Can we out-walk the type 2 diabetes mellitus epidemic?

This item was submitted to Loughborough University's Institutional Repository by the/an author.

Additional Information:

- Doctoral Thesis. Submitted in partial fulfillment of the requirements for the award of Doctor of Philosophy of Loughborough University.

Metadata Record: [https://dspace.lboro.ac.uk/2134/8077](https://dspace.lboro.ac.uk/2134/8077)

Publisher: © Thomas Edward Yates

Please cite the published version.
This item is held in Loughborough University’s Institutional Repository (https://dspace.lboro.ac.uk/) and was harvested from the British Library’s EThOS service (http://www.ethos.bl.uk/). It is made available under the following Creative Commons Licence conditions.

For the full text of this licence, please go to: http://creativecommons.org/licenses/by-nc-nd/2.5/
Can We Out-Walk the Type 2 Diabetes Mellitus Epidemic?

by

Thomas Edward Yates

Doctoral Thesis
Submitted in partial fulfilment of the requirements for the award of
Doctor of Philosophy of Loughborough University

September 2008

© by Thomas Edward Yates 2008
DEDICATION

For my wife, Rebekka, without whose unwavering support and love this thesis would not have been possible – thank you
ACKNOWLEDGEMENTS

Although this thesis is the original work of the author, I would like to acknowledge the support of the following individuals. First and foremost, it is no exaggeration to say that the work detailed in this thesis would not have been possible without the encouragement, guidance and access to resources I received from Professors Melanie Davies and Kamlesh Khunti of the University of Leicester. Many was the time that I arrived at a meeting with either or both of them feeling dejected and out of my depth, only to leave feeling positive and in command again; such rare interpersonal skills have been as crucial in guiding the development and direction of this thesis as the depth and breadth of their knowledge.

I would also like to thank all of the Diabetes Research Unit at University Hospitals of Leicester NHS Trust, all of whom helped me at one time or another. In particular, Panna Mandalia and Joe Henson were willing and able assistants during clinical measurements sessions, Jacqui Troughton provided much needed expertise and support surrounding the development and delivery of structured education and Dr Nick Taub helped me get to grips with the numerous statistical challenges presented by this thesis.

Duncan Talbot, my link with Unilever Corporate Research, provided his time and expertise to supervise the analysis of insulin and markers of chronic low-grade inflammation detailed in Chapters Three and Six of this thesis.

Last but not least, Dr Trish Gorely and Professor Fiona Bull, my supervisors, helped provide the scientific oversight needed to ensure that this thesis attained the required academic standards. In particular, I would like to thank Dr Gorely for helping me grasp the subtleties underlying the many health behaviour theories, her attentive reading of several drafts of each of the chapters of this thesis and her helpful and insightful comments and Professor Bull for her help in planning the systematic review detailed in Chapter Two as well as her uncompromising support on a number of practical issues.
Abstract

Background
Type 2 diabetes mellitus is a chronic and debilitating disease whose prevalence continues to rise inexorably. Type 2 diabetes is usually preceded by a condition called prediabetes, which is characterised by impaired glucose regulation. Those with prediabetes have a significantly increased risk of developing type 2 diabetes compared to those with normal glucose control and therefore represent a key population in the prevention of type 2 diabetes.

Physical inactivity is thought to be one of the key factors driving the increasing prevalence of prediabetes and type 2 diabetes and consequently forms a pivotal focus of initiatives aimed at their prevention.

Aims
The principal aims of this thesis were to: 1) conduct a systematic review investigating the effectiveness of lifestyle and physical activity interventions at promoting physical activity in individuals with prediabetes and the effect of physical activity change on the risk of developing diabetes; 2) investigate the effect of walking activity on markers of chronic low grade inflammation; and 3) design and evaluate with objectively measured endpoints a physical activity intervention for adults at risk of developing type 2 diabetes that is suitable for implementation in a health care or community setting if found to be effective.

Results
The main findings are listed in the order of the stated aims. 1) Due to the dearth of controlled exercise training studies in those with prediabetes and the absence of evidence that previous diabetes prevention programmes have been successful at initiating clinically significant increases in physical activity, the evidence for the efficacy of physical activity behaviour change at prevention or delaying the progression to type 2 diabetes in those with prediabetes is equivocal. 2) Walking at levels that are consistent with the current physical activity recommendations is associated with reduced chronic low-grade inflammation,
independent of other forms of physical activity. 3) The PREPARE programme, developed after a review of health behaviour theory and the current health care climate, is a theory-driven, group-based structured education programme designed to promote increased walking activity in individuals with prediabetes in a health care setting. A randomized controlled trial was conducted to test two versions of the PREPARE programme, a standard version and a pedometer version, against control conditions (advice leaflet). The standard version encouraged participants to set time-based goals based on generic exercise recommendations, whereas the pedometer version enabled participants to set personalized steps-per-day goals and to objectively self-monitor their daily physical activity levels using a pedometer. One hundred and three individuals were recruited to the study and follow-up was conducted at 3, 6 and 12 months. At 12 months both intervention conditions were successful at achieving significant increases in objectively measured ambulatory activity; compared to the control group, those who received the pedometer version of the PREPARE programme increased their ambulatory activity by 1952 steps per day (95% CI 953 to 2951) and those who received the standard version by 1480 steps per day (95% CI 436 to 2522). However, significant improvements in glucose tolerance were only seen in the pedometer group, where 2-h glucose levels decreased by -0.94 mmol/l (95% CI -1.79 to -0.10) compared to control conditions, despite no significant change in body weight or waist circumference.

Conclusions

This thesis has identified important limitations in the current evidence linking physical activity to the prevention of type 2 diabetes in those with prediabetes and has addressed several of these limitations by developing a theory-driven structured education programme which was shown to be successful at promoting physical activity and improving glucose tolerance in those with prediabetes to levels that are equal to or greater than previous multi-factor diabetes prevention programmes. This is likely to have important implications for future diabetes prevention trials and clinical practice in the United Kingdom.
# Table of Contents

**Chapter One: Introduction** .................................................................................................................. 12
  Primary research aims .......................................................................................................................... 18

**Chapter Two: The role of physical activity in the management of impaired glucose tolerance: a systematic review** .................................................................................................................. 19
  Introduction ...................................................................................................................................... 21
  Materials and Methods ..................................................................................................................... 23
    Search strategy ............................................................................................................................... 23
    Inclusion criteria ............................................................................................................................. 23
      Subjects ...................................................................................................................................... 23
      Outcome measures ......................................................................................................................... 24
      Type of study ................................................................................................................................. 24
    Analysis ........................................................................................................................................ 24
  Results ........................................................................................................................................... 25
    Study design .................................................................................................................................. 25
    Sample size .................................................................................................................................... 25
    Subjects ........................................................................................................................................ 27
    Gender .......................................................................................................................................... 27
    Intervention conditions ................................................................................................................. 27
    Outcomes ..................................................................................................................................... 28
    Incidence of diabetes and physical activity .................................................................................. 28
    2-hour post-challenge plasma glucose and physical activity ..................................................... 29
    Fasting glucose ............................................................................................................................... 30
  Discussion ........................................................................................................................................ 31
  Chapter Summary ............................................................................................................................ 35

**Chapter Three: Walking and markers of chronic low-grade inflammation** .................................... 36
  Background ..................................................................................................................................... 38
  Methods .......................................................................................................................................... 40
    Participants .................................................................................................................................... 40
    Measures ....................................................................................................................................... 40
      Physical activity .......................................................................................................................... 40
      Biochemical, clinical and demographic measurements .................................................................. 41
    Statistical analysis ......................................................................................................................... 41
  Results ........................................................................................................................................... 43
  Discussion ........................................................................................................................................ 47
  Chapter Summary ............................................................................................................................ 50

**Chapter Four: Development of a physical activity intervention: the PREPARE programme** ........ 51
  Part One: A consideration of the current health care climate ....................................................... 53
    Section summary: .......................................................................................................................... 55
  Part Two: A consideration of health behaviour theory ................................................................... 56
    Social cognitive theory ................................................................................................................... 59
    Implementation Intentions ............................................................................................................. 61
    Common Sense Model .................................................................................................................. 61
    Dual process theory ...................................................................................................................... 63

---
<table>
<thead>
<tr>
<th>Chapter</th>
<th>Title</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chapter One</td>
<td>Research</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter One</td>
<td>Research</td>
<td>Methods</td>
</tr>
<tr>
<td>Chapter One</td>
<td>Research</td>
<td>Background</td>
</tr>
<tr>
<td>Chapter One</td>
<td>Pilot Two</td>
<td>Chapter Summary</td>
</tr>
<tr>
<td>Chapter Two</td>
<td>Part Four: The PREPARE programme</td>
<td>Data analysis</td>
</tr>
<tr>
<td>Chapter Two</td>
<td>Part Four: The PREPARE programme</td>
<td>Ethics</td>
</tr>
<tr>
<td>Chapter Two</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Two</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Three</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Three</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Four</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Four</td>
<td>Part Four: The PREPARE programme</td>
<td>Measures</td>
</tr>
<tr>
<td>Chapter Five</td>
<td>Pilot One</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter Five</td>
<td>Pilot One</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter Five</td>
<td>Pilot One</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter Six</td>
<td>Part Four: The PREPARE programme</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter Six</td>
<td>Part Four: The PREPARE programme</td>
<td>Results</td>
</tr>
<tr>
<td>Chapter Six</td>
<td>Part Four: The PREPARE programme</td>
<td>Results</td>
</tr>
</tbody>
</table>

**Part One: The PREPARE programme**

**Part Two: A consideration of exercise mode and the pedometer**

**Part Three: A consideration of exercise mode and the pedometer**

**Part Four: The PREPARE programme**
List of Tables

Table 3.1: Characteristics of study participants across activity categories .................. 44

Table 4.1: Outline of the PREPARE programme ......................................................... 69

Table 5.1: Pre- and post-programme illness perception and efficacy scores .............. 78

Table 5.2: Baseline and follow-up data for physical activity, illness perceptions and exercise self-efficacy ................................................................. 85

Table 6.1: Clinical, lifestyle and demographic characteristics of study participants overall and by group ............................................................... 104

Table 6.2: Psychological characteristics of study participants overall and by group .......... 106

Table 6.3. Change from baseline and the associated intervention effect for selected biochemical and anthropometric data measured outcomes at 3, 6 and 12 months .... 108

Table 6.4: Change from baseline and the associated intervention effect for measures of physical activity and diet at 3, 6 and 12-months .............................................. 111

Table 6.5: Change from baseline and the associated intervention effect for illness perceptions and efficacy beliefs at 3, 6 and 12-months .............................................. 114

Table 6.6: partial correlation coefficients, adjusted for age and sex, showing the strength of the association of change in measures of physical activity and diet with selected biochemical variables at 12-months ................................................................. 118
List of Figures

Figure 2.1: Flow diagram of literature search..................................................26

Figure 3.1: Association of walking status with circulating interleukin-6 (IL-6),
tumor necrosis factor-α (TNFα), high sensitivity C-reactive protein (CRP)
and fasting insulin.......................................................................................46

Figure 4.1: Intervention mapping....................................................................58

Figure 6.1: PREPARE programme study profile.............................................103
List of abbreviations

2-h glucose 2-hour post-challenge plasma glucose
ADA American Diabetes Association
ADDITION Anglo-Danish-Dutch Study of Intensive Treatment In People with Screen Detected Diabetes
CI confidence interval
CRP C-reactive protein
DESMOND Diabetes Education and Self Management for Ongoing and Newly Diagnosed
DINE Dietary Instrument for Nutritional Education
DPP Diabetes Prevention Program
E group group randomized to receive structured education without pedometer use
EP group group randomized to receive structured education with pedometer use
FDPS Finnish Diabetes Prevention Study
HOMA-IR homeostasis model assessment of insulin resistance
IFG impaired fasting glucose
IGT impaired glucose tolerance
IL-6 interleukin-6
IMD index of multiple deprivation
IPAQ International Physical Activity Questionnaire
MRC Medical Research Council
MeSH medical search headings
MET metabolic equivalents
NHS National Health Service
NICE National Institute for Health and Clinical Excellence
OGTT oral glucose tolerance test
PREPARE Prediabetes Risk Education and Physical Activity Recommendation and Encouragement
SCT social cognitive theory
SD standard deviation
SE standard error
STAR Screening Targeted to those At Risk
TNFα tumor necrosis factor-α
TTM transtheoretical model
Chapter One

Introduction
Type 2 diabetes mellitus is a chronic and debilitating disease characterised by an inability to adequately regulate blood glucose levels. In the short term the symptoms of type 2 diabetes are associated with a reduced quality of life, whilst in the longer term the disease may lead to serious complications such as blindness, renal failure and amputation (Massi-Benedetti 2002). The life expectancy of individuals with type 2 diabetes may be shortened by as much as 15 years, with up to 75% dying of cardiovascular disease (Davies et al. 2004).

The prevalence of type 2 diabetes mellitus has risen so sharply over the past half century that it is now commonly referred to as an epidemic (Colagiuri et al. 2005, Wareham & Forouhi 2005) and it is currently estimated to be the fifth leading cause of mortality globally (Roglic et al. 2005). In the United Kingdom (UK), approximately 5% of the total National Health Service (NHS) resources and up to 10% of hospital inpatient resources are devoted to the care and treatment of type 2 diabetes (Department of Health 2001); these figures are set to rise in the future and will represent a serious clinical and financial challenge to the United Kingdom’s health system (Bagust et al. 2002).

Type 2 diabetes is at one end of a continuous glucose control spectrum with normal glucose control at the other. In between there exists a condition called prediabetes or intermediate hyperglycaemia, defined as impaired glucose tolerance and/or impaired fasting glucose (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 2003, World Health Organization 2006), where blood glucose levels are elevated above the normal range but do not satisfy the criteria for type 2 diabetes. In most countries between 10% and 20% of adults have prediabetes based on WHO criteria (World Health Organization 2006, Santaguida et al. 2005), of which an estimated 5 to 12 % develop type 2 diabetes per year (Santaguida et al. 2005). Indeed, the majority of individuals who develop type 2 diabetes have prediabetes in the preceding 5 years or so (Unwin et al. 2002). Individuals with prediabetes will therefore form a significant proportion of the health care burden associated with diabetes in the future and present a key population in the fight against type 2 diabetes.

Although there is a genetic component to the development of prediabetes and diabetes and their co-morbidities, deleterious effects are generally only seen in environments where energy-dense food is plentiful and the link between physical activity and food procurement has been broken. The fundamental reasons for this can be usefully explored by applying
Dobzhansky's famous maxim that "nothing in biology makes sense except in the light of evolution" (Dobzhansky 1973). Historically, the survival of a species has depended on its ability to adapt to the environment in which it finds itself and to continue to adapt to environmental change over time. Modern humans evolved during the Palaeolithic period, which began approximately 2.6 million years ago and ended 10,000 years ago (Cordain et al. 1998). During this period humans evolved into highly adapted hunter-gatherers, where the ability to undertake high levels of physical activity in the procurement of food was a prerequisite for survival (Chakravarthy & Booth 2004, Cordain et al. 1998). In particular it has been speculated that evolutionary pressures caused the human lineage to adapt to environments that demanded ever-increasing amounts of physical activity. For example, *Australopithecus afarensis*, one of the earliest known human like species, is estimated to have expended 76 KJ/kg/day due to physical activity, an amount that gradually increased through the evolving human phenotype to early *Homo sapiens*, which are estimated to have expended 94 KJ/kg/d due to physical activity, an amount consistent with modern hunter-gatherer societies (Cordain et al. 1998). This trend coincided with increasing brain size and an increasing reliance on animal-based food sources. Interestingly, this is in contrast to the chimpanzee lineage which maintained a primarily vegetarian diet and are estimated to expend around 43 KJ/Kg/day due to physical activity (Cordain et al. 1998).

Whilst the emergence of agriculture around 10,000 years ago had a dramatic effect on the structure of human society and profoundly changed the way food was procured, physical activity levels are likely to have remained high and to have continued to be linked to the production and procurement of food in agricultural societies for the majority of the population (Egger et al. 2001, James 1995). Not until the advent of the industrial revolution over a century ago was the link between physical activity and food production gradually broken in many societies, a process that occurred with particular rapidity after the second world war. This has led to a modern society that is characterised by high levels of physical inactivity (Chakravarthy & Booth 2004, Cordain et al. 1998). Indeed, a modern office worker is estimated to expend around 36 KJ/kg/day due to physical activity, which is a third of that of our hunter-gatherer ancestors and is therefore likely to be far removed from conditions required for optimum metabolic health (Chakravarthy & Booth 2004, Cordain et al. 1998).
The decreasing levels of physical activity in modern societies coincided with a dramatic increase in the incidence of type 2 diabetes; a six-fold increase occurred in the latter half of the 20th century (Booth et al. 2000). The decrease in levels of physical activity and increase in the prevalence of type 2 diabetes are thought to be linked through mechanisms that would have conferred an advantage during the Palaeolithic period but which become maladaptive in a sedentary industrialized society (Chakravarthy & Booth 2004, Cordain et al. 1998). Theoretical frameworks that have attempted to explain the mechanistic link between modern environmental and lifestyle factors, such as physical inactivity, and the increase in non-communicable diseases, such as type 2 diabetes, include the "thrifty genotype" and "thrifty phenotype" hypotheses (Hales & Barker 2001, Neel 1962).

However, regardless of the precise underlying evolutionary explanation for the increasing prevalence of type 2 diabetes, there are numerous proven mechanisms linking physical inactivity to metabolic dysfunction in humans (Hawley 2004, Ivy et al. 1999), and epidemiological studies have consistently shown a strong inverse association between levels of moderate- to vigorous-intensity physical activity and the risk of developing type 2 diabetes (Bassuk & Manson 2005).

Although physical inactivity is now recognized as serious public health burden on a global level, the majority of individuals in many industrialised countries, including the United Kingdom, fail to meet even the minimum exercise recommendations (Department of Health 2004a). Therefore it is important that successful methods of promoting physical activity are identified. Several successful lifestyle diabetes prevention trials in at-risk populations (Knowler et al. 2002, Tuomilehto et al. 2001) have attracted the attention of clinicians, governments and the media in recent years. However, these programmes have several important limitations, such as: they may not be cost-effective (Icks et al. 2007, Palmer et al. 2004); they involve intensive use of health care resources; and they are not proven in the United Kingdom (Davies et al. 2004). Moreover, given that previous diabetes prevention programmes have measured physical activity by self-report, the effectiveness of these programmes at promoting physical activity is uncertain (see Chapter Two). This mirrors a wider trend where traditional physical activity promotion interventions, largely based on one-to-one counselling, have met with limited success, particularly over the longer term (Dishman & Buckworth 1996, Hillsdon et al. 2005a). Therefore it is vital that innovative, successful ways of promoting physical activity are developed and evaluated using objective measures of physical activity. In particular, it is important that future
physical activity interventions are designed specifically for those identified with an increased risk of developing a serious chronic disease, such as those with prediabetes, and that they use methods that are compatible with the infrastructure of national health services and are cost-effective.

Chapter Two of this thesis describes a systematic review investigating the effectiveness of lifestyle and physical activity interventions at promoting physical activity in individuals with prediabetes and the effect of physical activity change on the risk of developing diabetes and improving glucose control. In the context of this thesis, this systematic review is of primary importance as it was instrumental in shaping and informing the direction of the research described in later chapters.

Chapter Three broadens the investigation of the effect of physical activity on metabolic health by investigating whether biochemical markers of chronic low-grade inflammation are associated with levels of walking activity. Markers of chronic low-grade inflammation are thought to play a key role in the pathogenesis of type 2 diabetes and therefore may form a mediating link between lifestyle behaviours and metabolic health. This is the first study to investigate the independent effect of walking activity on markers of chronic low-grade inflammation, therefore the findings will be of clinical importance considering that walking is, for the majority of individuals, the most accessible form of physical activity.

Chapter Four details the background, theoretical rationale and content of the Prediabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme. The PREPARE programme is a group-based structured education programme aimed at promoting physical activity, predominantly walking activity, in individuals who have been identified with an increased risk of developing type 2 diabetes. Structured group education, which is an alternative to more traditional one-to-one counselling strategies, is increasingly being advocated in the promotion of self-management skills in individuals diagnosed with a chronic disease, including type 2 diabetes. However, structured education has not been tested as a diabetes prevention strategy and has been under-utilized as a method of promoting physical activity. The PREPARE programme was developed to investigate whether this approach to patient care can be used to promote physical activity and improve glucose control in individuals identified with an increased risk of developing type 2 diabetes.
Chapter Five details two pilot studies that were carried out to inform the development of the PREPARE programme and to provide preliminary results indicating the feasibility and acceptability of the programme at initiating behaviour change.

Chapter Six reports a randomized controlled trial designed to test whether the PREPARE programme is effective at initiating long-term behaviour change and improving glucose tolerance in individuals with impaired glucose tolerance; the effect of the programme on other important biochemical, anthropometric and psychological variables was also investigated. This chapter is a culmination of the work described in earlier chapters and forms the central part of this thesis. This chapter reports some novel findings that are likely to have important implications for future research and health care practice.

Chapter Seven provides a concise summary and discussion of the overall importance of the work detailed in this thesis and highlights some important directions for future research.

Although each chapter contributes to the overall structure of this thesis and builds on the chapters before it, each chapter can also be read in isolation or in an order that suits the preference of the interested reader.

The themes and studies presented in this thesis have been widely disseminated through conference presentations and published papers (see Appendix One for a full publication list and copies of peer reviewed articles).

This thesis is the original work of the author. However, along with supervision from Professor Fiona Bull and Dr Trish Gorely of Loughborough University, the work detailed in this thesis was informed by guidance from Professor Melanie Davies and Professor Kamlesh Khunti of the University of Leicester. Professor Melanie Davies and Professor Kamlesh Khunti are founding members of the Diabetes Education and Self Management for Ongoing and Newly Diagnosed (DESMOND) collaborative and are co-directors of the South-East Midlands Diabetes Research Network. This collaboration between Loughborough University and University of Leicester combined the expertise in exercise behaviour available at Loughborough University with the resources, clinical knowledge of
List of primary research aims

The primary aims of this thesis are to:

1. Conduct a systematic review of intervention studies investigating the effect of physical activity on the risk of developing type 2 diabetes and/or improving glucose control in individuals with prediabetes.

2. Carry out a cross-sectional study investigating the effect of walking on markers of chronic low-grade inflammation in individuals screened for type 2 diabetes.

3. Design and test a theory-driven physical activity intervention for individuals identified with prediabetes that could feasibly be implemented in a primary health care setting.
Chapter Two

The role of physical activity in the management of impaired glucose tolerance: a systematic review
This chapter will present a systematic review of the effect of physical activity change on glucose tolerance and the risk of developing type 2 diabetes in at-risk individuals. The review presents a summary of the current evidence surrounding the role of physical activity in the prevention of type 2 diabetes and its conclusions were a significant influence on the work detailed in later chapters.

