0	0	3	0	2	1	0	0	0	0	0	0	1	0
040	0	0	0	1	0	0	0	0	1	0	0	1	2	0	0	0	0	1	0	0
041	1	1	1	0	1	2	3	0	2	1	1	3	2	0	0	1	1	1	0	0
042	0	0	0	2	2	2	1	0	2	1	1	1	1	0	0	2	1	0	1	1
043	0	0	1	0	1	1	0	0	0	0	2	1	2	0	0	0	0	0	1	0
044	0	1	2	2	1	0	0	1	2	0	1	1	1	1	0	1	0	0	1	0
045	0	1	0	0	0	1	0	0	1	0	1	1	2	1	1	2	1	0	1	2
046	2	3	1	2	1	1	1	0	0	0	0	2	0	0	2	1	1	1	2	2
047	3	2	2	2	1	3	3	3	2	2	3	3	3	3	1	1	3	2	3	3
048	2	3	2	1	0	1	1	0	2	0	1	2	3	0	3	1	0	0	3	1
049	1	2	0	0	0	1	1	1	2	0	2	0	1	0	2	0	1	1	1	0
050	0	0	3	0	1	2	3	2	1	2	1	1	2	2	0	2	2	0	0	3
051	0	0	1	1	0	0	0	0	1	0	1	0	0	0	0	1	0	0	0	0

QNC = questionnaire not completed

InSight study - hypoglycaemia questionnaire results of all subjects at baseline

	Question number							
	1	2	3	4	5	6	7	8
001	always	no	once_twice	once_twice	once a week	never	3.5-4.0	often
002	sometimes	yes	more	more	once a week	never	3.0-3.5	sometimes
003	always	no	never	never	2-3 times	never	3.5-4.0	often
004	always	yes	once_twice	never	once a week	never	3.0-3.5	often
005	sometimes	yes	more	once_twice	2-3 times	2-3 times	less than 3.0	sometimes
006	always	yes	never	never	2-3 times	never	3.0-3.5	often
007	no longer	yes	more	more	once a week	2-3 times	less than 3.0	rarely
008	sometimes	no	never	never	never	never	3.5-4.0	sometimes
009	always	yes	never	never	2-3 times	never	3.5-4.0	often
010	always	yes	never	never	once a week	never	3.5-4.0	always
011	always	no	never	never	1-3 times	never	3.5-4.0	often
012	sometimes	yes	never	never	daily	1-3 times	less than 3.0	often
013	sometimes	yes	more	never	once a week	1-3 times	less than 3.0	sometimes
014	sometimes	yes	never	never	daily	1-3 times	3.0-3.5	often
015	always	no	once_twice	more	1-3 times	never	3.5-4.0	always
016	sometimes	yes	never	never	2-3 times	1-3 times	3.0-3.5	often
017	always	no	once_twice	once_twice	daily	never	less than 3.0	often
018	always	no	never	never	once a week	never	3.5-4.0	always
019	sometimes	yes	once_twice	once_twice	2-3 times	1-3 times	3.5-4.0	often
020	always	yes	more	never	once a week	never	less than 3.0	always
021	no longer	yes	more	once_twice	once a week	2-3 times	less than 3.0	sometimes
022	sometimes	yes	never	never	1-3 times	once a week	less than 3.0	often
023	always	no	once_twice	never	1-3 times	never	3.5-4.0	often
024	always	yes	never	never	2-3 times	never	less than 3.0	always
025	always	no	never	never	2-3 times	never	3.5-4.0	always
026	always	no	never	never	1-3 times	never	3.5-4.0	often
027	sometimes	no	once_twice	never	once a week	never	3.5-3.5	always
028	sometimes	yes	more	never	once a week	1-3 times	3.5-3.5	sometimes
029	always	no	never	never	2-3 times	never	3.5-4.0	often
030	always	no	never	never	1-3 times	never	3.5-4.0	always

InSight study - hypoglycaemia questionnaire results of all subjects at baseline *contd*