The review detailed in this chapter was published in Diabetologia (Yates et al. 2007a), where it was the Editor's choice and the subject of an accompanying commentary article (Carnethon 2007). It was also the subject of a subsequent letter to the Editor (Laaksonen et al. 2007) and an accompanying response from the authors of the review (Yates et al. 2007b).
Introduction

Evidence of a link between physical activity and type 2 diabetes emerged in the 1960s and 1970s when studies looking at indigenous societies, such as the Pima Indians living in Arizona, found that a large proportion of indigenous people exposed to Westernized environments were obese and had type 2 diabetes, whereas half a century earlier both conditions had been rare in the same population (West 1974). This observational evidence was further augmented by studies involving immigrants to North America. For example, a study comparing Japanese immigrants living in Hawaii and their native counterparts in Hiroshima found that those living in Japan were half as likely to have diabetes and over twice as likely to engage in moderate- to vigorous-intensity occupational physical activity (Kawate et al. 1979). Further cross-sectional studies in the 1980s went on to quantify the link between physical inactivity and type 2 diabetes (Kriska et al. 1994).

Several large longitudinal studies subsequently showed an inverse association between physical activity and the risk of developing type 2 diabetes over time. For example, a study following 21,271 male physicians over an average of 5 years found a strong dose-response effect between levels of vigorous-intensity physical activity and the relative risk of developing type 2 diabetes (Manson et al. 1992). Similarly, the Nurses’ Health Study which followed 85,000 female nurses over an average of 16 years found that levels of moderate- to vigorous-intensity physical activity were inversely associated with the relative risk of developing type 2 diabetes in a dose-response manner (Hu et al. 2001).

A recent systematic review of epidemiological studies also found that there was a strong inverse association between moderate-intensity physical activity and the risk of developing type 2 diabetes and that compared to being sedentary those that engaged in regular moderate intensity physical activity had around a 30% lower risk of developing type 2 diabetes after adjustment for markers of adiposity (Jeon et al. 2007).

These studies, along with numerous proven biological mechanisms linking physical activity to metabolic health (Hawley 2004, Ivy et al. 1999), provide strong evidence for an association between physical activity and the risk of developing type 2 diabetes and suggest that increased physical activity may reduce the risk of developing diabetes. However, as with all cross-sectional and longitudinal studies, it is difficult to be certain of the strength
and direction of causation and it is possible that the magnitude of the results showing a beneficial effect of physical activity were affected by unknown confounders. Consequently, intervention studies are needed to confirm the association between physical activity and the risk of developing type 2 diabetes. Lifestyle intervention studies that have encouraged weight loss through a combination of dietary change and increased physical activity have reduced the risk of type 2 diabetes in individuals identified with an increased risk of developing diabetes (Hu et al. 2001, Knowler et al. 2002, Kosaka et al. 2005, Pan et al. 1997, Ramachandran A et al. 2006, Tuomilehto et al. 2001). However, because physical activity was not typically analysed independently of other variables, such as weight loss, it is difficult to determine the effectiveness of physical activity behaviour change at protecting against the risk of diabetes independent of other lifestyle variables.

The aim of this review is to investigate whether physical activity behaviour change can reduce the risk of type 2 diabetes in individuals with prediabetes independently of other health behaviours. Prediabetes is the collective term for people with impaired glucose tolerance and/or impaired fasting glucose (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 2003). Individuals with prediabetes are at a significantly increased risk of developing type 2 diabetes: each year between 5-12% of individuals with prediabetes will develop type 2 diabetes, which is up to 12 times the rate observed in those with normal glucose control (Santaguida 2005, World Health Organization 2006)(see Chapter One). Investigating the effect of physical activity change in individuals with prediabetes will determine whether physical activity behaviour change is effective in slowing/stopping the progression to type 2 diabetes in a high risk population.
Materials and Methods

Search strategy

MEDLINE (1966 to February week 4, 2006) and EMBASE (1980 to week 8, 2006) were searched for articles examining the effect of an exercise or lifestyle intervention on individuals with prediabetes. The search was carried out using medical search headings (MeSH) and by searching titles and abstracts for relevant words. For example, studies including individuals with prediabetes were found by using the MeSH “prediabetic state”, “insulin resistance”, “glucose intolerance”, and “diabetes mellitus” (subheading “prevention and control”) and by searching titles and abstracts for “prediabetes”, “impaired glucose tolerance”, “IGT”, “impaired fasting glucose”, and “IFG”. Studies that included an exercise intervention were found by using the MeSH “life style”, “sports”, “exercise therapy”, and “physical fitness” and by searching titles and abstracts for “exercise”, “physical activity”, “physical fitness”, “resistance training”, “strength training”, “circuit training”, “endurance training”, and “aerobic training”. In addition, the reference lists of relevant published original articles and reviews were hand-searched.

One reviewer (TY) performed the electronic and hand searches and reviewed the results. Studies that clearly did not meet the inclusion criteria were rejected during the initial review. Where uncertainty existed, the full text of the article was obtained and reviewed. Two reviewers (TY, KK) independently assessed all potentially relevant studies and performed data extraction. Disagreement was resolved by discussion and where necessary third party adjudication.

Inclusion criteria

Subjects

Participants were adults (age ≥ 18 years) diagnosed with prediabetes. Prediabetes was defined as impaired glucose tolerance (IGT) and/or impaired fasting glucose (IFG) using one of the sets of criteria previously recommended by the World Health Organization (WHO) (Alberti & Zimmet 1998, World Health Organization 1985) or the American Diabetes Association (ADA) (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 2003, Expert Committee on the Diagnosis and Classification of Diabetes
Mellitus 1997). Studies that defined IGT or IFG using other criteria were included if the mean value of the participants' plasma glucose fell within the range of IGT or IFG as defined by the WHO or ADA criteria (2-h glucose of ≥ 7.8 mmol/l and < 11.1 mmol/l and fasting glucose < 7.8 mmol/l for IGT; fasting glucose, ≥ 5.6 mmol/l and < 7.0 mmol/l and 2-h glucose < 7.8 mmol/l for IFG).

**Interventions**

Interventions that included an exercise programme were included. “Exercise programme” was taken to mean any intervention where physical activity was actively promoted and supported or a structured exercise training regime. Studies that only provided individuals with brief written or verbal physical activity advice were excluded. Studies investigating the effect of a single or acute episode of exercise were also excluded.

**Outcome measures**

Only studies with an outcome measure of physical activity and a relevant clinical measure were included. A relevant clinical measure was defined as progression to diabetes or a suitable measure of plasma glucose (2-h glucose for IGT or fasting glucose for IFG).

**Type of study**

Randomised and non-randomised controlled trials were included.

**Analysis**

As the heterogeneity in the type of exercise interventions and outcome measures did not lend itself to quantitative methods of analysis, a systematic narrative review was undertaken. Baseline and follow-up exercise, body mass and glucose parameters were reported as mean ± standard error (SE), or median (interquartile range). Results reporting the standard deviation or the 95% confidence interval (CI) were converted to SE using the formulas $SE = SD/\sqrt{n}$ and $SE = (CI_{upper} - CI_{lower})/2 \, t$ (were $t$ is the $t$-distribution value for a 95% CI) respectively. Where SE, SD or 95% CI for the change from baseline to follow-up was not reported, only the mean value is reported due to the potential error involved in imputing SE for this figure. When available, the relative risk of diabetes in the intervention group compared with the control group was also reported. Significance was reported at the level of $p = 0.05$, unless otherwise stated.
Results

The search produced 307 articles, from which 279 potential studies were identified, of these, eight trials met the inclusion criteria (see Figure 2.1). Study details for the eight included studies are shown in Appendix Two; the main outcomes are presented in Appendix Three.

Study design

Seven of the eight trials involved randomisation of subjects to a treatment group or control group (Carr et al. 2005, Knowler et al. 2002, Lindahl et al. 1999, Lindström et al. 2003, Mensink et al. 2003a, Oldroyd et al. 2006, Pan et al. 1997). The non-randomised trial identified control participants by using individuals who, for various (un-stated) reasons, were not enrolled in the intervention programme (Eriksson & Lindgärde 1991).

Sample size

Sample size ranged from 62 to 2161. Two studies reported a power calculation based on the expected difference in incidence of diabetes between groups (Knowler et al. 2002, Lindström J et al. 2003) and one reported a power calculation based on the expected difference between groups in the proportion of individuals with IGT at the end of the study (Oldroyd et al. 2006). In the latter case, Oldroyd et al. calculated that a total of 100 participants were required to detect a 0.6 mmol/l difference in fasting glucose and a 20% difference in the number of individuals with IGT, allowing for a 90% power at a significance of 0.05. Three studies had sample sizes of fewer than 100 participants at follow-up (Carr et al. 2005, Mensink et al. 2003a, Oldroyd et al. 2006).
Figure 2.1: Flow diagram of literature search

Medline (n=109) → Embase (n=196) → Hand search (n=2) → Potentially relevant articles (n=279)

Articles excluded (n=221):
- Studies not published in English (n=28)
- Not a controlled trial (n=152)
- Not a relevant cohort (n=28)
- Studies that did not include an intervention with an exercise programme (n=13)

Intervention trials that included an exercise component in individuals with prediabetes (n=58)

Studies that did not include a relevant outcome measure (n=16)

Duplicates (n=34)

Intervention trials to meet the inclusion criteria (n=8)
Subjects

All studies examined in this review included individuals with IGT and excluded those with isolated IFG.

Gender

Except for one trial that involved only men (Eriksson & Lindgärde 1991), all trials included both men and women. Within the included studies, 40% of participants were male.

Intervention conditions


Six of the lifestyle intervention studies were based on encouraging individuals to increase their physical activity to approximately 150 minutes of moderate to vigorous intensity exercise per week whilst also encouraging weight loss through a healthy hypo-calorific diet (Eriksson & Lindgärde 1991, Knowler et al. 2002, Lindström et al. 2003, Mensink et al. 2003a, Oldroyd et al. 2006, Pan et al. 1997). Participants in all six of these studies received regular encouragement and counselling from a trained dietician at least once every three months throughout the duration of the intervention. Two of the six studies also provided participants with the option of attending supervised exercise classes for some or all of the study duration (Eriksson & Lindgärde 1991, Lindström et al. 2003) and one provided discounted access to local gyms (Oldroyd et al. 2006). One study determined the effect of diet and exercise separately and in combination (Pan et al. 1997).

One lifestyle intervention included an initial one-month stay at a wellness centre where individuals were provided with healthy dietary options and encouraged to take part in 2.5 hours per day of light- to moderate-intensity exercise using the provided leisure facilities (Lindahl et al. 1999). After the stay at the wellness centre, participants were encouraged to
make plans detailing how they could incorporate healthier habits into everyday life and then received no further contact until follow-up.

The structured exercise intervention study used a training protocol of 180 minutes per week of aerobic exercise at 70% of heart rate reserve (Carr et al. 2005). Exercise training was supervised for the first six months and both groups were encouraged to eat a healthy isocalorific diet, with those in the exercise training group also being encouraged to eat a diet high in carbohydrate.

Outcomes

Four studies included incidence of diabetes as the main outcome (Eriksson, & Lindgärde 1991, Knowler et al. 2002, Lindström et al. 2003, Pan et al. 1997) and four used 2-h glucose levels as a direct measure of glucose control (Carr et al. 2005, Lindahl et al. 1999, Mensink et al. 2003a, Oldroyd et al. 2006). All the studies using incidence of diabetes as their main outcome were based on a multi-component lifestyle intervention (see Intervention conditions above).

Incidence of diabetes and physical activity

All four of the intervention studies that measured incidence of diabetes as their primary outcome found a significant reduction in the incidence of type 2 diabetes in the intervention group. Diabetes incidence was reduced by 42-63% compared to the control group (see Appendix Three). The study that looked at the effect of diet and physical activity separately and in combination found a greater reduction in the incidence of diabetes (46% reduced risk) in the physical activity-only group than in either the combined physical activity and diet group (42% reduced risk) or the diet-only group (31% reduced risk), although the difference between groups was not statistically significant (Pan et al. 1997). Three out of these four studies relied on self-reported measures of physical activity (Knowler et al. 2002, Lindström et al. 2003, Pan et al. 1997); validity was not reported for one study (Pan et al. 1997). All three of the studies relying on self-reported physical activity levels reported non-significant or small changes in physical activity levels. For example, the Diabetes Prevention Program (DPP) reported a mean increase in energy expenditure due to leisure time physical activity of around 6 MET-h/week (Knowler et al. 2002), which is
roughly equivalent to walking at a moderate pace for 15 minutes per day (Ainsworth et al. 2000). The Finnish Diabetes Prevention Study (FDPS) reported no significant change in total physical activity levels compared to the control group and a 9 minute/day increase in moderate- to vigorous-intensity physical activity (Lindström et al. 2003), and the Da Qing IGT and Diabetes Study reported no significant change in physical activity levels compared to the control group (Pan et al. 1997). The Malmo Feasibility Study, which used an objective outcome measure (cardiovascular fitness), reported an 8% increase in maximal oxygen uptake (Eriksson & Lindgärde 1991).

2-hour post-challenge plasma glucose and physical activity

Three of the studies that used incidence of diabetes as their primary outcome measure also measured pre- and post- 2-h glucose (Eriksson & Lindgärde 1991, Lindström et al. 2003, Pan et al. 1997). FDPS reported a 0.9 mmol/l decrease in 2-h glucose after one year, but no significant change after three years (Lindström et al. 2003); the Da Qing IGT and Diabetes Study found that 2-h glucose increased in all groups, but the increase in the control group was over twice that in either of the intervention groups (Pan et al. 1997); and the Malmö Feasibility Study reported a 1.1mmoU1 reduction in 2-h glucose in the intervention group (Eriksson & Lindgärde 1991). Of the three lifestyle intervention studies that used 2-h glucose levels rather than incidence of diabetes as primary indicator of improved glucose tolerance (Lindahl et al. 1999, Mensink et al. 2003a, Oldroyd et al. 2006), only one (Mensink et al. 2003a) reported a significant difference between groups in 2-h glucose at follow-up. Two of these studies used a measure of cardiovascular fitness as an indicator of physical activity levels (Lindahl et al. 1999, Mensink et al. 2003a) and one (Oldroyd et al. 2006) used distance walked in a shuttle test (Singh et al. 1992) as a measure of physical activity. Two studies found a small to moderate increase in cardiovascular fitness (< 10% increase on baseline value) (Lindahl et al. 1999, Mensink et al. 2003a) and the study using the shuttle test reported no change in distance walked during the test (Oldroyd et al. 2006). Similarly, the moderate increases in cardiovascular fitness observed in the structured exercise training study were not associated with significant improvements in 2-h glucose compared to the control group (Carr et al. 2005).
**Fasting glucose**

None of the included studies reported a significant change in fasting glucose in the intervention group compared to the control group at follow-up. One study did not report fasting glucose values (Eriksson & Lindgärde 1991).

**Body weight**

All studies reported a significant reduction in body weight in the intervention group of between 1.8 to 5.6 kg.
Discussion

Eight controlled trials in individuals with impaired glucose tolerance were included in this review. Four studies measured incidence of diabetes as a primary outcome measure and found that the risk of diabetes was reduced by approximately 50% (range 42 - 63%) in individuals who were encouraged to reduce their body mass through dietary and physical activity change (Eriksson & Lindgärde 1991, Knowler et al. 2002, Lindström et al. 2003, Pan et al. 1997). Although physical activity promotion was an important component of these studies, the effect of exercise independent of other factors on the risk of diabetes in individuals with IGT is still unclear. All but one (Pan et al. 1997) of the studies included in this review reported significant weight loss among participants. As weight loss is known to improve many of the factors associated with IGT, including insulin sensitivity and glycaemic control (American Diabetes Association 2002), and considering only modest increases in physical activity were found in these studies, the success of these interventions are likely to have been largely attributable to weight loss. The apparent success of the exercise-only intervention in the Da Qing IGT and Diabetes Study does not necessarily contradict this conclusion as it is likely that the significantly higher levels of physical activity at baseline in the exercise intervention group compared with the control group introduced an important source of confounding (Pan et al. 1997).

The separation of physical activity and weight loss may seem an overcorrection given that increased physical activity may encourage weight loss through increased energy expenditure, however two meta-analyses of controlled trials in individuals with type 2 diabetes found that exercise training was not associated with weight loss (Boulé et al. 2001, Thomas et al. 2006). Even though it has been argued that the negative results from these studies could be due to the fact that there are physiological reasons why individuals with type 2 diabetes may be less responsive to weight-loss interventions (Laaksonen et al. 2007), it is increasingly recognized that at least 60 minutes per day of moderate intensity exercise should be undertaken for the effective management of body fat (Brooks et al. 2004, Gill & Malkova 2006), an amount that none of the interventions included in this review achieved.

Three out of the four studies that investigated the effect of a lifestyle intervention in individuals with IGT on incidence of type 2 diabetes (Knowler et al. 2002, Lindström et al.
2003, Pan et al. 1997) relied on self-reported measures of physical activity. Given the
limitations of subjective measures of physical activity, especially for detecting change in
non-structured forms of moderate physical activity such as walking activity (Cooper 2003),
these lifestyle intervention studies provide uncertain information about the effect of physical
activity in individuals with IGT.

Results from the lifestyle intervention studies that relied on changes in 2-h glucose, rather
than incidence of diabetes as the primary measure of glucose control, were inconclusive.
Two out of three of these studies were unsuccessful at improving glucose tolerance (Lindahl
et al. 1999, Oldroyd et al. 2006). Similarly, the one study that used an aerobic exercise
training protocol found no improvements in glucose tolerance as measured by 2-h glucose
(Carr et al. 2005). However, it did find a significant improvement in insulin sensitivity at
both 6 and 24 months. This suggests that although the intervention goal of 3 hours per week
of moderate intensity exercise was enough to improve insulin sensitivity it was not great
enough in volume and/or intensity to elicit the necessary magnitude of change in insulin
sensitivity for this to be translated into a significant reduction in 2-h glucose.

Overall, non-significant results were seen in all but two of the studies that measured pre- and
post - 2-h glucose. However despite the link between 2-h glucose and diabetes risk, it does
not follow that the risk of diabetes was unchanged in these studies, as demonstrated by FDPS
which reported a non-significant change in 2-h glucose over the course of the intervention
but a greater than 50% reduction in the risk of diabetes (Lindström et al. 2003). One reason
for this discrepancy is likely to be the poor repeatability of 2-h glucose values (Ko et al.
1998), which along with the relatively small sample sizes in most of these studies makes it
possible that improvements in glucose tolerance were not detected using 2-h glucose. The
glucose area under the curve has been identified as a more reliable measure of glucose
tolerance than 2-h glucose (Feskens et al. 1991) and is therefore a more sensitive measure of
glucose tolerance. One study included in this review measured both 2-h glucose and glucose
area under the curve at baseline and follow-up (Carr et al. 2005). It found that although 2-h
glucose did not change significantly at any of the follow-up time points there was a
significant reduction in the glucose area under the curve at six months and a trend towards a
significance at 24 months.
Given the failure of the lifestyle interventions to substantially increase physical activity levels and the inconclusive result of the structured exercise training study, the role of physical activity independent of other lifestyle changes in the treatment of prediabetes remains equivocal. Statistical analysis of the independent effects of physical activity change, which has been carried out on some of the lifestyle intervention studies included in this review, also gives equivocal results. For example, the conclusion of the Malmo Feasibility Study that cardiovascular fitness and weight loss were equally correlated to improved glucose tolerance is supported by data from the Study on Lifestyle Intervention and Impaired Glucose Tolerance Maastricht (SLIM) (Mensink et al. 2003b) and a recent analysis of data from FDPS found a 49% difference in the risk of diabetes, after adjustment for changes to body mass and diet, when comparing those in the highest and lowest tertiles of moderate- to vigorous-intensity leisure time physical activity change (Laaksonen et al. 2005). In contrast, analysis of data from DPP did not find an independent association between physical activity change and the incidence of type 2 diabetes (Hamman et al. 2006).

Given the limitations of the studies included in this review it is not possible to make any recommendations as to the intensity and duration of exercise needed to improve glucose tolerance and/or reduce the risk of diabetes in individuals with IGT independent of other lifestyle changes. The equivocal nature of the evidence is reflected in the advice given by ADA, which recommends that individuals with IGT should include 150 minutes per week of moderate- to vigorous-intensity exercise as part of a weight management programme (Sigal et al. 2006). However, the aforementioned analysis of physical activity change in FDPS found that a 246 minutes per week difference in median values between those in the lowest and the highest tertiles of moderate- to vigorous-intensity physical activity was associated with a significant reduction in the risk of diabetes, after adjusting for changes in diet and body mass. However, the difference of 120 minutes per week in median values between the lowest and middle tertiles was not associated with a reduced risk of diabetes (Laaksonen et al. 2005). Although this result was gained by analysing the pooled cohort and so provides little information about the effectiveness of the intervention itself, it does suggest that 150 minutes per week of moderate to vigorous intensity exercise, independent of other lifestyle changes, is unlikely to be enough to significantly reduce the risk of type 2 diabetes in individuals with IGT. This conclusion is based on results for physical activity behaviour change and is in contrast to cross-sectional and epidemiological data which has tended to show that 150 minutes of moderate- to vigorous-intensity physical activity is associated with
a decrease in the relative risk of developing type 2 diabetes (Bassuk & Manson 2005), suggesting the presence of unknown or unmeasured confounding factors in these studies. However, given that the vast majority of the evidence to date comes from self-reported physical activity levels, further rigorously designed studies with objective measures of physical activity are needed.

All studies included in this review selected individuals using IGT as an inclusion criteria. Therefore, any conclusions from this review can only be applied to individuals with IGT and it is impossible to determine whether or not exercise may be effective in treating individuals with isolated IFG. However as individuals with isolated IFG account for a minority of individuals with prediabetes (Benjamin et al. 2003), conclusions about the effect of exercise on IGT drawn from this review will apply to the majority of individuals with prediabetes.

In summary, the majority of studies found for this review used interventions that encouraged dietary change and physical activity in order to initiate and maintain weight loss in individuals with IGT. Analysis of these studies found that the independent effect of physical activity in reducing the risk of type 2 diabetes in individuals with prediabetes is equivocal. Furthermore, given the limited evidence, no definite conclusion can be drawn as to the amount of physical activity needed to reduce the risk of diabetes in individuals with prediabetes or the effectiveness of a single component physical activity intervention in comparison to more conventional multi-component interventions.

Therefore, more evidence from rigorously designed randomised controlled trials with objective measures of physical activity is needed. As the majority of studies promoting lifestyle changes included in this review failed to substantially increase physical activity levels, strategies for effecting increased physical activity in this population also need to be researched thoroughly. This is particularly important as single-factor physical activity interventions have been shown to be more effective at promoting physical activity in primary care (Hillsdon et al. 2005b) and improving glycaemic control in individuals with type 2 diabetes (Conn et al. 2007). Therefore it is essential that single-factor physical activity interventions are developed and robustly tested before they are integrated into more traditional multi-factor diabetes prevention programmes.
Chapter Summary

Previous diabetes prevention initiatives have failed to demonstrate that they are effective at initiating clinically significant increases in physical activity, therefore the evidence for the effectiveness of physical activity, independent of dietary or weight loss change, in the prevention of type 2 diabetes in individuals with prediabetes is equivocal.
Chapter Three

Walking and markers of chronic low-grade inflammation
This chapter will investigate whether walking activity is associated with markers of chronic low-grade inflammation in individuals who have been screened for type 2 diabetes. Specifically, it will investigate whether achieving the current minimum recommendations for physical activity through walking activity is associated with lower levels of circulating interleukin-6, tumour necrosis factor-α, and C-reactive protein, independently of other forms of physical activity. By investigating the independent effect of walking on novel biomarkers linked to the development of metabolic dysfunction, this chapter will complement other epidemiological studies that have shown that walking for 150 minutes per week is associated with a significant reduction in the risk of developing type 2 diabetes. This chapter will help to quantify the importance of including markers of chronic low-grade inflammation as outcome variables in future walking intervention studies.

The work described in this chapter was the result of secondary analysis of data that was collected as part of the Leicester arm of the ADDITION study. The ADDITION study is a Europe-wide study designed to test the cost-effectiveness of screening for type 2 diabetes in the general population.

The findings of this Chapter were published in Preventive Medicine (Yates et al. 2008a).
Background

Although overweight and obesity are strongly associated with insulin resistance and the risk of developing type 2 diabetes, a mechanistic pathway linking overweight and obesity to insulin resistance and metabolic dysfunction in humans has remained elusive (Telford 2007). However, the discovery over a decade ago that adipose tissue is an important endocrine organ which releases proteins, called adipocytokines, that exert a widespread influence on human physiology, suggested the possibility that these proteins might directly link adipose tissue to metabolic dysfunction. In particular, it was discovered that adipose tissue releases the cytokines interleukin-6 (IL-6) and tumour necrosis factor-α (TNFα), which play a central role in the innate immune system. The innate immune system is activated in response to stressors such as infection and tissue injury; this activation causes a number of cells, including adipocytes, to secrete TNFα and IL-6, which in turn activate the liver to synthesize acute phase proteins, such as C-reactive protein (CRP). It has been hypothesised that chronic stressors associated with obesity, such as overnutrition, may cause a chronic activation of the innate immune system, resulting in an overload of the system’s resources leading to deleterious metabolic effects; therefore type 2 diabetes may in fact be a disease of the innate immune system (Pickup & Crook 1998). Subsequent research, both from mechanistic and epidemiology studies, have added weight to this hypothesis by consistently showing that type 2 diabetes is characterized by elevated levels of pro-inflammatory adipocytokines and that these factors are directly linked to the development of metabolic and vascular dysfunction (Tataranni and Ortega 2005).

Along with obesity, it has also been shown that levels of moderate- to vigorous-intensity physical activity levels are independently and inversely associated with markers of chronic low-grade inflammation, including IL-6, tumour TNFα and C-reactive protein (CRP) (Panagiotakos et al. 2005, Pischon et al. 2003). Markers of chronic low-grade inflammatory could therefore also be an important mediating link between physical activity and metabolic health, independent of adiposity.

Although walking has been shown to be associated with a reduced risk of developing diabetes and cardiovascular disease (Hu et al. 1999, Laaksonen et al. 2005, Manson et al. 2002, Tanasescu et al. 2002), when carried out at levels that are consistent with the current exercise recommendations (Department of Health 2004b, Haskell et al. 2007), the effect of walking on pro-inflammatory adipocytokines, independent of more vigorous forms of...
exercise, is not well documented. This is an important limitation considering walking has been shown to be the preferred choice of physical activity in the majority of the population in industrialized countries and is therefore likely to be the most effective mode of physical activity to promote in intervention studies (Booth et al. 1997, Crespo et al. 1996, Vaz de Almeida et al. 1999). For further discussion of the importance of walking activity in the context of this thesis see Chapter Four.

The aim of this Chapter was therefore to investigate the effect of walking on the pro-inflammatory adipocytokines IL-6 and TNFα, along with CRP and insulin, in an ethnically diverse population screened for type 2 diabetes. The primary hypothesis was that walking at levels that are consistent with the current exercise recommendations would be independently associated with reduced chronic low-grade inflammation.
Methods

Participants
The ADDITION study is a Europe-wide screening and treatment programme for type 2 diabetes. In the United Kingdom, the Leicester ADDITION study has undertaken a population-based screening programme based on the oral glucose tolerance test (Sandbaek et al. 2008). The inclusion criterion for screening was age between 40 and 75 years for White Europeans or between 25 and 75 years for those from a Black and Minority Ethnic group. Between 2005 and 2006, 573 (m = 304, f = 269) individuals screened as part of the ADDITION study also consented for a sub-study for which additional blood samples were taken for the analysis of inflammatory markers. The average age of the participants was 62 ± 9 years and 24% were from a South Asian ethnic background. Using American Diabetes Association criteria (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 1997), 287 (50%) were identified with normal glucose control, 242 (42%) with prediabetes and 44 (8%) with diabetes.

Measures

Physical activity
Physical activity was measured using the short last-seven-days self-administered format of the International Physical Activity Questionnaire (IPAQ) (Craig et al. 2003). IPAQ measures the frequency and duration of any moderate- to vigorous-intensity physical activity undertaken for more than 10 continuous minutes across all contexts (e.g. work, home and leisure) over a seven-day period. Importantly, IPAQ distinguishes walking activity from other forms of moderate- to vigorous-intensity physical activity across all contexts. It also enables the calculation of metabolic equivalents (MET-hours/week). The IPAQ questionnaire has been shown to correlate reasonably ($\rho = 0.4$) with accelerometer data in the United Kingdom (Craig et al. 2003). Walking activity categories were formed by distinguishing between those who reported walking for at least 30 minutes on at least five days per week and those who reported walking for less than this.
Biochemical, clinical and demographic measurements

All assays were measured by individuals blinded to the patients identity. Plasma glucose was measured using a glucose oxidase method on the Beckman Auto Analyzer (Beckman, High Wycombe, UK). C-reactive protein was analysed on an ABX Pentra clinical chemistry analyser using a latex-enhanced immunoturbidimetric assay, which has a lower sensitivity of 0.1mg/l (Horiba Group, Montpellier, France). TNFα and IL-6 were analysed using quantikine high-sensitivity enzyme linked immunosorbant assays (ELISA) (R&D Systems, Abingdon, UK). Insulin was analysed using a Perkin Elmer time-resolved fluoro-immuno assay on the AutoDELFIA. The intraassay and interassay coefficients of variation for the included assays did not exceed 10%, apart from TNFα, which had a maximum interassay coefficient of variation of 16.7%.

Arterial blood pressure was measured in the sitting position (Omron, Healthcare, Henfield, UK); three measurements were obtained and the average of the last two measurements was used. Body weight (Tanita TBE 611, Tanita, West Drayton, UK) and height were also measured.

Current smoking status and medication history were obtained by an interview-administered protocol. For the purposes of this study participants were defined as non-smokers, past smokers, or current smokers (≥ 1 cigarette per day). Blood pressure and statin medication status was defined by whether or not participants were currently taking these medications.

Social deprivation was determined by assigning an Index of Multiple Deprivation (IMD) score to each participants postcode (Office for the Deputy Prime Minister 2004). IMDs are publicly available continuous measures of compound social and material deprivation, and are calculated using a variety of data including current income, employment, health, education, and housing.

Statistical analysis

Variables are presented as mean value ± standard deviation. Chi-squared tests were used to analyse categorical variables, independent t-tests were used to analyse normally distributed continuous variables and Mann-Whitney tests were used to analyse non-parametric variables.
Multivariate analysis of variance was used to analyse the associations between walking activity categories and CRP, IL-6, TNFα, and insulin. Due to their skewed distribution, all dependant variables in the multivariate analysis were log-transformed. Multivariate analysis models were adjusted for non-walking physical activity levels and measured demographic variables (age, sex, ethnicity and social deprivation). Further adjustment was carried out for medication and smoking status if the inclusion of these factors as covariates in the multivariate analysis changed the coefficient for walking status by 10% or more for any of the included dependant variables (Maldonado & Greenland 1993). In addition, further adjustment was also made for waist circumference in order to establish the independent effect of walking activity status. All analyses were two sided; p <0.05 was considered significant. Analysis was carried out on SPSS 12.0 for Windows (SPSS, Chicago, USA).
Results

Complete physical activity data was available for 400 (70%) participants. Of these participants, 142 (36%) had prediabetes, 33 (8%) had diabetes and 15 (4%) had a previous history of myocardial infarction, stroke or angina. Those that completed the questionnaire were more likely to have normal glucose control and were more likely to come from less deprived areas than non-completers; no significant differences in inflammatory markers, age or ethnicity was observed between completers and non-completers. Data is reported for the subset of 400 participants who completed the physical activity questionnaire.

There was no significant difference between high and low walking activity groups in the incidence of diabetes, prediabetes or those with a history of myocardial infarction, stroke or angina. Table 3.1 shows the characteristics of the study participants overall and according to their walking status. Compared to White Europeans, those from a South Asian ethnic background were less likely to report walking for 30 minutes on at least 5 days a week.

Multivariate statistical analysis found that those who reported walking for at least 30 minutes on at least 5 days per week had lower IL-6, CRP and TNFα levels compared to those who reported lower walking activity, after adjustment for other modes of moderate- to vigorous-intensity physical activity, age, ethnicity, sex, social deprivation and smoking status (see Figure 3.1). Further adjustment for waist circumference attenuated the association of walking with TNFα, although the association with IL-6 and CRP remained significant. There was no association between fasting insulin and walking status. All results were unaffected by the inclusion of statin or blood pressure medication status as covariates in the statistical analysis.
Table 3.1: Characteristics of study participants across activity categories.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample (n=400)</th>
<th>Low walking activity (n=191)</th>
<th>High walking activity (n=209)</th>
<th>P for difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking activity (MET-hours /week)</td>
<td>15.4 [3.3 to 46.2]</td>
<td>3.3 [0 to 9.6]</td>
<td>35.8 [17.3 to 69.3]</td>
<td></td>
</tr>
<tr>
<td>Moderate- to vigorous-intensity physical activity (excluding walking activity) (MET-hours /week)</td>
<td>0 [0 to 30.6]</td>
<td>0 [0 to 8]</td>
<td>8.2 [0 to 44]</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>209 (52)</td>
<td>93 (49)</td>
<td>116 (55)</td>
<td>0.17</td>
</tr>
<tr>
<td>Female</td>
<td>191 (48)</td>
<td>98 (51)</td>
<td>93 (45)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White European</td>
<td>307 (77)</td>
<td>135 (71)</td>
<td>172 (82)</td>
<td>0.01</td>
</tr>
<tr>
<td>South Asian</td>
<td>93 (23)</td>
<td>56 (29)</td>
<td>37 (18)</td>
<td></td>
</tr>
<tr>
<td>Index of Multiple Deprivation score</td>
<td>19.2 [11.3 to 29.1]</td>
<td>18.6 [10.6 to 28.9]</td>
<td>21.0 [11.7 to 29.9]</td>
<td>0.23</td>
</tr>
<tr>
<td>Age (years)</td>
<td>61.8 ± 9.1</td>
<td>62.2 ± 9.0</td>
<td>61.4 ± 9.1</td>
<td>0.64</td>
</tr>
<tr>
<td>Blood Pressure Medication</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td></td>
<td></td>
<td></td>
<td>0.89 (medication)</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitors</td>
<td>52 (13)</td>
<td>27 (14)</td>
<td>25 (12)</td>
<td>vs. no medication</td>
</tr>
<tr>
<td>Statin medication</td>
<td>59 (14.8)</td>
<td>32 (16.8)</td>
<td>27 (12.9)</td>
<td>0.27</td>
</tr>
<tr>
<td>Systolic Blood Pressure (mmHg)</td>
<td>143 ± 21</td>
<td>143 ± 22</td>
<td>142 ± 19</td>
<td>0.83</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>86 ± 11</td>
<td>87 ± 12</td>
<td>85 ± 11</td>
<td>0.64</td>
</tr>
<tr>
<td>Current smokers</td>
<td>45 (11)</td>
<td>16 (8)</td>
<td>29 (14)</td>
<td>0.08</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29.1 ± 4.6</td>
<td>29.4 ± 4.4</td>
<td>29.3 ± 4.6</td>
<td>0.91</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>98.7 ± 12.1</td>
<td>98.8 ± 12.7</td>
<td>98.5 ± 11.3</td>
<td>0.79</td>
</tr>
</tbody>
</table>
Table 3.1 continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample (n=400)</th>
<th>Low walking activity (n=191)</th>
<th>High walking activity (n=209)</th>
<th>P for difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting insulin (uIU/ml)</td>
<td>7.6 [5.0 to 11.1]</td>
<td>7.9 [5.6 to 11.1]</td>
<td>7.2 [4.8 to 10.6]</td>
<td>0.05</td>
</tr>
<tr>
<td>Tumor necrosis factor-α (pg/ml)</td>
<td>1.9 [1.2 to 2.6]</td>
<td>2.1 [1.4 to 2.7]</td>
<td>1.7 [1.2 to 2.5]</td>
<td>0.01</td>
</tr>
<tr>
<td>Interleukin-6 (pg/ml)</td>
<td>1.9 [1.3 to 3.1]</td>
<td>2.2 [1.3 to 3.8]</td>
<td>1.8 [1.3 to 2.7]</td>
<td>0.01</td>
</tr>
<tr>
<td>High-sensitivity C-reactive protein (mg/l)</td>
<td>2.3 [0.7 to 5.5]</td>
<td>2.3 [0.4 to 5.6]</td>
<td>2.3 [0.6 to 5.5]</td>
<td>0.16</td>
</tr>
</tbody>
</table>

Categorical results as number (column percentage), continuous parametric results as mean ± SD and continuous non-parametric results as median [interquartile range]
Figure 3.1: Association of walking status with circulating interleukin-6 (IL-6), tumor necrosis factor-α (TNFα), high sensitivity C-reactive protein (CRP) and fasting insulin

![Graphs showing association of IL-6, CRP, TNFα, and Insulin with walking status.](image)

- ■ = low walking activity, □ = high walking activity. Graphs show geometric means and the error bars indicate the standard error. Model 1: adjusted for MET-hours/week from other modes of moderate to vigorous physical activity, age, ethnicity, sex, social deprivation and current smoking status. Model 2: adjusted for the above covariates plus waist circumference.
Discussion

In this cross-sectional study of individuals screened for type 2 diabetes, walking on at least five days per week for at least 30 minutes per day was associated with lower circulating IL-6, TNFα and CRP levels after adjustment for other modes of physical activity, demographic variables and smoking status. Further adjustment for waist circumference attenuated the association of walking categories with TNFα, although the association with IL-6 and CRP remained significant.

Although other studies have shown that walking for around 150 minutes per week is associated with a reduced risk of developing type 2 diabetes and cardiovascular disease (Hu et al. 1999, Laaksonen et al. 2005, Manson et al. 2002), to my knowledge this is the first study to investigate the effect of walking on inflammatory markers after adjustment for other forms of physical activity. As chronic low-grade inflammation is thought to play an important role in the pathogenesis of type 2 diabetes and cardiovascular disease (Libby et al. 2002, Pickup & Crook 1998, Tataranni and Ortega 2005, Xu et al. 2003), this study suggests that increased walking activity may reduce the risk of developing a debilitating chronic disease through reduced systemic inflammation. This is clinically important as, for the majority of individuals, walking is the most accessible form of physical activity. The findings reported in this study are consistent with other cross-sectional studies which have shown that overall levels of moderate- to vigorous-intensity physical activity are inversely associated with markers of chronic low-grade inflammation (Panagiotakos et al. 2005, Pischon et al. 2003, Wannamethee et al. 2002). However, few intervention studies have investigated the effect of walking on markers of chronic low-grade inflammation, those that have are underpowered and have produced conflicting results (Dekker et al. 2007, Giannopoulou et al. 2005, Marcell et al. 2005, Wegge et al. 2004).

This study further emphasises the clinical importance of promoting walking activity to levels that are consistent with the current physical activity recommendations in sedentary populations, particularly in those identified with an increased risk of developing a chronic disease. However, as Chapter Two points out, there is little
evidence that traditional methods of promoting health behaviour change in at-risk populations, such as previous diabetes prevention programmes, have been successful at promoting clinically significant increases in physical activity. Therefore there is a continuing need to develop and test innovative strategies for promoting physical activity, in particular walking activity, in health care and community settings.

The exact mechanisms linking physical activity to reduced inflammation have not been well described. However several studies have shown that exercise training does not affect cytokine production from adipose tissue (Klimcakova et al. 2006, Polak et al. 2006), although it may alter cytokine production from mononuclear cells, another important source of elevated cytokine levels (Smith et al. 1999). It has been hypothesised that the release of myokines (cytokines released from muscle, such as IL-6) from exercising muscle may, when performed regularly, cause adaptations to the innate immune system resulting in lower levels of cytokines being released from mononuclear cells (You & Nicklas 2008). However, as the interactive effects of exercise, muscle, body fat, and markers of chronic low-grade inflammation in the development of metabolic and vascular dysfunction are poorly defined (Telford 2007), more research is needed to quantify the overall significance of the findings from this study.

The finding that walking activity status was not associated with fasting insulin levels is in contrast to other studies that have shown an inverse association between moderate- to vigorous-intensity physical activity and fasting insulin levels (Irwin et al. 2000, Mayer-Davis et al. 1998). However, studies in individuals with type 2 diabetes have shown that ambulatory activity is generally accumulated at an intensity lower that that needed for health benefits and that exercise intensity, but not exercise volume, predict glycaemic control (Boulé et al. 2003, Johnson et al. 2005). Therefore, given that almost half of the participants in this study had prediabetes or diabetes it is possible that participants accumulated their walking activity at an intensity lower than that needed to improve insulin sensitivity.

This study also found that those from a South Asian Ethnic background were less likely than White Europeans to report walking at levels that are consistent with the current physical activity recommendations. This confirms findings from other studies
in the United Kingdom that have consistently shown that overall physical activity levels are lower in South Asians than White Europeans (Fischbacher et al. 2004). As walking is likely to be associated with fewer barriers than other forms of physical activity in a South Asian migrant population (Johnson 2000), this study emphasises the need to investigate effective, culturally acceptable, ways of promoting walking activity in ethnic minorities. This is all the more necessary considering migrant South Asians and their descendants have been shown to have a higher risk of developing type 2 diabetes and cardiovascular disease and have higher levels of circulating markers of chronic low-grade inflammation compared to White Europeans (Barnett et al. 2006, Chambers et al. 2001).

The main limitations of this study are that it was not possible to determine causality and the high percentage of missing physical activity data. The small sample size precluded meaningful sub-group analysis, which given the heterogeneity of the study sample is another important limitation. Furthermore, although the confounding effects of some important determinants, such as smoking and medication status, were determined, it is not possible to discount the influence of other factors in this study, such as dietary status. Therefore the findings from this study need to be confirmed with appropriately powered intervention studies. Given the specific limitations associated with self-reported measures of physical activity, the use of self-reported walking activity status in this study is another potential limitation. However, it is not yet feasible to objectively and accurately measure walking activity mode and bout length in large populations; pedometer and accelerometer data typically give a measure of overall ambulatory activity which, in addition to walking, can be accumulated by many other modes of activity such as running, house work and gardening.
Chapter Summary

Meeting the current exercise recommendations through walking activity is associated with reduced levels of circulating pro-inflammatory markers, independent of other forms moderate- to vigorous-intensity physical activity, in adults screened for type 2 diabetes.
Chapter Four

Development of a physical activity intervention: the PREPARE programme
Chapters Two and Three of this thesis concluded that more research is needed into the effect of physical activity change on traditional and novel risk markers for diabetes and cardiovascular disease in at-risk individuals. Moreover, as highlighted in Chapter Two, successful physical activity interventions for individuals with an increased risk of developing type 2 diabetes are needed. The PREPARE programme, which is a theory-driven, group-based, structured education programme aimed at the promotion of physical activity in those with impaired glucose tolerance, was developed in response to this need. The PREPARE programme was developed after a careful review of health behaviour theory and of the current health care climate. This chapter will highlight the rationale for, and background to, the PREPARE programme and describe the content of the programme itself.

The chapter is divided into four parts: the first will detail the rationale for developing a structured education programme; the second will describe the theoretical underpinning of the educational programme; the third will focus on the need to promote walking and issues around the use of pedometers; and the fourth will describe the structure and content of the PREPARE programme.

Some of the themes highlighted in this chapter were published in Diabetes and Primary Care (Yates et al. 2007c) and Primary Care Diabetes (Yates et al. 2007d). In particular a detailed rationale for, and description of, the PREPARE programme was published in Patient Education and Counseling (Yates et al. 2008b).
Part One: A consideration of the current health care climate

Although physical activity has consistently been associated with a reduced risk of developing diabetes (Jeon et al. 2007), there is little evidence that traditional multi-factor diabetes prevention programmes have been successful at initiating clinically significant increases in physical activity (see Chapter Two). This is mirrored in evidence from the United Kingdom where the majority of physical activity and lifestyle intervention studies, carried out in a variety of different settings, have proven ineffective at initiating clinically significant increases in physical activity, particularly over the longer term (Dyson et al. 1997, Harland et al. 1999, Hillsdon et al 2002, Kinmonth et al. 2008). More broadly, it has also been reported that many interventions aimed at promoting physical activity make use of methods that would be difficult to deliver in usual health care practice (Hillsdon et al. 2005a), and that there is a gap between physical activity intervention research and the delivery of evidence-based practice (Dzewaltowski et al. 2004). Therefore, there is a need to develop successful physical activity interventions that are appropriate for a health care or community setting; this conclusion is also true of multi-factor diabetes prevention programmes, as it has been pointed out that such initiatives will have limited feasibility and success unless they are tailored to the specific requirements of national health care services (Davies et al. 2004, Yates et al. 2007d). In order to be suitable for implementation in a community or health care setting, interventions aimed at promoting a healthy lifestyle in at-risk populations need to take account of the current health care climate and make use of existing strategies that are already being successfully utilized for the promotion of self-management in other patient groups (Yates et al. 2007d).

Structured education has been widely advocated as a method of promoting patient self-management skills for individuals with type 2 diabetes mellitus (Department of Health 2005), and the National Institute for Health and Clinical Excellence (NICE) advises that structured education should be available to all individuals with type 2 diabetes mellitus at the time of diagnosis (National Institute of Health and Clinical Excellence 2003 and 2008). Structured education refers to group-based, patient-centred educational programmes that: have a clear philosophy; have a written curriculum that is underpinned by appropriate learning and health behaviour theories; are evidence based; and are delivered by trained, quality assessed, educators (Department of Health 2005). The advantage of this approach to patient care is
that it delegates responsibility for patient education away from over-stretched doctors and nurses to individuals who have been specifically trained to have responsibility for patient education. Importantly, these programmes recognize that individuals, not health professionals, should take responsibility for their health status and should therefore take an active role in gaining knowledge about their condition, choosing their behaviour goals and managing their condition (Skinner et al. 2003).