	Question number							
031	1	2	3	4	5	6	7	8
032	always	no	never	never	1-3 times	never	3.5-4.0	often
033	always	yes	never	never	1-3 times	never	3.5-4.0	often
034	always	no	never	never	2-3 times	never	3.0-3.5	often
035	sometimes	yes	once_twice	never	2-3 times	never	3.0-3.5	often
036	always	no	never	never	2-3 times	1-3 times	3.0-3.5	always
037	always	no	never	never	never	1-3 times	3.0-3.5	always
038	always	no	never	never	1-3 times	never	3.5-4.0	often
039	no longer	yes	more	once_twice	never	1-3 times	less than 3.0	rarely
040	always	no	more	never	once a week	once a week	3.0-3.5	always
041	sometimes	yes	never	never	once a week	1-3 times	3.5-4.0	often
042	sometimes	yes	once_twice	never	never	never	3.5-4.0	often
043	always	no	never	never	2-3 times	never	3.5-3.5	always
044	always	no	never	never	daily	once a week	3.5-4.0	always
045	always	no	once_twice	never	1-3 times	never	3.5-4.0	always
046	sometimes	yes	more	once_twice	daily	2-3 times	3.5-4.0	rarely
047	always	no	never	never	once a week	never	3.5-4.0	always
048	sometimes	no	once_twice	once_twice	2-3 times	never	less than 3.0	often
049	sometimes	yes	never	never	2-3 times	1-3 times	less than 3.0	often
050	always	yes	never	never	once a week	never	3.5-4.0	often
051	always	no	never	never	once a week	never	3.5-4.0	always

InSight study - hypoglycaemia questionnaire results of all subjects at 6 months

	Question number							
	1	2	3	4	5	6	7	8
001	always	no	once or twice	never	never	never	3.5-4.0	always
002	always	no	once or twice	never	1-3 times	never	3.5-4.0	often
003	always	no	never	never	once	never	3.5-4.0	always
004	always	yes	never	never	1-3 times	never	3.0-3.5	always
005	always	yes	once or twice	never	1-3 times	1-3 times	3.0-3.5	often
006	always	yes	never	never	once	once_week	3.0-3.5	often
007	no longer	yes	more	more	1-3 times	2-3 times	less than 3.0	rarely
008	always	no	never	never	never	never	3.5-4.0	sometimes
009	always	no	once or twice	never	1-3 times	never	3.5-4.0	often
010	always	no	never	never	once	once_week	3.5-4.0	often
011	always	no	never	never	2-3 times	once_week	3.5-4.0	always
012	always	yes	never	never	2-3 times	never	3.0-3.5	always
013	always	yes	never	never	1-3 times	never	3.5-4.0	often
014	sometimes	yes	never	never	daily	1-3 times	3.0-3.5	often
015	always	no	never	never	1-3 times	never	3.5-4.0	always
016	always	yes	never	never	2-3 times	once_week	3.0-3.5	always
017	always	yes	once or twice	never	2-3 times	never	3.0-3.5	often
018	always	no	never	never	once a week	never	3.5-4.0	always
019	sometimes	yes	once or twice	never	1-3 times	never	3.0-3.5	often
020	always	no	more	never	once	never	3.5-4.0	always
021	sometimes	yes	never	never	1-3 times	1-3 times	less than 3.0	often
022	sometimes	yes	never	never	2-3 times	once_week	3.0-3.5	often
023	always	no	never	never	1-3 times	never	3.5-4.0	always
024	always	yes	never	never	2-3 times	never	less than 3.0	always
025	always	no	never	never	2-3 times	never	3.5-4.0	always
026	always	no	never	never	once	never	3.5-4.0	often
027	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
028	sometimes	yes	more	once_twice	2-3 times	once_week	3.0-3.5	often
029	always	no	never	never	2-3 times	never	4.0-4.5	often
030	always	no	never	never	1-3 times	never	3.5-4.0	always

QNC = questionnaire not completed

InSight study - hypoglycaemia questionnaire results of all subjects at 6 months *contd*

	Question number							
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**
031	always	no	never	never	1-3 times	never	3.5-4.0	often
032	always	no	never	never	1-3 times	never	3.5-4.0	often
033	always	yes	never	never	1-3 times	never	3.5-4.0	always
034	always	no	never	never	1-3 times	never	3.5-4.0	always
035	always	yes	never	never	1-3 times	never	3.0-3.5	often
036	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
037	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
038	always	no	never	never	1-3 times	never	3.5-4.0	often
039	no longer	yes	more	more	2-3 times	2-3 times	less than 3.0	rarely
040	always	no	never	never	once	never	3.0-3.5	often
041	always	yes	never	never	once	never	3.5-4.0	often
042	always	yes	once or twice	never	never	never	3.5-4.0	always
043	always	no	never	never	2-3 times	never	3.0-3.5	often
044	always	no	never	never	daily	once a week	3.5-4.0	always
045	always	no	never	never	2-3 times	once_week	3.5-4.0	always
046	sometimes	yes	more	once_twice	daily	2-3 times	3.5-4.0	rarely
047	always	no	never	never	1-3 times	never	3.5-4.0	always
048	always	yes	once or twice	once_twice	1-3 times	1-3 times	less than 3.0	often
049	no longer	yes	never	never	once	2-3 times	3.5-4.0	sometimes
050	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
051	always	no	never	never	once	never	3.5-4.0	always