Recent evidence has shown that established structured education programmes for individuals with type 2 diabetes, such as the DESMOND, X-PERT and TURIN programmes, are successful at promoting health behaviour change and improving glycaemic control and other clinical end points (Davies et al. 2008, Deakin et al. 2006, Skinner et al. 2005, Trento et al. 2004). Whilst these programmes have been successful at initiating physical activity behaviour change (Davies et al. 2008, Deakin et al. 2006, Skinner et al. 2005), their curricula have tended to prioritize diet over physical activity in the promotion of a healthy lifestyle. For example, the DESMOND programme devotes 5% of its curriculum to physical activity, compared to over 25% for diet (The DESMOND Collaborative 2005). This suggests that structured education has not been used to its full potential in the promotion of physical activity.

Despite being advocated for numerous patient groups, structured education has not been tested as a type 2 diabetes prevention strategy and there are no national initiatives for individuals identified as having an increased risk of developing diabetes. However, the Department of Health recognizes that strategies that encourage at-risk individuals to make lifestyle changes are important (Department of Health 2005) and recent white papers have stressed the need for a stronger emphasis on preventative health care and patient empowerment (Department of Health 2004c, Department of Health 2006). Indeed, the Department of Health has recently announced plans to introduce a systematic and integrated programme for vascular disease risk assessment and management (Department of Health 2008). Given that structured education has been widely used for, and is well tolerated by, individuals diagnosed with type 2 diabetes and that it is compatible with the infrastructure of the National Health Service, it is important to test whether this approach to patient care can be utilized successfully to promote clinically meaningful changes in physical activity in at-risk populations (Yates et al. 2007c).
Another important factor in favour of group-based structured education is that it may be more cost-effective than one-to-one counselling strategies (Heller et al. 1988, National Institute for Clinical Excellence 2003, Rickheim et al. 2002). This is an important issue as diabetes prevention programmes have traditionally utilized one-to-one counselling strategies which may not be cost-effective in a “real world” UK or European primary health care setting (Icks et al. 2007, Palmer et al. 2004, Lauritzen et al. 2007).

These considerations guided the development of the PREPARE structured education programme for individuals with impaired glucose tolerance, which is described in Part Four. However, it is crucial that structured education programmes and physical activity interventions are based on known learning techniques and health behaviour theory, as the Department of Health and others have emphasized (Department of Health 2005, Michie & Abraham 2004). Consequently, Part Two of this chapter will give a brief background to and rationale for the key theoretical models that were used to guide the development and content of the PREPARE programme.

**Section summary:** As structured education programmes have been widely used for individuals diagnosed with type 2 diabetes and have been proven to be compatible with the infrastructure of the National Health Service, it is important to test whether this approach to patient care can be utilized successfully to promote physical activity in individuals identified with a high risk of developing type 2 diabetes.
Part Two: A consideration of health behaviour theory

In order to maximize behaviour change, it is important that health behaviour change interventions, including structured education programmes, are based on known learning techniques and health behaviour theory (Department of Health 2005). However, given that there are more than 20 contending health behaviour theories to choose from (Michie et al. 2005), deciding on an appropriate theory on which to ground an intervention is no easy task. The difficulty is compounded by a lack of empirical evidence for the efficacy of many health behaviour theories at effecting behaviour change, which means that there is often no sound basis for choosing one particular health behaviour theory over another (Brug et al. 2005, Michie et al. 2005, Rothman 2004). A good example of this is the popular transtheoretical model (TTM). Despite being widely advocated and used there is little evidence that TTM is successful at effecting or predicting physical activity behaviour change (Brug et al. 2005, Sutton 2000, van Sluijs et al. 2004). It has also been pointed out that, given its complexity, TTM may be inappropriate for use by practitioners and health promoters (Adams & White 2005).

In order to select appropriate health behaviour theories on which to base an educational programme aimed at promoting physical activity, the core processes proposed by Bartholomew's intervention mapping were used in the development of the PREPARE programme (Bartholomew et al. 2001). Intervention mapping is an ecological and systematic approach to developing health education programmes that provides the interventionist with a useful and coherent method for identifying which theoretical determinants are likely to be important in the development of their intervention (Bartholomew et al. 2001). This approach ensures that empirical evidence is used to confirm or reject a broad range of potentially useful theoretical domains that are not necessarily confined to a particular theory or theories, thus ensuring that the interventionist arrives at a comprehensive set of domains that are likely to be important in the promotion of a given health behaviour. Figure 4.1 shows the core steps proposed by intervention mapping and how this approach was adapted for use in this study. The key finding from this process was that, although various theoretical approaches have been used in the promotion of physical activity, successful physical activity and multi-factor intervention programmes in individuals with IGT and diabetes, regardless of their theoretical underpinning, have consistently utilized methods that are central to Bandura's social cognitive theory (Di Loreto et al. 2003, Kirk et al. 2004, Knowler et al. 2002, Lindström J et
al. 2003, Mensink et al. 2003a). As a result the development of the PREPARE programme was primarily based on social cognitive theory. However, as highlighted in Figure 4.1, Leventhal's common sense model (Leventhal et al. 1980), Gollwitzer's implementation intention (Gollwitzer 1999) and Chaiken's dual process theory (Chaiken 1987) were also identified as relevant and instructive theories in the promotion of physical activity in an at-risk population in a health care setting. These theories were identified as having constructs that were likely to be of importance over and above those associated with social cognitive theory. A brief overview and discussion of each theoretical construct on which the PREPARE programme is based is given in the sections below.
Figure 4.1: Intervention mapping

**Core processes for intervention mapping (Bartholomew et al. 2001)**

1. Pose the question
2. Brainstorm a list of determinants that could help answer the question
3. Go back to the literature and find evidence to support, refute or add to the list of determinants
4. Go back to the literature related to the topic and look for theoretical explanations for the determinants listed
5. Fine tune the provisional list using theory. Use a construct approach by reviewing the list of determinants and, either grouped or singly, translate them into constructs that are linked to social and behavioural science theories.
6. Review general theories to see if additional theories or constructs might help answer the question

**Intervention mapping processes adapted for this study**

1. **Phase 1**
   - Question: how can physical activity be successfully promoted in individuals with IGT in a health care setting?

2. **Phase 2**
   - Review the domains that were identified by a multi-disciplinary group of experts (Michie et al. 2005) as being potentially important in the development and implementation of health behaviour interventions.

3. **Phase 3**
   - Use successful free-living physical activity and diabetes prevention programmes to identify which of the domains highlighted by Michie et al. are important in the promotion of physical activity.
   - **Main findings.** The following domains are consistently emphasized in successful free-living physical activity and diabetes prevention programmes: beliefs about capabilities, beliefs about consequences, motivation and goals, behavioural regulation and social influences.

4. **Phase 4**
   - Find a theory/theories that provides a theoretical explanation for the identified domains.
   - **Main findings.** Social cognitive theory can be used to incorporate the elements identified in the previous phase into a theoretical framework.

5. **Phase 5**
   - Use social cognitive theory to refine the list of domains into integrated theoretical constructs.

6. **Phase 6**
   - Review additional health behaviour theories to establish whether any key constructs are missing.
   - **Main findings.** Lethal’s common sense model, Gollwitzer’s implementation intentions and Chaiken’s dual process theory may usefully complement social cognitive theory.
Social cognitive theory (SCT) explains how and why people initiate and maintain certain behavioural patterns and it provides an instructive framework for use in the development of behaviour change interventions. Fundamentally SCT predicts that behaviour change is influenced by current behaviour, personal factors and environmental factors (Bandura 1986). Personal factors include cognitive, affective and biological states and environmental factors include factors that are external to the person, such as physical and social environments. In contrast to many of the behaviourist theories that came before it, which tended to overemphasis the role of external stimuli in predicting and causing behaviour, SCT is rooted in the view that individuals are agents who have the capacity to be proactively engaged in their own development and are capable of making things happen by their own actions (Bandura 1986). In particular SCT is based on the concept that what people think, feel and believe affects how they behave. Key to this is the construct of self-efficacy, defined as: people's judgments of their capabilities to organise and execute courses of action required to attain designated types of performances (Bandura 1986). SCT postulates that self-efficacy is constructed from four principal sources of information: mastery experiences, vicarious experiences, verbal persuasion, and psychological affective states. Mastery experiences are the most influential of these four sources of efficacy information because they serve as a powerful indicator of one's personal capacity to engage in a given health behaviour. Although efficacy beliefs are central to SCT, they act as one of several determinants that regulate motivation, affect and behaviour.

SCT also predicts that outcome expectations contribute to behaviour change (Bandura 1986). SCT states that outcome expectations come in three main forms: positive and negative physical effects that accompany the health behaviour, positive and negative social sanction, and positive and negative self-evaluative reactions to one's behaviour. In each case the perceived positive outcomes act as incentives and the perceived negative outcomes as disincentives. Outcome expectations are linked to self-efficacy because those with high self-efficacy beliefs are more likely to form positive outcome expectations and those with low self-efficacy are more likely to form negative outcome expectations (Bandura 1986).

Impairments or barriers to health behaviour change are also recognized as important factors, both in predicting and effecting exercise behaviour change (Bandura 1986). SCT states that there are two types of barriers: personal/situational and socio-structural. Personal/situational
barriers include lack of time, tiredness and bad weather, while socio-structural barriers include the socio-economic structure of health services. Personal efficacy beliefs are strongly linked to beliefs in one’s capacities to overcome personal/situational barriers, for example those with high self-efficacy are more likely to view personal barriers as surmountable and those with low self-efficacy are more likely to view their attempts at overcoming personal barriers as futile and pointless (Bandura 1986).

Along with these motivational constructs it is increasingly recognized by Bandura and others that self-regulation or self-management is likely to be fundamental to the success of a health promotion intervention (Bandura 2005, Bodenheimer et al. 2002). In the past, many social cognitive models of behaviour assumed that intentions were the most powerful predictor of behaviour. However, intentions are often poor predictors of action (Johnston et al. 2004) and intentions to change are seldom successfully carried out (Sutton 1994). This discrepancy has been termed the “intention-behaviour gap” (Orbell & Sheeran 1998). Self-regulation has been widely reported as an important mechanism for bridging the intention-behaviour gap (de Ridder & de Wit 2006). SCT is one of the few social cognitive theories to explicitly predict that intention and motivation on their own are unlikely to produce behaviour change if not accompanied by the development of self-regulatory skills (Bandura 1997, Bandura 2004).

Therefore, whilst motivational constructs are likely to form important pre-conditions to behaviour change, it is crucial that interventions promoting behaviour change are successful at targeting and promoting self-regulatory skills and effort. SCT states that self-regulation operates through three sub-functions: self-monitoring, goal setting, and enlistment of self-incentive for personal change (Bandura 1986). Self-monitoring provides individuals with the necessary feedback to set realistic proximal goals. Distal and proximal goals act as self-reactive motivators as well as figuring prominently in the development of self-efficacy through mastery experiences. Self-incentives serve as important self-motivators and provide incentives for individuals to undertake behaviour that they might otherwise put off or avoid. Along with self-regulation, social support from friends and family, as well as continued support from health professionals, is also deemed important in maintaining behaviour change because of its effect on motivation, self-efficacy, and affective states.

Several well described physical activity interventions have been based on SCT and have been shown to be successful at initiating long term physical activity behaviour change (Di
Loreto et al. 2003, Stewart et al. 2001). For example, Di Loreto et al. developed a physical activity intervention for individuals with type 2 diabetes that used both the motivational and self-regulatory constructs of SCT (Di Loreto et al. 2003). At two years the intervention was associated with an increase in physical activity energy expenditure of 26 MET-h/week; equivalent to over 60 minutes of moderate-intensity physical activity per day. This remains the most effect free-living physical activity intervention in this patient group.

**Implementation Intentions**

SCT has proven seminal in providing a framework from which subsequent self-regulation theories have been developed (de Ridder & de Wit 2006), the most instructive and widely used of which is Gollwitzer’s implementation intentions (Gollwitzer 1999). The general premise behind implementation intentions is that forming algorithmic “when, where and how plans” will enable individuals to make strategic use of environmental cues that prompt action without requiring any conscious forethought. For example, by making the plan to walk my dog around the woods for half an hour immediately after arriving home from work every week day, I will come to associate arriving home from work with walking the dog, and so I will not need to be consciously thinking about walking the dog on the way home from work, because arriving home will automatically provide the necessary environmental cue for action.

Implementation intentions have proven successful in promoting many health behaviours, including physical activity (Gollwitzer & Sheeran 2004) and the self-regulatory constructs linked to implementation intentions have proven to be strong predictors of physical activity behaviour change (Sniehotta et al. 2005a, Sniehotta et al. 2005b, Sniehotta et al. 2005c, Sniehotta et al. 2006). Implementation intentions are in essence the same construct as “goals” in SCT, however, they do provide the interventionist with a useful and instructive framework for helping participants set appropriate goals by forming simple action plans and should be considered as a key part of any intervention aimed at initiating and maintaining behaviour change.

**Common Sense Model**

Although SCT suggests that knowledge of the health risks/benefits of health behaviours creates the pre-condition for change (Bandura 1986), knowledge is not a fully developed
theoretical construct. Therefore, apart from suggesting that perceived controllability of an illness/risk factor is linked to coping efficacy, SCT does not provide a clear framework for how illness knowledge and beliefs should be targeted. Leventhal's common sense model suggests that individuals conceptualize a health threat in terms of the cause, consequences, identity, control/treatment and timeline of the threat and that these domains will influence subsequent coping behaviour (Leventhal et al. 1980). The common sense model has been demonstrated across a variety of patient groups (Hagger & Orbell 2003). In particular, perceived control has been shown to significantly and positively correlate with illness outcomes and coping behaviour, whereas illness identity and consequences have been shown to significantly and negatively correlate with illness outcomes and coping behaviour in chronic disease (Hagger & Orbell 2003).

In individuals with type 2 diabetes it has been shown that perceived personal control and the perceived effectiveness of treatment predict adherence to treatments, quality of metabolic control and general quality of life (Hampson et al. 2000, Stenstrom et al. 1998, Watkins et al. 2000); whereas perceived lack of control, worse anticipated consequences and perceived symptom load are associated with depression (Hampson et al. 2000, Paschalides et al. 2004). Recently it has also been shown that change in illness perceptions are associated with change in several health behaviours, including physical activity, in individuals with type 2 diabetes (Skinner et al. 2005). Given the similarity in phenotypes between those with diabetes and those with IGT, these associations are likely to be relevant to individuals who are informed they have prediabetes.

Importantly it has also been suggested that individuals conceptualize any identified health threat in terms of the domains of the common sense model (Leventhal et al. 1980). Furthermore, it is also likely that if the information participants receive about an identified health threat does not include all of the domains identified by the common sense model, individuals are likely to acquire this information from elsewhere and are therefore at risk of forming a false set of illness beliefs, which could negatively affect subsequent coping behaviour. Therefore, if an intervention aimed at promoting health behaviour in individuals with prediabetes does not target the domains of the common sense model, it is likely that the intervention could be undermined by participants forming spurious health beliefs. For example, if individuals believe that their prediabetes status is attributable to their genetics, they may be more resistant to increasing their physical activity levels than if they believed that prediabetes was caused by a sedentary lifestyle.
In summary, the Common Sense Model therefore provides an instructive and evidence based framework for targeting illness knowledge and belief that complements SCT.

**Dual process theory**
The Department of Health puts particular emphasis on the importance of adopting a patient-centred approach to group education (Department of Health 2005). In the past, most patient education initiatives relied on imparting knowledge through heuristic processing (Skinner et al. 2003). In this style of learning, the health professional is placed as the expert who imparts their knowledge to their patients verbally; the patient is therefore a passive entity, who absorbs information imparted by an expert. The disadvantages of this approach are that individuals can easily rationalize information provided to them in this manner as being irrelevant to their personal circumstances, and that where attitudes do change they are susceptible to further influence from other sources of information perceived as “expert”, and so are not stable over time (Skinner et al. 2003).

Chaiken’s dual process theory (Chaiken 1987) provides an alternative to the traditional heuristic learning model: dual process theory makes a distinction between heuristic and systematic processing. Whereas in heuristic processing the patient takes a passive role, systematic processing occurs when an individual takes an active role in their learning experience by looking for evidence, examples, reasoning, and logic within the information they are being provided with. The use of a systematic processing approach in patient education encourages individuals to scrutinise information, ask questions, and work things out for themselves. Systematic processing requires greater cognitive effort from the patient but results in patients making a stronger link between theoretical concepts and their personal situation (Skinner et al. 2003).

The DESMOND (Diabetes Education and Self Management for Ongoing and Newly Diagnosed) programme is one of the few patient education programmes that uses a systematic processing approach to patient education (Skinner et al. 2006). By using simple non-technical language, analogies and visual aids, and by asking open questions, the DESMOND programme encourages participants to work out for themselves what is happening in their bodies, how their diabetes relates to them and what can be done to control it. It has been reported that individuals who have attended the DESMOND programme are able to give detailed descriptions of the programme a year later (Skinner et al. 2003), in stark
contrast to more traditional forms of patient consultation (Parkin & Skinner 2002). Furthermore, the DESMOND programme has been found to be successful at targeting illness perceptions and promoting physical activity (Skinner et al. 2005). It can therefore be concluded that education programmes that encourage participants to actively engage in their learning experience will have a greater long-term impact on individuals in terms of their illness knowledge/beliefs, self-efficacy, and outcome expectations than a programme which relies on the traditional heuristic approach.

**Section summary:** A structured education programme that aims to promote physical activity in individuals identified with an increased risk of developing diabetes is likely to maximize its effectiveness if it is based on Bandura’s social cognitive theory (Bandura 1986), Gollwitzer’s implementation intentions (Gollwitzer 1999), Leventhal’s common sense model (Leventhal et al. 1980) and Chaiken’s dual process theory (Chaiken 1987).
Part Three: A consideration of exercise mode and the pedometer

Along with a clear theoretical framework it is also important that physical activity interventions promote forms of physical activity that are appropriate and acceptable to their target populations. It is of little practical benefit promoting gym-based interventions or vigorous intensity physical activity if the majority of the individuals with the most to gain are unable or unwilling to access their local gyms or undertake intensive exercise. A failure to consider the preferences and needs of target populations may explain the poor take-up of, and adherence to, some exercise-on-prescription schemes (Thurston & Green 2004).

Data from epidemiological and intervention studies in the United Kingdom and other developed countries has consistently shown walking to be the preferred choice of physical activity in the general as well as diseased populations (Booth et al. 1997, Cooper et al. 2000, Crespo et al. 1996, Di Loreto et al. 2003, Vaz de Almeida et al. 1999). Interventions that promote walking activity have been shown to be successful at increasing physical activity and improving glycaemic control and cardiovascular risk markers in individuals with diabetes (Di Loreto et al. 2003). Epidemiological data has shown that as little as 30 minutes of walking activity per day has a significant impact on the risk of developing type 2 diabetes compared to being sedentary, even after adjustment for body mass and other likely confounding variables (Hu et al. 1999, Laaksonen et al. 2005). Walking would therefore seem to be an appropriate mode of exercise to use when promoting physical activity in individuals identified with an increased risk of developing diabetes. It is also likely that walking will be associated with fewer barriers than other forms of physical activity in black and minority ethnic populations (Johnson 2000).

The pedometer is widely recognized as a useful aid in the promotion of walking activity. In particular, given that pedometers provide objective feedback to the wearer and that they facilitate clear and simple goals setting, their use is especially relevant to theory-driven physical activity interventions specifically aimed at promoting self-regulatory strategies. In studies where pedometers have been a central component of an exercise intervention, participants have successfully increased their activity levels in the short to medium term (< 1 year) (Bravata et al. 2007). Several studies have also investigated which components are most likely to influence the success of a pedometer intervention study. A study of 400
women health-care workers who purchased a pedometer and were instructed to walk 10,000 steps per day concluded that setting steps/day goals, wearing the pedometer continuously during waking hours, and keeping a record of steps taken were the indicators most likely to predict subsequent levels of physical activity and self-efficacy (Rooney et al. 2003). In another study with 37 individuals, participants identified the following components as having an impact on their ambulatory activity during the study: having step-count goals and strategies to achieve them; being able to examine the number of steps recorded throughout the day; and keeping a log of their daily step count (Croteau 2004). The effectiveness of wearing a pedometer, keeping a log book and having a daily step-count goal was further emphasized in a recent study by Hultquist et al. (2005). In this study, 58 sedentary women were randomized into two groups. Individuals in the first group were told to walk 10,000 steps per day whilst wearing both a sealed pedometer (i.e. one which does not feature a step counter screen) and an unsealed pedometer and told to record their daily step count. Those in the second group, the "30 minutes" group, were told to take a brisk 30 minute walk on most days of the week and to wear a sealed pedometer throughout the day. Those in the 10,000 steps per day group increased their physical activity over a four-week period by 4,550 steps per day, which was twice the increase in steps per day observed in the "30 minutes" group.

Therefore it would appear that encouraging individuals to wear a pedometer, keep a log book and form realistic, personalized steps-per-day goals is likely to aid successful physical activity behaviour change in any programme aiming to promote physical activity. However, in a recent review of the evidence NICE found that pedometer interventions studies are associated with methodological limitations and that consequently the success of pedometer based intervention studies, particularly over the longer term, remains equivocal (National Institute of Health and Clinical Excellence 2006).

Given the primary focus of structured education is to enable participants to actively self-regulate their behaviour using self-monitoring and goal-setting strategies, any structured education programme aimed at increasing walking activity should consider providing participants with pedometers and personalized step per day goals. However, considering the limitation in the current evidence, future research investigating the efficacy of physical activity interventions that incorporate pedometer use could make a valuable contribution to the current evidence by designing a methodology that allows for the direct comparison of providing participants with a pedometer and steps-per-day goals against providing participants with time based goals and an exercise diary.
Section summary: Walking is likely to be the most appropriate and successful mode of physical activity to promote in those identified with an increased of developing diabetes; the pedometer is likely to aid the promotion of walking activity.
Part Four: The PREPARE programme

The PREPARE programme was developed following the review of health behaviour theory, evidence from previous physical activity interventions and NICE guidelines highlighted in the previous three parts of this chapter. The development of the PREPARE programme was also informed by initial pilot studies which are described in Chapter Five. The PREPARE programme is designed to promote physical activity, primarily walking activity, in individuals identified with IGT by targeting perceptions and knowledge of impaired glucose tolerance, physical activity self-efficacy, barriers to physical activity and self-regulatory skills.

The PREPARE programme is based on a single group session which is three hours long and is designed to be delivered to between 5 and 10 individuals. The programme emphasizes a person-centred approach to group education which utilizes simple non-technical language, analogies, visual aids and open questions to encourage the participants to become actively involved in the programme. The PREPARE programme curriculum is divided into four modules. Table 4.1 gives a broad overview of the content, theoretical underpinning and weighting of each module. Although the PREPARE programme is aimed at increasing physical activity, a brief dietary session was included as qualitative research conducted at the University of Leicester had revealed that diet is strongly linked to illness perceptions surrounding IGT (unreported data). For example most participants believed that their IGT status was the direct result of eating too much sugar. Therefore the dietary sessions was used to tackle some common dietary misconceptions linked to metabolic health. However, participants are not encouraged to set dietary goals or action plans.