QNC = questionnaire not completed

InSight study - hypoglycaemia questionnaire results of all subjects at 1 year

	Question number							
	1	2	3	4	5	6	7	8
001	always	no	once or twice	never	never	never	3.5-4.0	always
002	always	no	once or twice	never	1-3 times	never	3.5-4.0	often
003	always	no	never	never	once	never	3.5-4.0	always
004	always	yes	once or twice	never	1-3 times	1-3 times	3.0-3.5	often
005	always	yes	once or twice	never	1-3 times	1-3 times	3.0-3.5	often
006	always	yes	never	never	once	once-week	3.0-3.5	often
007	no longer	yes	more	more	1-3 times	2-3 times	less than 3.0	rarely
008	always	no	never	never	never	never	3.5-4.0	always
009	always	no	once or twice	never	1-3 times	never	3.5-4.0	often
010	always	no	never	never	once	once_week	3.5-4.0	often
011	always	no	never	never	2-3 times	once_week	3.5-4.0	always
012	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
013	always	yes	never	never	1-3 times	never	3.5-4.0	often
014	always	yes	never	never	daily	1-3 times	3.0-3.5	always
015	always	no	never	never	1-3 times	never	3.5-4.0	always
016	always	yes	never	never	2-3 times	once_week	3.0-3.5	always
017	always	yes	once or twice	never	2-3 times	never	3.0-3.5	often
018	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
019	sometimes	yes	once or twice	never	1-3 times	never	3.0-3.5	often
020	always	no	more	never	once	never	3.5-4.0	always
021	sometimes	yes	never	never	1-3 times	1-3 times	less than 3.0	often
022	sometimes	yes	never	never	2-3 times	once_week	3.0-3.5	often
023	always	no	never	never	1-3 times	never	3.5-4.0	always
024	always	yes	never	never	2-3 times	never	less than 3.0	always
025	always	no	never	never	2-3 times	never	3.5-4.0	always
026	always	no	never	never	once	never	3.5-4.0	often
027	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
028	sometimes	yes	more	once or twice	2-3 times	once_week	3.0-3.5	often
029	always	no	never	never	2-3 times	never	4.0-4.5	often
030	always	no	never	never	1-3 times	never	3.5-4.0	always

InSight study - hypoglycaemia questionnaire results of all subjects at 1 year *contd*

	Question number							
	1	2	3	4	5	6	7	8
031	always	no	never	never	1-3 times	never	3.5-4.0	always
032	always	no	never	never	1-3 times	never	3.5-4.0	always
033	always	yes	never	never	1-3 times	never	3.5-4.0	always
034	always	no	never	never	1-3 times	never	3.5-4.0	always
035	always	yes	never	never	1-3 times	never	3.0-3.5	often
036	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
037	QNC	QNC	QNC	QNC	QNC	QNC	QNC	QNC
038	always	no	never	never	1-3 times	never	3.5-4.0	often
039	no longer	yes	more	more	2-3 times	2-3 times	less than 3.0	rarely
040	always	no	never	never	once	never	3.0-3.5	often
041	always	yes	never	never	once	never	3.5-4.0	often
042	always	yes	once or twice	never	never	never	3.5-4.0	always
043	always	no	never	never	once	never	3.5-4.0	often
044	always	no	never	never	2-3 times	once_week	3.5-4.0	often
045	always	no	never	never	2-3 times	1-3 times	3.5-4.0	often
046	always	yes	more	once_twice	daily	2-3 times	3.5-4.0	always
047	always	no	never	never	once a week	never	3.5-4.0	always
048	always	no	once_twice	never	2-3 times	never	less than 3.0	always
049	sometimes	yes	never	never	2-3 times	1-3 times	less than 3.0	often
050	always	yes	never	never	once a week	never	3.5-4.0	often
051	always	no	never	never	once a week	never	3.5-4.0	always

QNC questionnaire not completed