Two versions of the PREPARE programme were developed, a standard version and a pedometer version. The standard version encourages participants to set time-based goals based on generic exercise recommendations whereas the pedometer version aims to enable participants to set personalized steps-per-day goals and to objectively self-monitor their daily physical activity levels with a provided pedometer. Participants in the pedometer group are helped to set personalised goals by being informed of their baseline ambulatory activity levels and encouraged to set new goals that would enable them to move up an activity category based on pedometer indices proposed by Tudor-Locke and Bassett (2004). Apart
Table 4.1: Outline of the PREPARE programme

<table>
<thead>
<tr>
<th>Module:</th>
<th>Main aims:</th>
<th>Example activity:</th>
<th>Theoretical underpinning:</th>
<th>Time weighting:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient Story</td>
<td>Give participants a chance to share their knowledge and perception of IGT and highlight any concerns they may want the programme to address.</td>
<td>1) Participants are asked to share their story, how they were diagnosed with IGT and their current knowledge of IGT.</td>
<td>Common Sense Model (Leventhal et al. 1980)</td>
<td>10% (20 minutes)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Professional story</td>
<td>Use simple non-technical language, analogies, visual aids and open questions to provide participants with an overview of healthy glucose metabolism, the aetiology of prediabetes, the risk factors and complications associated with prediabetes, the possible causes of prediabetes and possible symptoms associated with prediabetes.</td>
<td>1) The following model for insulin resistance is used: Glucose moves from the blood into cells to be used as energy via a door with a lock on it. Insulin keys are used to open the lock; insulin resistance occurs when the cell locks get rusty. 2) Individuals are given their individual glucose, cholesterol and blood pressure scores and a risk chart and helped to work out their individual risk areas.</td>
<td>Common Sense Model (Leventhal et al. 1980) Dual Process Theory (Chaiken 1987)</td>
<td>35% (60 minutes)</td>
</tr>
<tr>
<td>Diet</td>
<td>Give participants an accurate understanding of the link between dietary macro-nutrients and metabolic dysfunction</td>
<td>1) Participants are asked to group models of fats and oils into saturated, polyunsaturated and monounsaturated categories.</td>
<td>Social Cognitive Theory (Bandura 1986) Dual Process Theory (Chaiken 1987)</td>
<td>15% (25 minutes)</td>
</tr>
<tr>
<td>Module: Physical activity</td>
<td>Main aims: Use simple non-technical language, analogies, visual aids and open questions to help participants: identify how physical activity improves glucose control; understand the current physical activity recommendations; explore options for incorporating physical activity (primarily walking) into everyday life; identify barriers to exercise; form action plans; encourage participants to use their provided physical activity diaries; and set personal goals (based on baseline pedometer counts for the pedometer version of the programme and generic exercise recommendations for the non-pedometer version).</td>
<td>Example activity: 1) Participants are encouraged to share their knowledge of the various exercise recommendations and to work out how each recommendation may affect their health. 2) Participants are provided with a physical activity diary and encouraged to set their first action plan.</td>
<td>Theoretical underpinning: Social Cognitive Theory (Bandura 1986) Implementation Intentions (Gollwitzer 1999) Dual Process Theory (Chaiken 1987)</td>
<td>Time weighting: 40% (75 minutes)</td>
</tr>
</tbody>
</table>
from these differences the two versions of the PREPARE programme were identical. Participants in both groups were given an exercise diary and encouraged to record their daily physical activity levels. As the primary focus of structured education is to enable participants to actively self-regulate their behaviour using self-monitoring and goal-setting strategies, for which the pedometer is ideally suited, and considering the fact that few intervention studies in the UK have achieved sustained increases in physical activity over the long-term, it was hypothesized that only the pedometer version of the PREPARE programme would initiate clinically significant increases in physical activity over the longer term (see Chapter Six where this hypothesis is tested).

A comprehensive written curriculum was developed for the PREPARE programme and is shown in Appendix Four; the physical activity diary developed for the PREPARE programme and the accompanying participant materials are presented in Appendix Five.

**Section summary:** The PREPARE programme is a single-session group-based structured education programme designed to promote physical activity, primarily walking activity, by targeting perceptions and knowledge of impaired glucose tolerance, self-efficacy, barriers and self-regulatory skills.
Chapter Five

PREPARE programme pilot studies
Chapter Five details two pilot studies that were carried to test the main aims of the PREPARE programme. The aim of these pilot studies described in this chapter were to: test the logistics of running the programme; identify whether the programme was targeting the psychological determinants on which it was grounded; test whether the programme was successful at initiating physical activity behaviour change; and collect some instructive feedback from individuals with prediabetes. The methods, results and conclusions of both pilot studies are described below. The results from Pilot Two were presented at the 2007 Diabetes UK Annual Professional conference (Yates et al. 2007e).
Introduction

As the Medical Research Council’s Framework for the Design and Evaluation of Complex Interventions to Improve Health makes clear, exploratory pilot work is a crucial phase between developing and formally testing a complex intervention (Medical Research Council, 2000). Pilot studies allow researchers to ensure that their planned intervention is practicable in the context for which they were developed and to gather preliminary data on whether the intervention is targeting the intended theoretical constructs and behaviour. Information gathered from pilot studies can be used to inform both the content of the intervention and subsequent trials that formally test the intervention. Therefore two exploratory pilot studies were used to aid the development of the PREPARE programme and to inform the design of a subsequent randomized controlled trial.

Pilot One

Aims

The aims of this pilot study were to identify potential problems with the logistics of running the PREPARE programme, ensure that the educators had received sufficient training to be able to deliver the programme curriculum, identify whether the programme was targeting the psychological determinants on which it was grounded and benefit from instructive feedback from individuals with prediabetes.

Methods

Recruitment

A letter was sent out to 50 individuals with prediabetes, inviting them to participate in the pilot. The recipients were identified from a database of individuals screened for type 2 diabetes as part of the STAR (Screening Targeted to those At Risk) study. Twenty six individuals responded to the letter of invitation, eighteen of whom attended the pilot. Of the eighteen participants who took part, nine were male (of which five were White European and four were South Asian) and nine were female (of which seven were White European and 2 were South Asian). The average age of the participants was 60 ± 10 years.
Intervention

The pilot took place in the Diabetes Research Unit at Leicester Royal Infirmary, University Hospitals of Leicester, 2006. When participants arrived, they were asked to fill out a questionnaire pack. The three-hour long PREPARE programme was then delivered by two trained educators. The curriculum of the PREPARE programme was amended slightly in order to make it applicable to individuals with prediabetes in general and not just those with impaired glucose tolerance. At the start of the programme, participants were each given a folder that contained prediabetes definition and risk information, a physical activity diary covering one year and blank pages for taking notes. At the end of the programme, participants were given a follow-up questionnaire to fill out.

Measurements

The pre- and post-interventions questionnaires given to the participants were intended to establish whether the PREPARE programme was successful at targeting the main determinants associated with Bandura's social cognitive theory and Leventhal's common sense model. Feedback from the participants after the intervention was also obtained through a questionnaire.

Psychological determinants

Five domains associated with Leventhal's common sense model (consequences, timeline, personal control, treatment control, and identity) plus illness coherence and two emotional representations (concern and emotional affect) were measured using the brief version of the illness perception questionnaire, which has been found to be a reliable and valid measure of illness perceptions (Broadbent et al. 2006). This questionnaire measures each construct with a single item using a 11 point Likert scale.

A single-item questionnaire was used to measure participants' exercise self-efficacy. As the PREPARE programme is predominantly aimed at increasing walking activity, the question was specifically focused on walking. Participants were asked to rate how confident they were that they could increase the time they spent walking everyday for six incremental time periods (from 10 minutes to 60 minutes in 10 minute increments) using a 100% confidence rating scale (from 0% - not at all confident to 100% - completely confident). A total efficacy score was obtained by summing the confidence scores and dividing by the total number of
increments. Exercise self-efficacy measures using the 100 confidence rating scale are the most common method of measuring exercise self-efficacy (Keller et al. 1999) and have been shown to have good internal reliability (Hu et al. 2005, McAuley 1991, McAuley E et al. 1993, McAuley et al. 2003); Cronbach's alpha values for this study were over 0.9 for both time points.

**Participant feedback questionnaire**

Following the intervention the participants perceptions of the PREPARE programme were measured using a series of closed questions dealing with aspects of the programmes presentation and content using a 5 point response scale ranging from strongly agree to strongly disagree. Participants were also asked whether they would recommend the programme to others with prediabetes and about their exercise intentions. Open ended questions were also used to encourage participants to identify areas of the programme they thought could be improved.

**Statistical analysis**

Data were analysed using SPSS v12.0 software. Data were analysed using Wilcoxon signed ranks test.
Results

Table 5.1 shows the pre- and post-programme scores for the programme outcomes, along with the number of participants included in each analysis. The results show that the programme significantly increased participants’ perceived control and knowledge about prediabetes and significantly decreased the perceived symptom load and consequences associated with prediabetes. The observed increase in the time that participants expected their prediabetes to last (timeline) also tended towards significance. There was also a significant increase in participants’ walking self-efficacy, see table 5.1.

Results from the general feedback questionnaire found that all participants (n=18) strongly agreed or agreed that they would recommend the programme to others with prediabetes and strongly agreed or agreed that the content of the programme was easy to understand. The majority of participants strongly agreed or agreed that the programme: was well structured (n = 17) and well presented (n = 16); provided them with good examples of ways to become more active in their day to day lives (n = 16); and was aimed at the right level (n = 16). In addition, all participants agreed or strongly agreed that they intended to become more active and the majority (n = 16) strongly agreed or agreed that keeping a diary would help them to achieve their activity goals. Participants also generally answered neutrally or disagreed that there was too much audience participation.
Table 5.1: Pre- and post-programme illness perception and efficacy scores. Results are median [interquartile range]

<table>
<thead>
<tr>
<th>Outcome</th>
<th>n</th>
<th>Pre-programme score</th>
<th>Post-programme score</th>
<th>P value for change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perceived affect of prediabetes (consequences)</td>
<td>11</td>
<td>1 [1 to 6]</td>
<td>1 [1 to 3]</td>
<td>0.041</td>
</tr>
<tr>
<td>Timeline associated with prediabetes</td>
<td>12</td>
<td>4 [2 to 6]</td>
<td>5 [3 to 8]</td>
<td>0.095</td>
</tr>
<tr>
<td>Perceived control over prediabetes</td>
<td>12</td>
<td>6 [2 to 7]</td>
<td>8 [3 to 9]</td>
<td>0.024</td>
</tr>
<tr>
<td>Perceived response efficacy of physical activity at treating prediabetes</td>
<td>13</td>
<td>9 [3 to 10]</td>
<td>9 [7 to 9]</td>
<td>0.138</td>
</tr>
<tr>
<td>Perceived symptom load</td>
<td>11</td>
<td>1 [1 to 5]</td>
<td>1 [1 to 1]</td>
<td>0.027</td>
</tr>
<tr>
<td>Concern</td>
<td>12</td>
<td>8 [5 to 10]</td>
<td>9 [5 to 10]</td>
<td>0.581</td>
</tr>
<tr>
<td>Prediabetes coherence (knowledge)</td>
<td>13</td>
<td>4 [2 to 7]</td>
<td>8 [2 to 10]</td>
<td>0.044</td>
</tr>
<tr>
<td>Emotional affect</td>
<td>12</td>
<td>3 [1 to 6]</td>
<td>2 [1 to 4]</td>
<td>0.498</td>
</tr>
<tr>
<td>Walking self-efficacy</td>
<td>7</td>
<td>44 [23 to 70]</td>
<td>63 [32 to 100]</td>
<td>0.017</td>
</tr>
</tbody>
</table>

All scores are between 1 and 10, except walking self-efficacy which was between 0 and 100.
Discussion

The PREPARE programme is predominantly grounded in Bandura’s social cognitive theory (Bandura 1986), Leventhal’s common sense model (Leventhal et al. 1980) and Gollwitzer’s implementation intentions (Gollwitzer 1999). In particular, the PREPARE programme aims to: target perceptions and knowledge of prediabetes; increase exercise self-efficacy; and promote self-regulatory skills. The main aim of this pilot study was to establish whether the PREPARE programme would effectively target the key theoretical determinants associated with these theories. The results of this pilot study show that the programme was successful at targeting some illness perceptions, perceived knowledge of prediabetes and increasing walking self-efficacy. These results suggest that the PREPARE programme is setting the necessary pre-conditions for behaviour change. Additional feedback from the participants also indicated that the programme was aimed at the right level and encouraged an acceptable degree of audience participation.

This pilot also identified several practical problems with the PREPARE programme and the outcomes measured. Firstly, the running time of three hours was insufficient to allow individuals to develop action plans at the end of the programme. Moreover, the two researchers presenting the programme both recognised that participants were becoming increasingly restless and unfocused towards the end of the programme. Some of these problems can be attributed to the fact that the number of individuals who attended Pilot 1 was significantly higher than the maximum of ten that the programme was designed for. Nonetheless, it was agreed that the running time of the PREPARE programme needs to be increased to 3.5 hours in order to allow for an additional break to be incorporated before participants are asked to develop their action plans, this would enable individuals to refocus their attention before this final and important part of the PREPARE programme. Secondly, several participants suggested that the folders given to each participant should have included a summary of the main points raised in the PREPARE programme to serve as a reference afterwards. Therefore, a summary sheet highlighting the most important points addressed in the programme will be included in the participants’ folders in future PREPARE programme sessions. Thirdly, a poor completion rate for the walking efficacy questionnaire, mainly due to individuals giving incomplete answers, indicated that the question was poorly understood. Consequently, any questions using this format in the future evaluation of the programme need to be re-drafted and re-formatted in order to make it easier to understand. In addition, the fact that several individuals failed to complete any section of the questionnaire shows that
it is necessary to check each completed questionnaire pack before participants leave the site. It should be noted that the limited time and relatively large numbers attending the pilot session meant that this was not possible here.

There are several limitations with the design of Pilot 1, the greatest being the lack of a follow-up to assess how illness perceptions, exercise self-efficacy and physical activity levels changed over time after the PREPARE programme. Consequently, it is not known whether the changes observed in this pilot study were maintained in the days and weeks after the programme, or what effect the programme had on physical activity levels. However, this pilot does indicate that the PREPARE programme would be successful at initiating important changes in illness perceptions and efficacy beliefs that are likely to be necessary pre-conditions for the successful initiation and maintenance of physical activity behaviour change. Pilot 1 therefore provides encouraging indications as to the effectiveness of the PREPARE programme but further pilot programmes that include some follow-up measures are needed.
Pilot Two

Aim

The main aim of this pilot study was to establish whether the pedometer version of the PREPARE programme was successful at initiating physical activity behaviour change in those with prediabetes.

Methods

Recruitment

A letter was sent out to 30 individuals with prediabetes, inviting them to participate in the pilot; recipients were identified from a database of individuals who had been screened for type 2 diabetes as part of the STAR (Screening Targeted to those At Risk) Study. Twelve individuals responded to the letter of invitation, ten of whom attended the PREPARE programme, which took place in the Diabetes Research Unit at Leicester Royal Infirmary, University Hospitals of Leicester. Of the ten participants who took part, six were male and four were female. The ethnicity of the male and female participants was equally divided between white Europeans and South Asians. The average age of the participants was 62 ± 10 years.

Intervention

The pedometer version of the PREPARE programme was delivered on two consecutive days, with four individuals attending on the first day and six on the second. When participants arrived, they were asked to complete a questionnaire pack. The pedometer version of the PREPARE programme was then delivered by two trained educators. At the start of the programme, participants were each given a folder that contained information on the definition of prediabetes and the risks associated with it, a physical activity diary covering one year and blank pages for taking notes. In response to the feedback for Pilot 1, participants were also given a brief summary of the content of the PREPARE programme.
Measurements

Baseline measurements were taken in the week before participants attended the programme and follow-up measurements were taken two weeks after participants had attended the programme.

Physical activity

Stormlite™ pedometers and log sheets were posted to participants who had confirmed they wanted to attend the PREPARE programme. Preliminary testing of the pedometers found them to have moderate validity when compared to a criterion pedometer (NL-800) (within ± 20% of criterion value) and intra-instrument reliability when worn in free-living conditions for a 12-hour period (within ± 10% of each other when worn concurrently on the right and left hip). The participants were instructed to wear the pedometer for a seven-day period before they attended the PREPARE programme and to record the number of steps they took each day on the log sheet. In order to ensure the pedometer was worn correctly, clear written instructions and a diagram depicting correct pedometer placement were sent with the pedometer. Participants were asked to keep the pedometer for two weeks after the PREPARE programme and continue recording their daily step counts.

Physical activity was also measured by the international physical activity question (IPAQ) (last 7 day short form) and is reported in MET-minutes/week derived from vigorous-intensity, moderate-intensity, and walking activity. IPAQ has been found to be a valid and reliable measure of physical activity (Craig et al. 2003).

Diet

Food intake was measured using the dietary instrument for nutritional education (DINE) food frequency questionnaire. The DINE food frequency questionnaire was designed to be a quick and easy way of measuring fibre, total fat and unsaturated fat intake in primary care (Roe et al. 1994). As dietary fat and fibre intake are the most likely dietary variables to influence glucose control and the development of diabetes (Steyn et al. 2004), the DINE food frequency questionnaire is an ideal instrument for use in the proposed study.
Psychological determinants

Illness perceptions were measured with the brief illness perception questionnaire (Broadbent et al. 2006), details of which are given in Pilot 1.

Exercise self-efficacy was measured using the methods described in Pilot 1. However, in order to overcome comprehension difficulties that became apparent in Pilot 1 when participants were asked to fill in the appropriate percentage themselves, the 100% confidence rating scale was set out on the page in a manner that mimicked an 11 point Likert-scale using increases in 10-unit intervals from 0% (no confidence) to 100% (complete confidence). This format has been identified as an acceptable use of the 100% confidence scale by Bandura (Bandura 2001), and was chosen because it combines the sensitivity and reliability of the 100% confidence scale with the ease of use of the Likert scale.

Participant feedback

Participants were given the same feedback questionnaire as described in Pilot 1. In addition, participants were asked an open-ended question about what key message they took away with them from the programme.

Statistical analysis

Data were analysed using SPSS v12.0 software. Paired sample t tests were used to test for within group differences between the pre- and post- programme measurements if the data was normally distributed. Non-parametric data were analysed using the Wilcoxon signed ranks test.
Results

Baseline and follow-up pedometer data was unavailable for three participants, due to non-submission of follow-up log sheets. Pedometer counts increased significantly (P<0.05) by 1690 ± 1760 in the two weeks after the PREPARE programme (see Table 5.2).

Baseline and follow-up information for self-reported physical activity energy expenditure was unavailable for four individuals due to IPAQ questions being incompletely answered (n=1) or non-submission of follow-up data (n=3) (see Table 5.2). The observed increase in self-reported physical activity energy expenditure was equivalent to just under 2 hours of walking activity per day.

Baseline and follow-up illness perception data was unavailable for four individuals due to non-submission of follow-up data (n=3) or refusal to answer the questionnaire items at baseline (n=1). None of the changes to illness perception determinants, apart from the increase in perceived knowledge, achieved significance (see Table 5.2).

Baseline and follow-up self-efficacy data was unavailable for three participants due to non-submission of follow-up data. Exercise self-efficacy increased at follow-up compared to baseline, however this increase was not significant (see Table 5.2).

Baseline and follow-up dietary data was only available for 7 participants due to the non-submission of follow-up data. No change was found in fibre, total fat, or unsaturated fat intake.

Data from the feedback questionnaire was available for all participants. All participants strongly agreed or agreed that: they would recommend the programme to others with prediabetes; the programme helped them understand their prediabetes better; the programme was easy to understand; the programme provided them with good examples of ways to become more active; and that they intended to become more physically active. When asked, “What key messages did you take away from this programme?”, all participants replied that they needed to change their lifestyle and/or that they have the chance to turn their prediabetes around. Participants also indicated that they were happy with the programme and, apart from the suggestion that an interpreter would be a good idea, no one offered any suggestions about how the programme could be improved.
Table 5.2: Baseline and follow-up data for physical activity, illness perceptions and exercise self-efficacy. Results are mean ± SD or median [interquartile range].

<table>
<thead>
<tr>
<th>Outcome</th>
<th>n</th>
<th>Baseline</th>
<th>Follow-up</th>
<th>P value for change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steps per day</td>
<td>7</td>
<td>8021 ± 2765</td>
<td>9710 ± 1673</td>
<td>0.044</td>
</tr>
<tr>
<td>Energy expenditure (MET-min/week)</td>
<td>6</td>
<td>1540 [280 to 1870]</td>
<td>4158 [2133 to 5484]</td>
<td>0.043</td>
</tr>
<tr>
<td>Perceived affect of prediabetes (consequences)*</td>
<td>6</td>
<td>2 [0 to 4]</td>
<td>2 [0 to 5]</td>
<td>0.296</td>
</tr>
<tr>
<td>Timeline associated with prediabetes*</td>
<td>6</td>
<td>5 [2 to 7]</td>
<td>4 [1 to 6]</td>
<td>0.130</td>
</tr>
<tr>
<td>Perceived control over prediabetes*</td>
<td>6</td>
<td>5 [3 to 9]</td>
<td>8 [5 to 9]</td>
<td>0.141</td>
</tr>
<tr>
<td>Perceived response efficacy of physical activity at treating prediabetes*</td>
<td>6</td>
<td>7 [3 to 10]</td>
<td>9 [5 to 10]</td>
<td>0.419</td>
</tr>
<tr>
<td>Perceived symptom load*</td>
<td>6</td>
<td>3 [0 to 7]</td>
<td>0 [0 to 3]</td>
<td>0.285</td>
</tr>
<tr>
<td>Concern at having prediabetes*</td>
<td>6</td>
<td>5 [3 to 9]</td>
<td>6 [5 to 8]</td>
<td>0.581</td>
</tr>
<tr>
<td>Prediabetes coherence (knowledge)*</td>
<td>6</td>
<td>3 [1 to 5]</td>
<td>8 [7 to 9]</td>
<td>0.003</td>
</tr>
<tr>
<td>Emotional representations*</td>
<td>6</td>
<td>5 [2 to 6]</td>
<td>2 [0 to 5]</td>
<td>0.565</td>
</tr>
<tr>
<td>Exercise self-efficacy†</td>
<td>7</td>
<td>36 [15 to 69]</td>
<td>60 [37 to 97]</td>
<td>0.282</td>
</tr>
</tbody>
</table>

* Questionnaire score between 1 and 10

† Questionnaire score between 0 and 100
Discussion

The main finding of Pilot Two was that the PREPARE programme was successful at initiating physical activity behaviour change in individuals with prediabetes. In the two weeks after participants attended the PREPARE programme, their pedometer counts increased significantly by 1690 ± 1760 steps/day compared to before the PREPARE programme. As a key aim of the PREPARE programme is to stress the importance to participants of using small incremental steps on the way to achieving their final activity goals, participants were encouraged to form action plans scheduling a maximum increase of 500 steps/day in walking activity per week; this target may have limited the increases in walking activity compared to what might have been expected if no such instructions had been given to the participants. Nevertheless, the increase in walking activity seen in this pilot is comparable to the increase in steps/day reported in the first weeks after other pedometer-based physical activity interventions in both the general population (Chan et al. 2004, Croteau 2004) and individuals with type 2 diabetes (Tudor-Locke et al. 2002a, Tudor-Locke et al. 2004), although the moderate reliability and validity of the pedometers used for this pilot study make direct comparisons between studies difficult.

Apart from perceived knowledge of prediabetes, Pilot 2 failed to demonstrate a significant effect on the psychological determinants underpinning the PREPARE programme. However, the small sample size makes it likely that a type 2 error occurred in the analysis of these variables. This conclusion is supported by the fact that the magnitude of change in perceived control over prediabetes, exercise efficacy and symptom load were similar to pilot one, where these changes were significant. The 40% increase in self-efficacy seen in this pilot study is in contrast to a study that used a similar group-based behavioural modification programme for cardiac rehabilitation patients, where initially high levels of exercise self-efficacy significantly decreased in the first month after the programme, which the authors hypothesized was due to the participants being confronted with the reality of attempting and maintaining lifestyle exercise change (Moore et al. 2006). The small increases in self-efficacy seen in this pilot study are therefore encouraging.

Despite brief dietary advice being included in the curriculum of the PREPARE programme, no change in dietary intake was seen. This suggests that the PREPARE programme is achieving its aim of primarily targeting physical activity behaviour change, thereby making
it likely that any physiological effect that may be attributed to the PREPARE programme in future studies will be caused by physical activity, rather than dietary, change.

In Pilot 2, in contrast to Pilot 1, the delivery of the PREPARE programme was completed in under three hours on both occasions. The discrepancy between the running time of the PREPARE programme in the two pilot studies is likely to be attributable to the smaller group sizes in Pilot 2. As the group sizes in Pilot 2 were representative of the group size for which the PREPARE programme was designed, three hours seems like a reasonable upper time limit for the programme. The PREPARE programme will therefore continue to be a three-hour programme.

Overall, Pilot 2 provides further encouraging evidence for the effectiveness of the PREPARE programme at promoting physical activity behaviour change. However, a randomised controlled trial is needed to adequately test the efficacy of the PREPARE programme at promoting physical activity and improving relevant biological endpoints.
**Chapter Summary**

*Exploratory pilot work revealed that the PREPARE programme is well tolerated by participants and successful at initiating physical activity behaviour change.*
Chapter Six

The PREPARE Programme Study
This chapter details a randomized controlled trial designed to test the efficacy of the PREPARE programme at initiating physical activity behaviour change and improving glucose tolerance in individuals with impaired glucose tolerance. This randomized controlled trial is the culmination of the work detailed in the preceding chapters; consequently, the previous chapters provide a detailed introduction and background to this study. For the sake of clarity, the background to this chapter provides a brief summary of the main findings and conclusions reached in this thesis thus far.

The randomized controlled trial detailed in this chapter was funded by a small grant from Diabetes UK. In addition, links were formed – through the University of Leicester – with Unilever Corporate Research, who agreed to fund and carry out analysis of fasting insulin, C-reactive protein, interleukin-6 and tumour necrosis factor-a.

Intermediary results from the PREPARE programme study have been presented at the Second International Congress of Physical Activity and Public Health, Amsterdam, 2008 (Yates et al. 2008d) and at the Annual Meeting of the European Association for the Study of Diabetes (EASD), Rome, 2008 (Yates et al. 2008e). An abstract detailing results at 3-months was also short-listed for the Education Award at the Diabetes UK Annual Professional Conference, Glasgow, 2008 (Yates et al. 2008c). A full paper detailing the complete set of results detailed in this chapter is currently being prepared for submission to a high impact medical journal.
Background

The prevalence of diabetes is reaching epidemic proportions and the costs associated with its treatment are set to represent a serious clinical and financial challenge to national health systems (Bagust et al. 2002). It is therefore of primary importance to develop diabetes prevention strategies in high-risk populations in order to counter this worrying trend. Individuals with impaired glucose tolerance (IGT) have an increased risk of developing diabetes and cardiovascular disease compared to those with normal glucose tolerance and are therefore a suitable population for diabetes prevention initiatives (Unwin et al. 2002)(see Chapter One).

Although increased physical activity has consistently been associated with a reduced risk of developing diabetes (Jeon et al. 2007), the success of traditional multi-factor diabetes prevention programmes, which have typically utilized one-to-one counselling strategies, at promoting physical activity in individuals with IGT has been limited (see Chapter 2). This mirrors findings from other randomized controlled trials that have shown that traditional one-to-one counselling strategies are ineffective at promoting long term physical activity behaviour change when conducted in a primary health care or community setting in the UK (Hillsdon et al. 2002, Kinmonth et al. 2008). There is, therefore, a need to develop effective alternative methods of promoting physical activity for individuals identified with an increased risk of developing type 2 diabetes, that are cost-effective and appropriate for a primary health care or community setting.

Structured education has recently been shown to be successful at promoting self-management and increasing physical activity in individuals with type 2 diabetes in the UK (Davies et al. 2008, Deakin et al. 2006)(see Chapter 4). Structured education has also proven to be compatible with the infrastructure of the National Health Service and has been welcomed by patient groups, including those with type 2 diabetes (Department of Health 2005). Therefore this approach to patient care could be a feasible way of promoting increased physical activity and a healthy lifestyle in those identified with an increased risk of developing type 2 diabetes. However, despite being advocated for numerous patient groups, structured education has not yet been adequately tested as a method of promoting self-management and a healthy lifestyle in individuals identified with an increased risk of developing type 2
diabetes, although preliminary pilot data suggests that it may be a successful method of promoting physical activity in a health care setting with at-risk individuals (see Chapter Five).

The Prediabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme is a structured education programme aimed at promoting physical activity and improving glucose tolerance in individuals identified with IGT (see Chapter Four). This randomized controlled trial was designed to test the pedometer and standard versions of the PREPARE programme against control conditions (see Chapter Four). The primary aim of the study was to test the hypothesis that the pedometer version of the PREPARE programme is effective at improving glucose tolerance in individuals identified with IGT over a one year period. The secondary aim of the study was to test the hypothesis that, when compared to control conditions, only the pedometer version of the PREPARE programme results in clinically significant improvements in glucose tolerance. Additional secondary aims included investigating whether the PREPARE programme is effective at: targeting the key psychological determinants that underpin the programme, initiating sustained increases in ambulatory activity and reducing markers of chronic low-grade inflammation.
Methods

This study was developed and reported according to criteria for reporting parallel randomized controlled trials detailed in the revised CONSORT statement (Moher et al. 2001).

Research design

The study was powered to detect a 1 mmol/l difference in 2-hour glucose levels between the primary intervention and control group at 12 months. Using a power of 80%, a significance level of 0.05, a standard deviation of 1 mmol/l (Knowler et al. 2002, Tuomilehto et al. 2001), and allowing for a 50% drop-out rate, two groups of 34 individuals were required to test the primary hypothesis (Campbell et al. 1995). After including a third group of the same size to test the secondary hypothesis, a total of 102 participants was required.

Although the PREPARE programme was a behaviour change strategy designed to promote increased physical activity, 2-h glucose was chosen as the primary outcome because if a behaviour change initiative aimed at increased physical activity in a group of individuals with IGT is to be advocated in a wider health care setting it must be shown that the initiative is successful at targeting the primary biological outcome which defines the condition – namely elevated 2-h glucose levels. This is all the more important given that, as was shown in Chapter Two, there is not enough evidence from randomized controlled trials to allow for an accurate quantification of the amount of physical activity required to achieve clinically significant reductions in 2-h glucose. Given this, objectively measured ambulatory activity and self-reported moderate- to vigorous-intensity physical activity were assigned as secondary outcomes.

Given the relatively small sample size needed to test the primary hypothesis, participants were randomized using a block design and stratified by age and sex in order to increase the likelihood of randomization producing equivalent groups. Randomization was conducted using opaque envelopes and a randomly generated number sequence (SPSS, Chicago, USA) by a member of our research team with no prior knowledge of recruited individuals, other than their age and sex. Participants were informed of their allocated group once baseline measurements were completed.
Participants were followed at 3, 6 and 12 months after baseline. Whilst our primary hypothesis is related to change at 12-months, results at 3-months and 6-months were included in this study in order to assess the impact of both intervention conditions over time on measured outcomes.

**Recruitment**

Participants were recruited from ongoing population-based diabetes screening programmes in Leicester, UK, between September 2006 and March 2007. Individuals were invited to take part in the study if, at screening, they had impaired glucose tolerance (2-hour blood glucose of ≥ 7.8 mmol/l and < 11.1 mmol/l and fasting glucose < 7.0 mmol/l) (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 1997) and had a body mass index (BMI) of 25 Kg/m² or greater (23Kg/m² or greater for those from a South Asian ethnic background) (World Health Organization Expert Consultation 2004). Individuals who reported taking steroids or who were unable to take part in moderate-intensity physical activity were excluded. Individuals diagnosed with type 2 diabetes during the study period were treated according to the protocol of the screening studies from which they were recruited and withdrawn from this trial. Those not attending the 12-month follow-up were also excluded from the analysis of this study.

**Treatment regimens**

Participants were randomized to receive either usual care, the standard PREPARE programme (E group) or the pedometer version of the PREPARE programme (EP group).

**PREPARE programme**

The PREPARE programme is a single-session group education programme designed to promote increased physical activity, primarily walking activity, by targeting perceptions and knowledge of impaired glucose tolerance, physical activity self-efficacy, barriers, and self-regulatory skills. The programme is designed to be delivered to between 5 to 10 participants and is three hours long. Chapter Four describes the PREPARE programme in greater detail. Each PREPARE programme session was delivered by two educators. Educators held an undergraduate degree in a relevant discipline (dietician, sports scientist) and were trained to
deliver the DESMOND curriculum (Skinner et al. 2006), which is an established structured education programme with a similar philosophy and theoretical underpinning to the PREPARE programme. In addition, all educators completed at least two pilot sessions of the PREPARE programme and received instructive feedback from an experienced and accredited DESMOND educator before delivering the PREPARE programme in this randomized controlled trial.

Individuals randomized into one of the two intervention groups also received a brief (10 minute) review of progress counselling session with a trained educator at their 3-month and 6-month clinical measurement session. There was no additional contact with the research team.

**Standard version**
Participants randomized to the standard version of the PREPARE programme were encouraged to set physical activity goals based on generic exercise recommendations, such as 30 minutes of moderate intensity exercise on most days of the week (Department of Health 2004b, Haskell et al. 2007). Participants were provided with an activity diary and encouraged to self-monitor their activity by recording the time they spent in moderate- to vigorous-intensity physical activity each day (see Chapter Four).

**Pedometer version**
Participants randomized to the pedometer version of the PREPARE programme were provided with a pedometer and encouraged to set personalized steps-per-day goals and action plans based on their baseline ambulatory activity levels and steps-per-day categories proposed by Tudor-Locke (Tudor-Locke & Bassett 2004); participants were encouraged to wear their pedometer on a daily basis and to self-monitor their ambulatory activity using a steps per day log (see Chapter Four).

**Usual care**
Participants randomized to the control group were sent a brief information sheet in the post detailing the likely causes, consequences, symptoms and timeline associated with IGT, along with information about how physical activity can be used to treat/control the condition (see Appendix Six). No additional advice or encouragement was given to the control group.
Measures

The PREPARE study was evaluated using biochemical variables, anthropometric variables, physical activity and dietary measures, as well as relevant psychological determinants. The primary outcome was 2-h glucose. All other outcomes were secondary.

Biochemical variables

At their baseline appointments participants underwent an oral glucose tolerance test (OGTT). Participants arrived at their appointment after a 12-hour fast and 24 hours of avoiding vigorous intensity exercise. Those who had a fasting or 2-hour blood glucose level in the diabetes range (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus 1997) were called back for a second oral glucose tolerance test; if the participant also had a fasting or 2-hour glucose level in the diabetes range at the second test a diagnosis of diabetes was confirmed. Venepuncture and OGTT timings were undertaken by trained phlebotomists who were not part of the scientific advisory team for this study and who were blinded to treatment allocation. All biochemical analysis was also conducted blinded to treatment group.

Plasma glucose was measured using a glucose oxidase method on the Beckman Auto Analyzer (Beckman, High Wycombe, UK). Serum cholesterol was analysed using the cholesterol enzymatic assay (Abbott Clinical Chemistry, IL, USA). High density lipoprotein (HDL) cholesterol was analysed using the ultra HDL assay (Abbott Clinical Chemistry, IL, USA). Serum triglyceride was analysed using the triglyceride glycerol phosphate oxidase assay (Abbott Clinical Chemistry, IL, USA). Glucose and lipid profile measurements were undertaken in the same laboratory located within Leicester Royal Infirmary using stable methodology standardised to external quality assurance reference values.

CRP was analysed on an ABX Pentra clinical chemistry analyser using a latex-enhanced immunoturbidimetric assay, which has a lower sensitivity of 0.1 mg/l (Horiba Group, Montpellier, France). TNF-α and IL-6 were analysed using quantikine high-sensitivity enzyme-linked immunosorbent assays (R&D Systems, Abingdon, UK). All assays were conducted twice on the same occasion and the average value obtained. If the intraassay
coefficient of variation exceeded 10% for IL-6, CRP or insulin or 20% for TNFα then the assay was rerun using the same technique. The interassay coefficients of variation did not exceed 10%, apart from TNF-α which had a maximum interassay coefficient of variation of 17%. Adipocytokine, CRP and insulin analysis were conducted at Unilever Cooperate Research, Colworth, Bedfordshire.

Insulin resistance was determined using homeostasis model assessment (HOMA-IR), obtained from the following formula: HOMA-IR = [fasting insulin (µU/ml) x fasting (mmol/l) glucose]/22.5 (Matthews et al. 1985).

Physical activity

Physical activity was measured objectively using a pedometer and subjectively with a questionnaire. Sealed piezoelectric pedometers with a seven-day memory (NL-800, Newlifestyles, USA) were used for this study. These pedometers have been shown to have good reliability (α = 0.995) and are more accurate than traditional spring-levered pedometers for use on overweight and obese individuals (Crouter et al. 2005, Schneider et al. 2004). In general, as well as being an accurate and reliable measure of ambulatory activity, pedometers have also been shown to have a reasonable degree of correlation with other objective measures of overall physical activity levels and energy expenditure, such as accelerometers (median of reported correlations r = 0.86) and indirect calorimetry and heart-rate derived energy expenditure (median of reported correlations r = 0.68) (Tudor-Locke et al. 2002b). At baseline, all participants were fitted with a pedometer and instructed to wear it during waking hours for seven consecutive days and to keep a daily log of the time the instrument was worn. At the end of the seven day period participants returned the pedometers by post to the research centre, where the data was extracted from the instrument and matched to the time the pedometer was worn. For the purposes of this study at least three valid days of data were required; a valid day constituted at least 12 hours of wear time. It has been shown that the average steps per day of any weekly three day combination is highly correlated (r > 0.8) with the average steps per day taken over the full seven day period; consequently, three or more days of data provides an acceptable measure of walking activity levels over seven consecutive days (Tudor-Locke et al. 2005).
Physical activity was also measured using the long last-seven-days self-administered format of the International Physical Activity Questionnaire (IPAQ)(see Appendix Seven). This questionnaire provides a comprehensive measure of walking and other moderate- to vigorous-intensity activities carried out for more than 10 continuous minutes at work, in the home, as transport and during leisure time. IPAQ has been shown to have reasonable validity compared to accelerometer data ($\rho \sim 0.4$) and test-retest reliability ($\rho \sim 0.7$) in the United Kingdom when used as a measure of total moderate- to vigorous-intensity physical activity (Craig et al. 2003). For this study IPAQ was used to measure total walking activity accumulated at work, for transport and in leisure time as well as an overall measure of moderate- to vigorous-intensity physical activity (including walking activity) accumulated over all contexts.

Diet

Diet was measured using the DINE food frequency questionnaire (see Appendix Eight), which was designed to be a quick and easy way of measuring fibre, fat and unsaturated fat intake in primary care (Roe et al. 1994). As dietary fat and fibre are the only dietary variables that have consistently been associated with the development of type 2 diabetes (Steyn et al. 2004), this questionnaire is highly appropriate for this study. The DINE food frequency questionnaire has been shown to have reasonable correlation with weighted food records ($0.45 < r < 0.51$) (Little et al. 1999, Roe et al. 1994).

Psychological variables

Perceptions and perceived knowledge of IGT

Perceptions and perceived knowledge of IGT were measured with the validated brief illness perceptions questionnaire (Broadbent et al. 2006)(see Appendix Nine). This eight item instrument uses a 11 point Likert scale (0 = no effect, 10 = complete effect) to measure five cognitive illness representations (consequences, timeline, personal control, treatment control, and identity), two emotional representations (concern and emotion) and illness comprehensibility (perceived knowledge of IGT). The brief illness perception question provides a practical and comprehensive measurement of determinants identified in Leventhal’s common sense model (Leventhal et al. 1980), one of the key theoretical models underpinning the content and structure of the PREPARE programme. The brief illness
perception questionnaire has been shown to have reasonable test-retest reliability and concurrent validity when compared to the revised illness perception questionnaire in several patient groups (Broadbent et al. 2006).

**Walking and exercise self-efficacy**

Walking self-efficacy was measured using the 100% confidence rating scale (from 0% = no confidence to 100% = complete confidence) (Keller et al. 1999). This self-efficacy questionnaire measures participants' confidence in their ability to walk for 10 minute periods, increasing incrementally from 10 minutes to one hour each day (see Appendix Ten). The same scale was used to measure participants' confidence in their ability to undertake any other form of exercise (see Appendix Eleven). An overall score for walking and exercise self-efficacy was calculated by summing the efficacy scores for each time period divided by the number of time periods. Exercise self-efficacy measures using the 100% confidence rating scale have been shown to have good (α > 0.8) internal reliability (Cox et al. 2003, Hu et al. 2005, McAuley et al. 1993, McAuley et al. 2003).

**Exercise self-regulatory efficacy**

Participants' confidence in their ability to self-regulate their exercise behaviour in the face of five commonly identified barriers (tired, bad mood, bad weather, lack of time and holiday) was also measured (Marcus et al. 1992) (see Appendix Twelve). This five item questionnaire has been shown to have adequate (α > 0.7) internal reliability (Cox et al. 2003, Marcus et al. 1992). The 100% confidence rating scale was used; an overall score for self-regulatory efficacy was calculated by summing the efficacy scores for each barrier divided by the number of barriers.

**Demographic and anthropometric measurements**

Arterial blood pressure was measured in the sitting position (Omron, Healthcare, Henfield, UK); three measurements were obtained and the average of the last two measurements was used. Body weight (Tanita TBE 611, Tanita, West Drayton, UK), waist circumference (midpoint between the lower costal margin and iliac crest) and height were also measured, to the nearest 0.1 kg and 0.5 cm respectively. Information on current smoking status, medication history, and ethnicity were obtained by self-report.
Ethics

All applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research. This study was approved by the Leicestershire, Northamptonshire and Rutland NHS Research Ethics Committee in June 2006.

Data analysis

Those not attending the final 12-month follow-up measurement session were excluded from the analysis. For those included in the analysis missing pedometer, adipocytokines and CRP data at 12-months were imputed using group-specific regression modelling with the last known value included as the covariate. In order to have numeric parity across time points, missing pedometer, biochemical and anthropometric data at 3-months and 6-months, resulting from those not attending all intermediary follow-up sessions, were imputed using the next observation carried backwards. This method of imputation has been shown to work well when it is anticipated that the overall trend will be for worse outcomes over time (Engels & Diehr 2003), which was hypothesised to be the likely scenario in all groups after the three-month follow-up. As missing baseline data were not imputed, the number of participants included in the analysis for anthropometric, biochemical and pedometer count data at each time point is equal to number of complete baseline datasets. Due to high variability and low predictability across time points, missing questionnaire data were not imputed. Those who were included in the analysis at 12-months were analysed in the group to which they were assigned.

Differences between groups at baseline were analysed using analysis of variance (ANOVA) procedures, Kruskal-Wallis tests, and chi-square tests for, respectively, normally distributed continuous data, nonparametric continuous data and categorical data.

Between-group comparisons of change in measured outcomes at 3, 6 and 12 months was conducted using analysis of co-variance (ANCOVA) procedures; randomization stratifiers (age and sex) and baseline data were included as covariates. Each intervention group was compared to the control group using simple a priori contrasts; as this study had one primary hypothesis, adjustment was not made for multiple group, time or outcome comparisons (Altman 1991, Bender & Lange 2001, Schulz & Grimes 2005). All variables were checked
for normality using the Kolmogorov-Smirnov test and visual inspection after the removal of extreme outliers (a value at least 4 standard deviations from the mean). Non-parametric data was log₁₀ transformed. Associations of change in measured biochemical and anthropometric variables with change in measures of physical activity were quantified partial Pearson correlation. Tests were two sided; p<0.05 was considered significant. All analysis was carried out on SPSS 14.0 for Windows (SPSS, Chicago, USA).
Results

The trial profile is shown in Figure 6.1. In total, 326 individuals were invited to take part in the study, 103 (32%) of whom consented to take part. Those who consented to take part were of a similar age and ethnicity compared to those who declined the invitation; however, relatively more men than women agreed to take part (63% of study participants were male compared to 55% of those invited to take part; p = 0.03). These 103 individuals were assigned to one of the three treatment conditions. Over the course of the trial nine participants were excluded as per protocol due to a diagnosis of type 2 diabetes and a further 11 were lost to 12-month follow-up. There were no significant demographic, biochemical or lifestyle differences between attendees and non-attendees at 12 months. Of those who attended 12-month follow-up, two participants assigned to the E group and one assigned to the EP group did not receive the intervention. This study reports results for participants without type 2 diabetes who attended 12-month follow-up.

Randomization produced similar groups: Table 6.1 shows the measured demographic, anthropometric, biochemical and lifestyle baseline characteristics of the study participants whilst Table 6.2 shows their psychological baseline characteristics. The study population comprised a multi-ethnic, mixed-sex group, the majority of whom were inactive based on pedometer counts (Tudor-Locke & Bassett 2004). However participants were confident that exercise was an effective treatment for IGT and were confident in their ability to carry out daily walking activity.

Biochemical and anthropometric outcomes

All individuals had complete glucose and lipid profile data at baseline and 12-month follow-up, six (7%) had missing data at 3-months and 3 (4%) at 6-months. Due to the cessation of bleeding during venepuncture only 77 out of 83 (93%) participants had complete adipocytokine, CRP and insulin data at baseline; of these 62 (75%), 66 (80%) and 72 (87%) had complete data at 3 months, 6 months and 12 months respectively.

All individuals had complete anthropometric data at baseline and 12-month follow-up, six (7%) had missing data at 3-months and 3 (4%) at 6-months.
326 invited to take part in the study

103 randomly assigned

35 allocated to the control group
34 allocated to the PREPARE (E) group
34 allocated to the PREPARE + pedometer (EP) group

3 lost to 3-month follow-up
- 1 excluded due to a diagnoses of type 2 diabetes
- 1 on holiday
- 1 family illness

4 lost to 3-month follow-up
- 2 excluded due to a diagnoses of type 2 diabetes
- 2 moved away

1 lost to 3-month follow-up
- 1 excluded due to a diagnoses of type 2 diabetes

32 completed 3-month follow-up
30 completed 3-month follow-up
33 completed 3-month follow-up

7 lost to 6-month follow-up
- 4 excluded due to a diagnoses of type 2 diabetes
- 1 family illness
- 2 illness

6 lost to 6-month follow-up
- 4 excluded due to a diagnoses of type 2 diabetes
- 2 moved away

4 lost to 6-month follow-up
- 1 excluded due to a diagnoses of type 2 diabetes
- 1 unwilling to attend
- 1 unable to contact
- 1 illness

28 completed 6-month follow-up
28 completed 6-month follow-up
30 completed 6-month follow-up

9 lost to 12-month follow-up
- 4 excluded due to a diagnoses of type 2 diabetes
- 1 family illness
- 2 illness
- 1 unable to contact
- 1 unwilling to attend

6 lost to 12-month follow-up
- 4 excluded due to a diagnoses of type 2 diabetes
- 2 moved away

5 lost to 12-month follow-up
- 1 excluded due to a diagnoses of type 2 diabetes
- 1 unwilling to attend
- 1 holiday
- 1 unable to contact
- 1 work commitments

26 completed 12-month follow-up
28 completed 12-month follow-up
29 completed 12-month follow-up
### Table 6.1: Clinical, lifestyle and demographic characteristics of study participants overall and by group at baseline.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Control</th>
<th>PREPARE minus pedometer</th>
<th>PREPARE plus pedometer</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>65 ± 8</td>
<td>65 ± 10</td>
<td>64 ± 8</td>
<td>66 ± 8</td>
<td>0.61</td>
</tr>
<tr>
<td>Sex</td>
<td>0.72</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>56 (67)</td>
<td>16 (61)</td>
<td>20 (71)</td>
<td>20 (69)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>27 (33)</td>
<td>10 (39)</td>
<td>8 (29)</td>
<td>9 (31)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td>0.15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| White     | 61 (73) | 17 (65) | 19 (68)                  | 25 (86)                | (for White)
| South Asian | 21 (26) | 9 (35)  | 8 (29)                   | 4 (14)                 | White vs. other |
| Black     | 1 (1)  | 0 (0)   | 1 (3)                    | 0 (0)                  |      |
| Blood pressure medication | | | | | |
| Beta-blockers | 27 (33) | 8 (31)  | 11 (39)                  | 8 (28)                 | 0.64 |
| Ace-inhibitors | 16 (19) | 5 (19)  | 3 (11)                   | 8 (28)                 | 0.27 |
| Statins   | 46 (55) | 16 (62) | 14 (50)                  | 16 (55)                | 0.59 |
| Smoking status | 7 (8)  | 4 (15)  | 2 (7)                    | 1 (3)                  | 0.27 |
| Pedometer counts (steps per day) | 6750 ± 3490 | 6938 ± 3669 | 6733 ± 4432 | 6600 ± 2402 | 0.92 |
| Self-reported walking activity | 1015 [446 to 2400] | 809 [322 to 2079] | 940 [280 to 3300] | 1386 [594 to 2772] | 0.23 |
| Total self-reported energy expenditure (MET-min/wk) | 2580 [1116 to 4749] | 2142 [867 to 4164] | 2550 [813 to 4183] | 3480 [1524 to 6339] | 0.31 |
| Self-reported fiber intake score | 36 ±15 | 34 ±13 | 37 ±18 | 37 ±15 | 0.75 |
| Self-reported total fat intake score | 23 ± 11 | 22 ± 10 | 22 ± 11 | 24 ± 12 | 0.63 |
| Self-reported unsaturated fat intake score | 9 ± 2 | 10 ± 1 | 9 ± 2 | 10 ± 2 | 0.36 |
| BMI (kg/m²) | 29.0 ± 4.2 | 29.4 ± 3.6 | 29.0 ± 4.3 | 28.7 ± 4.8 | 0.83 |
| Waist circumference (cm) | 102 ± 10 | 103 ± 9 | 102 ± 11 | 99 ± 12 | 0.65 |
| Weight | 80.2 ± 14.9 | 81.2 ± 14.6 | 81.2 ± 13.9 | 79.4 ± 16.4 | 0.88 |
| Systolic blood pressure (mmHg) | 143 ± 16 | 142 ± 14 | 144 ± 17 | 139 ± 15 | 0.82 |
| Diastolic blood pressure (mmHg) | 81 ± 9 | 81 ± 10 | 82 ± 8 | 79 ± 10 | 0.57 |
Table 6.1 continued

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Control</th>
<th>PREPARE minus pedometer</th>
<th>PREPARE plus pedometer</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-h Glucose (mmol/l)</td>
<td>8.3 ± 2.0</td>
<td>8.1 ± 2.0</td>
<td>8.0 ± 1.8</td>
<td>8.8 ± 2.2</td>
<td>0.33</td>
</tr>
<tr>
<td>Fasting glucose (mmol/l)</td>
<td>5.6 ± 0.5</td>
<td>5.6 ± 0.5</td>
<td>5.6 ± 0.6</td>
<td>5.6 ± 0.5</td>
<td>0.96</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>4.7 ± 1.0</td>
<td>4.7 ± 0.9</td>
<td>4.9 ± 0.9</td>
<td>4.7 ± 1.1</td>
<td>0.88</td>
</tr>
<tr>
<td>HDL-cholesterol (mmol/l)</td>
<td>1.3 [1.1 to 1.5]</td>
<td>1.3 [1.1 to 1.5]</td>
<td>1.3 [1.1 to 1.5]</td>
<td>1.2 [1.1 to 1.4]</td>
<td>0.70</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.3 [0.6 to 1.8]</td>
<td>1.3 [1.0 to 1.5]</td>
<td>1.3 [1.0 to 1.8]</td>
<td>1.4 [0.8 to 1.9]</td>
<td>0.80</td>
</tr>
<tr>
<td>Fasting insulin (uIU/ml)</td>
<td>8.8 [6.4 to 13.7]</td>
<td>8.7 [5.9 to 13.2]</td>
<td>11.2 [7.1 to 15.7]</td>
<td>7.9 [6.1 to 13.0]</td>
<td>0.35</td>
</tr>
<tr>
<td>TNFa (pg/ml)</td>
<td>1.5 [1.2 to 1.9]</td>
<td>1.5 [1.2 to 1.7]</td>
<td>1.4 [1.2 to 2.0]</td>
<td>1.6 [1.2 to 1.9]</td>
<td>0.89</td>
</tr>
<tr>
<td>IL-6 (pg/ml)</td>
<td>2.3 [1.7 to 3.9]</td>
<td>2.3 [1.6 to 4.1]</td>
<td>2.4 [1.8 to 3.9]</td>
<td>2.2 [1.7 to 3.3]</td>
<td>0.83</td>
</tr>
<tr>
<td>C-Reactive protein (mg/l)</td>
<td>1.7 [0.5 to 3.8]</td>
<td>1.7 [0.5 to 4.3]</td>
<td>0.9 [0.3 to 3.8]</td>
<td>1.9 [0.9 to 3.9]</td>
<td>0.27</td>
</tr>
</tbody>
</table>

Categorical data presented as number (column percent), parametric continuous data as mean ± SD and non-parametric data as median [interquartile range]
Table 6.2: Psychological characteristics of study participants overall and by group at baseline.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Total</th>
<th>Control</th>
<th>PREPARE</th>
<th>PREPARE plus pedometer</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Illness perceptions for IGT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consequences</td>
<td>0 [0 to 2]</td>
<td>0 [0.0 to 0]</td>
<td>0 [0.0 to 1.0]</td>
<td>1.0 [0.0 to 2.5]</td>
<td>0.72</td>
</tr>
<tr>
<td>(0 = no consequences, 10 = severe consequences)</td>
<td>2.0]</td>
<td>3.0]</td>
<td>2.5]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Timeline</td>
<td>4.0 [1.5 to 5.0]</td>
<td>3.0 [1.0 to 3.0]</td>
<td>3.0 [0.5 to 5.0]</td>
<td>4.5 [2.0 to 5.5]</td>
<td>0.34</td>
</tr>
<tr>
<td>(0 = a very short time, 10 = forever)</td>
<td>5.0]</td>
<td>5.0]</td>
<td>5.0]</td>
<td>7.0]</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>5.5 [3.0 to 8.0]</td>
<td>7.0 [3.0 to 8.0]</td>
<td>5.0 [3.5 to 5.5]</td>
<td>5.5 [3.0 to 8.0]</td>
<td>0.95</td>
</tr>
<tr>
<td>(0 = no control, 10 = complete control)</td>
<td>8.0]</td>
<td>8.0]</td>
<td>8.0]</td>
<td>8.0]</td>
<td></td>
</tr>
<tr>
<td>Treatment (physical activity) control</td>
<td>8.0 [5.0 to 10.0]</td>
<td>7.0 [5.0 to 10.0]</td>
<td>8.0 [5.5 to 10.0]</td>
<td>8.0 [5.5 to 10.0]</td>
<td>0.38</td>
</tr>
<tr>
<td>(0 = not at all effective, 10 = extremely effective)</td>
<td>10.0]</td>
<td>9.0]</td>
<td>10.0]</td>
<td>10.0]</td>
<td></td>
</tr>
<tr>
<td>Symptoms/Identity</td>
<td>0.0 [0 to 2.5]</td>
<td>0.0 [0.0 to 1.0]</td>
<td>0.0 [0.0 to 3.0]</td>
<td>0.0 [0.0 to 3.0]</td>
<td>0.33</td>
</tr>
<tr>
<td>(0 = no symptoms, 10 = many symptoms)</td>
<td>2.5]</td>
<td>1.0]</td>
<td>3.0]</td>
<td>3.0]</td>
<td></td>
</tr>
<tr>
<td>Concern</td>
<td>6.0 [4.0 to 8.0]</td>
<td>5.0 [3.0 to 8.0]</td>
<td>6.0 [2.5 to 8.0]</td>
<td>7.0 [5.0 to 9.0]</td>
<td>0.06</td>
</tr>
<tr>
<td>(0 = not at all concerned, 10 = extremely concerned)</td>
<td>8.0]</td>
<td>8.0]</td>
<td>8.0]</td>
<td>9.5]</td>
<td></td>
</tr>
<tr>
<td>Comprehension (perceived knowledge)</td>
<td>5.0 [3.0 to 5.0]</td>
<td>4.0 [1.0 to 5.0]</td>
<td>5.0 [2.0 to 5.0]</td>
<td>5.0 [4.0 to 5.0]</td>
<td>0.28</td>
</tr>
<tr>
<td>(0 = no understanding, 10 = complete understanding)</td>
<td>8.0]</td>
<td>7.0]</td>
<td>9.0]</td>
<td>7.5]</td>
<td></td>
</tr>
<tr>
<td>Emotional affect</td>
<td>2.0 [0.0 to 3.0]</td>
<td>2.0 [1.0 to 3.0]</td>
<td>5.0 [0.0 to 5.0]</td>
<td>2.0 [0.0 to 4.5]</td>
<td>0.95</td>
</tr>
<tr>
<td>(0 = no emotional affect, 10 = extreme emotional affect)</td>
<td>3.0]</td>
<td>3.0]</td>
<td>5.0]</td>
<td>4.5]</td>
<td></td>
</tr>
<tr>
<td><strong>Efficacy beliefs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking self-efficacy</td>
<td>98 [62 to 100]</td>
<td>90 [36 to 100]</td>
<td>100 [72 to 100]</td>
<td>100 [76 to 100]</td>
<td>0.29</td>
</tr>
<tr>
<td>(0 = no confidence, 100 = complete confidence)</td>
<td>100]</td>
<td>100]</td>
<td>100]</td>
<td>100]</td>
<td></td>
</tr>
<tr>
<td>Exercise self-efficacy</td>
<td>53 [21 to 63]</td>
<td>40 [0 to 56]</td>
<td>56 [18 to 63]</td>
<td>63 [34 to 93]</td>
<td>0.10</td>
</tr>
<tr>
<td>(0 = no confidence, 100 = complete confidence)</td>
<td>88]</td>
<td>67]</td>
<td>88]</td>
<td>93]</td>
<td></td>
</tr>
<tr>
<td>Self-regulatory efficacy</td>
<td>47 ± 28</td>
<td>53 ± 32</td>
<td>47 ± 28</td>
<td>50 ± 23</td>
<td>0.65</td>
</tr>
<tr>
<td>(0 = no confidence, 100 = complete confidence)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data presented as median [interquartile range] or mean ± SD
Table 6.3 shows the mean change in fasting glucose, 2-h glucose, fasting insulin, insulin resistance, markers of chronic low-grade inflammation and body weight in the study groups at 3, 6 and 12 months: the associated intervention effect for each intervention group, adjusted for baseline value, age and sex is also included. Two hour glucose decreased significantly in the EP group compared to the control group at 3 months and 12 months; at 6 months the difference in 2-h glucose between the EP group and control group was not significant, although the within-group change from baseline was significant in both groups. Fasting glucose was significantly decreased in the EP group compared to the control group at 3 months, but not at 6 or 12 months; however, compared to baseline the within-group change in fasting glucose was significant in the EP group at all follow-up time points. In the E group there was no change in either fasting or 2-h glucose compared to the control group at any time point.

There was also a significant improvement in insulin resistance in both intervention groups at 3 months, although this was only maintained in the E group at 12 months.

Although there was a small intervention effect favouring the EP group in TNFα, IL-6 and CRP at 12 months, these results failed to reach significance. No intervention effect was observed in measured markers of chronic low-grade inflammation at any time point in either group. However, at 12 months there was a significant within-group increase compared to baseline in TNFα in all groups.

There was no significant within- or between-group change in body weight at any follow-up time point.

No significant intervention effect was also observed in measured blood lipids, blood pressure or waist circumference at any time point (see Appendix Thirteen).
Table 6.3. Change from baseline and the associated intervention effect for selected biochemical and anthropometric data measured outcomes at 3, 6 and 12 months

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Intervention 1 (E group)</th>
<th>Intervention 2 (EP group)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>2-h glucose (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.08 (-.90 to 0.74)</td>
<td>0.21 (-.48 to 0.91)</td>
<td>-1.50 (-2.24 to -0.76)</td>
<td>0.18 (-0.68 to 1.04)</td>
<td>0.681</td>
<td>-1.01 (-1.95 to -0.19)</td>
<td>0.018</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.73 (-1.39 to -0.08)</td>
<td>0.00 (-.66 to 0.66)</td>
<td>-1.40 (-2.26 to -0.54)</td>
<td>0.61 (-0.23 to 1.44)</td>
<td>0.150</td>
<td>-0.35 (-1.18 to 0.48)</td>
<td>0.403</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.47 (-1.28 to 0.34)</td>
<td>0.11 (-.49 to 0.72)</td>
<td>-1.75 (-2.57 to -0.94)</td>
<td>0.43 (-0.41 to 1.28)</td>
<td>0.310</td>
<td>-0.94 (-1.79 to -0.10)</td>
<td>0.029</td>
</tr>
<tr>
<td><strong>Fasting glucose (mmol/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.00 (-0.16 to 0.16)</td>
<td>0.04 (-.13 to 0.22)</td>
<td>-0.25 (-0.43 to -0.07)</td>
<td>0.03 (-0.20 to 0.27)</td>
<td>0.788</td>
<td>-0.25 (-0.49 to -0.02)</td>
<td>0.017</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.20 (-0.35 to -0.04)</td>
<td>-0.20 (-.34 to -0.06)</td>
<td>-0.35 (-0.57 to -0.13)</td>
<td>-0.03 (-0.26 to 0.20)</td>
<td>0.787</td>
<td>-0.17 (-0.40 to 0.06)</td>
<td>0.146</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.00 (-0.22 to 0.21)</td>
<td>-0.04 (-.19 to 0.12)</td>
<td>-0.20 (-0.40 to -0.01)</td>
<td>-0.05 (-0.31 to 0.21)</td>
<td>0.700</td>
<td>-0.20 (-0.46 to 0.03)</td>
<td>0.119</td>
</tr>
<tr>
<td><strong>Fasting insulin (μU/ml)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.76 (-1.41 to 2.92)</td>
<td>-1.11 (-2.64 to 0.42)</td>
<td>-0.55 (-1.78 to 0.70)</td>
<td>-1.49 (-3.45 to 0.47)</td>
<td>0.133</td>
<td>-1.62 (-3.56 to 0.32)</td>
<td>0.100</td>
</tr>
<tr>
<td>6-months</td>
<td>0.07 (-1.22 to 1.36)</td>
<td>-1.91 (-3.93 to 0.10)</td>
<td>-0.48 (-2.39 to 1.42)</td>
<td>-1.16 (-3.34 to 1.02)</td>
<td>0.293</td>
<td>-0.41 (-2.54 to 1.72)</td>
<td>0.702</td>
</tr>
<tr>
<td>12-months</td>
<td>0.07 (-1.54 to 1.68)</td>
<td>-2.12 (-3.79 to -0.45)</td>
<td>-0.50 (-2.26 to 1.26)</td>
<td>-2.00 (-4.09 to 0.08)</td>
<td>0.060</td>
<td>-0.86 (-2.91 to 1.18)</td>
<td>0.403</td>
</tr>
<tr>
<td><strong>HOMA-IR</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.44 (0.21 to 0.68)</td>
<td>-0.21 (-0.58 to 0.15)</td>
<td>-0.24 (-0.58 to 0.10)</td>
<td>-0.56 (-1.01 to -0.11)</td>
<td>0.015</td>
<td>-0.67 (-1.12 to -0.23)</td>
<td>0.004</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.04 (-0.37 to 0.28)</td>
<td>-0.52 (-1.00 to -0.04)</td>
<td>-0.32 (-0.82 to 0.17)</td>
<td>-0.29 (-0.83 to 0.26)</td>
<td>0.297</td>
<td>-0.22 (-0.76 to 0.31)</td>
<td>0.405</td>
</tr>
<tr>
<td>12-months</td>
<td>0.01 (-0.38 to 0.39)</td>
<td>-0.70 (-1.15 to -0.24)</td>
<td>-0.35 (-0.74 to 0.04)</td>
<td>-0.63 (-1.13 to -0.14)</td>
<td>0.013</td>
<td>-0.43 (0.84 to -0.06)</td>
<td>0.087</td>
</tr>
</tbody>
</table>
### Table 6.3 continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (E group)</th>
<th>Intervention 1 (EP group)</th>
<th>Intervention 2 (EP group)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Tumor necrosis factor-α (pg/ml)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>0.09 [0.05 to 0.30]†</td>
<td>0.06 [-1.11 to 0.54]</td>
<td>0.06 [-0.20 to 0.31]</td>
<td>1.00 (0.89 to 1.14)‡</td>
<td>0.952</td>
<td>0.95 (-0.12 to 1.08)‡</td>
<td>0.431</td>
</tr>
<tr>
<td>6-months</td>
<td>0.81 [0.43 to 1.14]†</td>
<td>0.83 [0.50 to 1.09]‡</td>
<td>0.85 [0.52 to 0.92]‡</td>
<td>0.95 (0.86 to 1.05)‡</td>
<td>0.339</td>
<td>0.96 (0.87 to 1.05)‡</td>
<td>0.371</td>
</tr>
<tr>
<td>12-months</td>
<td>0.97 [0.55 to 1.29]†</td>
<td>0.86 [0.39 to 1.25]†</td>
<td>0.90 [0.67 to 1.25]†</td>
<td>0.92 (0.80 to 1.05)‡</td>
<td>0.216</td>
<td>0.96 (0.84 to 1.10)‡</td>
<td>0.567</td>
</tr>
<tr>
<td><strong>Interleukin-6 (pg/ml)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>0.06 (-0.51 to 0.62)</td>
<td>0.20 (-0.28 to 0.67)</td>
<td>0.12 (-0.32 to 0.56)</td>
<td>0.12 (-0.55 to 0.80)‡</td>
<td>0.720</td>
<td>-0.03 (-0.70 to 0.65)</td>
<td>0.939</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.59 (-1.09 to -0.09)</td>
<td>-0.03 (-0.55 to 0.48)</td>
<td>-0.30 (-0.94 to 0.33)</td>
<td>0.49 (-0.14 to 1.11)‡</td>
<td>0.127</td>
<td>-0.01 (-0.63 to 0.62)</td>
<td>0.984</td>
</tr>
<tr>
<td>12-months</td>
<td>0.00 (-0.69 to 0.69)</td>
<td>0.27 (-0.28 to 0.82)</td>
<td>0.03 (-0.46 to 0.53)</td>
<td>0.16 (-0.55 to 0.88)‡</td>
<td>0.360</td>
<td>-0.17 (-0.89 to 0.54)</td>
<td>0.358</td>
</tr>
<tr>
<td><strong>C-reactive protein (mg/l)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>-0.11 [-1.07 to 0.27]</td>
<td>0.07 [-0.55 to 1.27]</td>
<td>-0.25 [-0.91 to 0.16]</td>
<td>1.53 (0.93 to 2.54)‡</td>
<td>0.094</td>
<td>0.97 (0.60 to 1.58)‡</td>
<td>0.904</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.22 [-1.17 to 0.27]</td>
<td>-0.09 [-0.81 to 0.08]</td>
<td>-0.06 [-1.06 to 0.46]</td>
<td>1.20 (0.64 to 2.26)‡</td>
<td>0.562</td>
<td>1.10 (0.59 to 2.05)‡</td>
<td>0.761</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.05 [-1.04 to 0.66]</td>
<td>-0.13 [-0.67 to 0.35]</td>
<td>-0.29 [-1.18 to 0.06]</td>
<td>1.00 (0.53 to 1.88)‡</td>
<td>0.991</td>
<td>0.89 (0.48 to 1.64)‡</td>
<td>0.694</td>
</tr>
<tr>
<td><strong>Weight (kg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>-0.69 (-2.01 to 0.67)</td>
<td>0.24 (-0.83 to 1.32)</td>
<td>-0.36 (-1.40 to 0.68)</td>
<td>0.90 (-0.74 to 2.54)‡</td>
<td>0.278</td>
<td>0.32 (-1.31 to 1.94)</td>
<td>0.698</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.57 (-1.48 to 0.34)</td>
<td>-0.17 (-1.34 to 1.00)</td>
<td>-0.61 (-1.84 to 0.61)</td>
<td>0.30 (-1.28 to 1.88)‡</td>
<td>0.708</td>
<td>-0.03 (-1.60 to 1.54)</td>
<td>0.972</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.82 (-2.22 to 0.59)</td>
<td>-0.57 (-1.93 to 0.80)</td>
<td>0.49 (-0.91 to 1.89)</td>
<td>0.18 (-1.80 to 2.12)‡</td>
<td>0.854</td>
<td>1.34 (-0.62 to 3.30)</td>
<td>0.177</td>
</tr>
</tbody>
</table>

Data displayed as mean (95% confidence interval) unless stated otherwise.

All reported intervention effects were adjusted for age, sex and baseline value.

* = variables with non-parametric distribution (displayed as median [interquartile range]), † = non-parametric data significantly different (p<0.05) from baseline (Wilcoxon signed rank test), ‡ = data displayed as a dimensionless ratio of geometric means; 1 represents no intervention effect and < 1 represents an intervention effect favouring the intervention
**Physical Activity and Diet**

Valid pedometer data was available for 78 participants (94%) at baseline; of these 70 (84%), 75 (90%) and 75 participants (90%) had valid data at 3-months, 6-months and 12-months respectively.

Complete IPAQ data was available for 76 participants (91%) at baseline; of these 69 (83%), 73 (88%) and 75 (90%) individuals had complete data at 3-months, 6-months and 12-months respectively.

Complete dietary data was available for 81 individuals (98%) at baseline: of these 75 (90%), 76 (92%) and 80 (96%) participants had complete dietary data at 3-months, 6-months and 12-months respectively.

Table 6.4 shows the change in pedometer counts and self-reported walking and moderate-to-vigorous-intensity physical activity, fibre intake, total fat intake and unsaturated fat intake at 3, 6 and 12 months; the associated intervention effect for each intervention group, adjusted for baseline value, age and sex, is also shown. Ambulatory activity and self-reported walking and overall moderate- to vigorous-intensity physical activity increased significantly in the EP group compared to the control group at 3, 6 and 12 months. In the E group, ambulatory activity and self-reported moderate- to vigorous-intensity physical activity increased significantly compared to the control group at 12 months; however there was no significant increase in self-reported walking activity and no significant within-group change in ambulatory activity compared to baseline. There was also no significant increase in any measure of physical activity in the E group compared to the control group at 3 or 6 months.

Individuals in the EP group reported a significant increase in fibre intake at 3 months compared to the control group. Those in the E group reported a significant decrease in total fat intake at 12 months compared to the control group. No other significant changes in measured dietary variables were observed at any time point.
Table 6.4. Change from baseline and the associated intervention effect for measures of physical activity and diet at 3, 6 and 12-months.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (E group)</th>
<th>Intervention 1 (EP group)</th>
<th>Intervention 2 (EP group)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ambulatory activity (steps/day)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>615 (-312 to 1541)</td>
<td>1198 (350 to 2045)</td>
<td>2395 (1285 to 3505)</td>
<td>672 (-741 to 2084)</td>
<td>0.346</td>
<td>1720 (368 to 3073)</td>
<td>0.013</td>
</tr>
<tr>
<td>6-months</td>
<td>-105 (-780 to 591)</td>
<td>882 (-36 to 1799)</td>
<td>2093 (944 to 3242)</td>
<td>904 (-413 to 2221)</td>
<td>0.175</td>
<td>2163 (902 to 3424)</td>
<td>0.001</td>
</tr>
<tr>
<td>12-months</td>
<td>-965 (-1680 to -251)</td>
<td>573 (-304 to 1449)</td>
<td>1039 (135 to 1943)</td>
<td>1480 (436 to 2522)</td>
<td>0.006</td>
<td>1952 (953 to 2951)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Self-reported walking activity (MET.hours/week)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>334 (-414 to 1082)</td>
<td>412 (-190 to 1014)</td>
<td>1605 (712 to 2498)</td>
<td>37 (-1077 to 1151)</td>
<td>0.948</td>
<td>1400 (363 to 2437)</td>
<td>0.009</td>
</tr>
<tr>
<td>6-months</td>
<td>172 (-675 to 1018)</td>
<td>78 (-677 to 832)</td>
<td>1083 (517 to 1649)</td>
<td>463 (-1078 to 771)</td>
<td>0.742</td>
<td>889 (23 to 1755)</td>
<td>0.044</td>
</tr>
<tr>
<td>12-months</td>
<td>-379 (-925 to 167)</td>
<td>358 (-306 to 1021)</td>
<td>708 (72 to 1344)</td>
<td>681 (-137 to 1499)</td>
<td>0.101</td>
<td>1139 (366 to 1912)</td>
<td>0.004</td>
</tr>
<tr>
<td>Total moderate- to vigorous-intensity physical activity (MET.hours/week)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>245 (-2124 to 2712)</td>
<td>755 (-1098 to 2610)</td>
<td>3403 (1214 to 5991)</td>
<td>404 (-2446 to 3255)</td>
<td>0.778</td>
<td>3534 (847 to 6223)</td>
<td>0.011</td>
</tr>
<tr>
<td>6-months</td>
<td>488 (-1082 to 2380)</td>
<td>1291 (-506 to 3188)</td>
<td>3830 (1637 to 6024)</td>
<td>667 (-2008 to 3242)</td>
<td>0.620</td>
<td>3230 (670 to 5760)</td>
<td>0.013</td>
</tr>
<tr>
<td>12-months</td>
<td>-1460 (-3129 to 210)</td>
<td>1202 (177 to 2228)</td>
<td>1589 (48 to 3130)</td>
<td>2183 (203 to 4163)</td>
<td>0.031</td>
<td>3045 (1194 to 4896)</td>
<td>0.002</td>
</tr>
</tbody>
</table>
Table 6.4 continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (E group)</th>
<th>Intervention 1 (EP group)</th>
<th>Intervention 2 (EP group)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibre Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-0.7 (-5.0 to 3.6)</td>
<td>-2.2 (-7.0 to 2.6)</td>
<td>4.9 (-0.2 to 9.9)</td>
<td>-1.1 (-7.4 to 5.2)</td>
<td>0.738</td>
<td>6.5 (0.4 to 12.6)</td>
<td>0.037</td>
</tr>
<tr>
<td>6-months</td>
<td>-1.5 (-5.3 to 2.2)</td>
<td>0.2 (-5.0 to 5.4)</td>
<td>3.1 (-2.8 to 9.1)</td>
<td>4.0 (-2.3 to 10.2)</td>
<td>0.214</td>
<td>6.1 (-0.2 to 12.4)</td>
<td>0.058</td>
</tr>
<tr>
<td>12-months</td>
<td>0.9 (-6.3 to 8.0)</td>
<td>-1.6 (-8.1 to 4.8)</td>
<td>1.8 (-3.6 to 7.2)</td>
<td>-0.3 (-7.4 to 6.6)</td>
<td>0.912</td>
<td>3.7 (-3.4 to 10.7)</td>
<td>0.306</td>
</tr>
<tr>
<td>Total Fat Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.1 (-3.9 to 4.1)</td>
<td>-1.8 (-5.1 to 1.5)</td>
<td>-0.8 (-3.6 to 2.0)</td>
<td>-1.3 (-5.4 to 2.8)</td>
<td>0.537</td>
<td>-0.0 (-4.0 to 4.0)</td>
<td>0.990</td>
</tr>
<tr>
<td>6-months</td>
<td>1.0 (3.9 to 5.8)</td>
<td>0.4 (-3.6 to 4.5)</td>
<td>1.4 (-2.6 to 5.4)</td>
<td>-0.1 (-5.7 to 5.4)</td>
<td>0.961</td>
<td>0.8 (-4.7 to 6.3)</td>
<td>0.770</td>
</tr>
<tr>
<td>12-months</td>
<td>2.0 (-1.8 to 5.7)</td>
<td>-2.0 (-5.8 to 1.9)</td>
<td>-1.5 (-5.0 to 2.1)</td>
<td>-4.4 (-8.3 to -0.5)</td>
<td>0.029</td>
<td>-1.9 (-5.8 to 2.0)</td>
<td>0.335</td>
</tr>
<tr>
<td>Unsaturated Fat Score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.4 (-0.2 to 0.9)</td>
<td>0.5 (-0.1 to 1.1)</td>
<td>0.6 (-0.0 to 1.3)</td>
<td>-0.2 (-0.8 to 0.4)</td>
<td>0.553</td>
<td>0.2 (-0.5 to 0.8)</td>
<td>0.598</td>
</tr>
<tr>
<td>6-months</td>
<td>0.4 (-0.2 to 1.0)</td>
<td>0.5 (0.1 to 1.0)</td>
<td>0.6 (-0.1 to 1.3)</td>
<td>0.2 (-0.7 to 1.1)</td>
<td>0.676</td>
<td>0.3 (-0.6 to 1.1)</td>
<td>0.557</td>
</tr>
<tr>
<td>12-months</td>
<td>0.6 (-0.3 to 1.5)</td>
<td>0.4 (-0.4 to 1.1)</td>
<td>0.5 (-0.2 to 1.2)</td>
<td>-0.5 (-1.6 to 0.7)</td>
<td>0.411</td>
<td>-0.2 (-1.3 to 0.9)</td>
<td>0.715</td>
</tr>
</tbody>
</table>

Data displayed as mean (95% confidence interval)

All reported intervention effects were adjusted for age, sex and baseline value.
Psychological variables

Complete perception and knowledge of IGT data was available for 77 participants (93%) at baseline; of these 71 (86%), 72 (87%) and 76 (92%) participants had complete data at 3-month, 6-months and 12-months respectively.

Complete self-efficacy data was available for 80 participants (96%) at baseline; of these 74 (89%), 76 (92%) and 79 (95%) participants had complete data at 3-month, 6-months and 12-months respectively.

Table 6.5 shows the change in illness perceptions, perceived knowledge of IGT, emotional representations due to IGT and measured efficacy beliefs at 3, 6 and 12 months: the associated intervention effect for each intervention group, adjusted for baseline value, age and sex is also included. Both intervention groups positively influenced perceived knowledge of IGT, perceived effectiveness of exercise as a treatment for IGT and measured efficacy beliefs compared to the control group at 12 months.
Table 6.5. Change from baseline and the associated intervention effect for illness perceptions and efficacy beliefs at 3, 6 and 12-months

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control (PREPARE)</th>
<th>Intervention 1 (PREPARE + pedometer)</th>
<th>Intervention 2 (PREPARE + pedometer)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consequences</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-0.7 (-1.6 to 0.1)</td>
<td>0.0 (-0.8 to 0.9)</td>
<td>0.1 (-0.8 to 1.1)</td>
<td>0.7 (-0.2 to 1.7)</td>
<td>0.130</td>
<td>1.0 (0.1 10 1.9)</td>
<td>0.034</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.1 (-0.9 to 1.2)</td>
<td>0.3 (-0.2 to 0.9)</td>
<td>-0.1 (-0.9 to 0.6)</td>
<td>0.3 (-0.7 to 1.2)</td>
<td>0.556</td>
<td>-0.1 (-1.0 to 0.8)</td>
<td>0.844</td>
</tr>
<tr>
<td>12-months</td>
<td>0.2 (-1.1 to 1.4)</td>
<td>-0.5 (-0.3 to 1.3)</td>
<td>-0.0 (-1.0 to 0.9)</td>
<td>0.4 (-0.8 to 1.6)</td>
<td>0.479</td>
<td>0.1 (-1.0 to 1.3)</td>
<td>0.842</td>
</tr>
<tr>
<td>Timeline</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.5 (-1.0 to 2.1)</td>
<td>-0.0 (-1.4 to 1.3)</td>
<td>0.3 (-1.0 to 1.6)</td>
<td>-0.4 (-2.1 to 1.3)</td>
<td>0.671</td>
<td>0.1 (-1.5 to 1.7)</td>
<td>0.907</td>
</tr>
<tr>
<td>6-months</td>
<td>0.9 (-0.8 to 2.5)</td>
<td>-0.2 (-1.8 to 1.4)</td>
<td>0.3 (-1.4 to 2.1)</td>
<td>-0.7 (-2.8 to 1.5)</td>
<td>0.539</td>
<td>0.0 (-2.0 to 2.1)</td>
<td>0.970</td>
</tr>
<tr>
<td>12-months</td>
<td>1.8 (0.2 to 3.4)</td>
<td>-0.4 (-1.7 to 0.9)</td>
<td>0.5 (-0.9 to 1.8)</td>
<td>-2.0 (-3.8 to -0.1)</td>
<td>0.035</td>
<td>-0.7 (-2.5 to 1.1)</td>
<td>0.429</td>
</tr>
<tr>
<td>Personal control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.9 (-0.6 to 2.3)</td>
<td>1.2 (-0.2 to 2.6)</td>
<td>0.3 (-0.5 to 1.1)</td>
<td>0.1 (-1.4 to 1.5)</td>
<td>0.928</td>
<td>-0.8 (-2.2 to 0.6)</td>
<td>0.270</td>
</tr>
<tr>
<td>6-months</td>
<td>0.4 (-1.3 to 2.0)</td>
<td>1.7 (0.4 to 3.1)</td>
<td>0.7 (-0.4 to 1.8)</td>
<td>1.5 (0.2 to 2.9)</td>
<td>0.026</td>
<td>0.6 (-0.7 to 1.9)</td>
<td>0.351</td>
</tr>
<tr>
<td>12-months</td>
<td>0.8 (-0.9 to 2.5)</td>
<td>1.9 (0.6 to 3.1)</td>
<td>1.2 (0.2 to 2.2)</td>
<td>1.0 (-0.3 to 2.4)</td>
<td>0.131</td>
<td>0.8 (-0.5 to 2.1)</td>
<td>0.249</td>
</tr>
<tr>
<td>Treatment control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>1.1 (-0.0 to 2.1)</td>
<td>0.8 (0.2 to 1.4)</td>
<td>0.7 (0.0 to 1.4)</td>
<td>0.2 (-0.7 to 1.0)</td>
<td>0.713</td>
<td>-0.0 (-0.8 to 0.8)</td>
<td>0.935</td>
</tr>
<tr>
<td>6-months</td>
<td>0.2 (-0.8 to 1.2)</td>
<td>1.2 (0.4 to 2.0)</td>
<td>1.2 (0.3 to 2.2)</td>
<td>1.6 (0.7 to 2.5)</td>
<td>0.001</td>
<td>1.7 (0.9 to 2.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>12-months</td>
<td>0.6 (-0.3 to 1.4)</td>
<td>1.2 (0.4 to 2.0)</td>
<td>1.3 (0.4 to 2.1)</td>
<td>1.0 (0.2 to 1.7)</td>
<td>0.016</td>
<td>1.1 (0.3 to 1.9)</td>
<td>0.005</td>
</tr>
</tbody>
</table>
Table 6.5 continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Intervention 1 (PREPARE)</th>
<th>Intervention 2 (PREPARE + pedometer)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.1 (-0.5 to 0.7)</td>
<td>-0.5 (-1.2 to 0.2)</td>
<td>-0.9 (-1.8 to -0.0)</td>
<td>-0.2 (-1.0 to 0.6)</td>
<td>0.616</td>
<td>-0.5 (-1.3 to 0.3)</td>
<td>0.215</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.0 (-0.6 to 0.5)</td>
<td>-0.9 (-1.5 to 0.2)</td>
<td>-1.0 (-1.8 to -0.1)</td>
<td>-0.4 (-1.2 to 0.3)</td>
<td>0.273</td>
<td>-0.4 (-1.2 to 0.3)</td>
<td>0.245</td>
</tr>
<tr>
<td>12-months</td>
<td>0.3 (-0.4 to 0.9)</td>
<td>-0.8 (-1.8 to 0.2)</td>
<td>-0.7 (-1.5 to 0.0)</td>
<td>-0.6 (-1.6 to 0.4)</td>
<td>0.219</td>
<td>-0.6 (-1.6 to 0.4)</td>
<td>0.228</td>
</tr>
<tr>
<td><strong>Perceived knowledge</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>0.6 (-1.3 to 2.4)</td>
<td>2.0 (0.3 to 3.8)</td>
<td>1.1 (0.2 to 2.1)</td>
<td>2.0 (0.4 to 3.6)</td>
<td>0.017</td>
<td>1.4 (-0.2 to 3.0)</td>
<td>0.078</td>
</tr>
<tr>
<td>6-months</td>
<td>1.2 (-0.2 to 2.6)</td>
<td>2.3 (0.6 to 3.9)</td>
<td>1.7 (0.4 to 3.0)</td>
<td>1.9 (0.2 to 3.4)</td>
<td>0.019</td>
<td>1.7 (0.2 to 3.3)</td>
<td>0.024</td>
</tr>
<tr>
<td>12-months</td>
<td>0.9 (-0.6 to 2.5)</td>
<td>2.2 (0.5 to 3.8)</td>
<td>2.5 (1.7 to 3.4)</td>
<td>1.8 (0.4 to 3.4)</td>
<td>0.013</td>
<td>2.5 (1.1 to 3.9)</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Concern at having IGT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-0.5 (-2.1 to 1.2)</td>
<td>0.0 (-1.4 to 1.4)</td>
<td>0.3 (-0.7 to 1.2)</td>
<td>0.7 (-1.0 to 2.4)</td>
<td>0.414</td>
<td>1.6 (-0.1 to 3.3)</td>
<td>0.067</td>
</tr>
<tr>
<td>6-months</td>
<td>0.4 (-1.5 to 2.3)</td>
<td>2.3 (0.3 to 4.3)</td>
<td>-0.4 (-1.5 to 0.7)</td>
<td>1.8 (0.4 to 3.3)</td>
<td>0.015</td>
<td>1.2 (-0.3 to 2.6)</td>
<td>0.118</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.3 (-1.6 to 0.9)</td>
<td>-0.7 (-1.5 to 0.2)</td>
<td>-0.7 (-2.0 to 0.7)</td>
<td>-0.2 (-1.7 to 1.4)</td>
<td>0.826</td>
<td>0.2 (-1.4 to 1.6)</td>
<td>0.827</td>
</tr>
<tr>
<td><strong>Negative emotional affect attributed to having IGT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-1.3 (-2.7 to -0.1)</td>
<td>-0.0 (-0.7 to -0.6)</td>
<td>-0.3 (-1.1 to 0.5)</td>
<td>1.1 (-0.0 to 2.2)</td>
<td>0.050</td>
<td>1.0 (-0.2 to 2.0)</td>
<td>0.078</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.5 (-2.1 to 1.2)</td>
<td>-0.2 (-0.7 to 0.3)</td>
<td>-0.7 (-1.4 to -0.1)</td>
<td>0.1 (-1.0 to 1.2)</td>
<td>0.874</td>
<td>-0.3 (-1.4 to 0.8)</td>
<td>0.569</td>
</tr>
<tr>
<td>12-months</td>
<td>-0.3 (-1.8 to 1.3)</td>
<td>-0.2 (1.1 to 0.7)</td>
<td>0.1 (-0.6 to 0.9)</td>
<td>0.0 (-1.3 to 1.4)</td>
<td>0.960</td>
<td>0.4 (-0.9 to 1.7)</td>
<td>0.543</td>
</tr>
</tbody>
</table>

115
Table 6.5 continued

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Intervention 1 (PREPARE)</th>
<th>Intervention 2 (PREPARE + pedometer)</th>
<th>Adjusted Intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted Intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking self-efficacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-2 (-10 to 6)</td>
<td>-9 (-20 to 3)</td>
<td>5 (-1 to 11)</td>
<td>-2 (-13 to 10)</td>
<td>0.767</td>
<td>13 (1 to 24)</td>
<td>0.032</td>
</tr>
<tr>
<td>6-months</td>
<td>-2 (-7 to 3)</td>
<td>-5 (-14 to 05)</td>
<td>2 (-5 to 9)</td>
<td>1 (9 to 11)</td>
<td>0.799</td>
<td>10 (-0.0 to 20)</td>
<td>0.059</td>
</tr>
<tr>
<td>12-months</td>
<td>-8 (-14 to -1)</td>
<td>-1 (-8 to 6)</td>
<td>1 (-6 to 8)</td>
<td>10 (1 to 19)</td>
<td>0.034</td>
<td>14 (5 to 23)</td>
<td>0.004</td>
</tr>
<tr>
<td>Exercise (non-walking) self-efficacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-2 (-20 to 17)</td>
<td>-7 (-16 to 3)</td>
<td>4 (-8 to 17)</td>
<td>5 (-13 to 23)</td>
<td>0.592</td>
<td>16 (-1 to 33)</td>
<td>0.066</td>
</tr>
<tr>
<td>6-months</td>
<td>4 (-16 to 3)</td>
<td>-5 (-15 to 6)</td>
<td>-1 (-14 to 12)</td>
<td>-1 (17 to 15)</td>
<td>0.926</td>
<td>6 (-0.0 to 20)</td>
<td>0.465</td>
</tr>
<tr>
<td>12-months</td>
<td>-8 (-20 to 5)</td>
<td>0 (-9 to 10)</td>
<td>-3 (-17 to 12)</td>
<td>15 (0 to 30)</td>
<td>0.046</td>
<td>17 (2 to 32)</td>
<td>0.030</td>
</tr>
<tr>
<td>Self-regulatory efficacy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-7 (-18 to 4)</td>
<td>1 (-9 to 11)</td>
<td>7 (-0 to 15)</td>
<td>10 (-1 to 23)</td>
<td>0.082</td>
<td>17 (5 to 29)</td>
<td>0.005</td>
</tr>
<tr>
<td>6-months</td>
<td>1 (-6 to 7)</td>
<td>7 (-4 to 17)</td>
<td>6 (-2 to 13)</td>
<td>8 (-2 to 19)</td>
<td>0.126</td>
<td>8 (-2 to 19)</td>
<td>0.120</td>
</tr>
<tr>
<td>12-months</td>
<td>-7 (-19 to 6)</td>
<td>7 (-4 to 17)</td>
<td>7 (-3 to 16)</td>
<td>14 (0 to 27)</td>
<td>0.046</td>
<td>14 (1 to 28)</td>
<td>0.037</td>
</tr>
</tbody>
</table>

Data displayed as mean (95% confidence interval)

All reported intervention effects were adjusted for age, sex and baseline value.
Correlations

Table 6.6 shows the partial correlation coefficients between change in 2-h glucose, fasting glucose, insulin resistance and markers of chronic low-grade inflammation and change in measured lifestyle variables, after adjustment for age and sex. Although change in 2-h glucose and change in steps per day were inversely correlated, this failed to reach significance (r = 0.20, p = 0.089). However, change in steps per day was significantly inversely correlated with change in fasting glucose and TNFα. Change in self-reported total fat intake was correlated with change in fasting glucose. These correlations remained significant after further adjustment for treatment group.

In order to assess the independent association of change in total fat intake and steps per day with change in fasting glucose, further partial correlation analysis was conducted. The independent association of change in steps per day with change in fasting glucose was assessed by controlling for age, sex and change in total fat intake. The independent association of change in total fat intake with change in fasting glucose was assessed using the same method. Both associations were somewhat attenuated, although the association of change in fasting glucose with change in steps per day remained significant (r = -0.26, p = 0.029), whereas the association with total fat intake did not (r = 0.23, p = 0.051).

Partial correlation analysis adjusted for age and sex revealed a significant correlation between change in steps per day and change self-reported walking activity (r = 0.39, P = 0.001) and change in self-reported total moderate- to vigorous-intensity physical activity (r = 0.30, p = 0.015) at 12 months.

Change in perceived knowledge of IGT was correlated with change in self-reported moderate- to vigorous-intensity physical activity after adjustment for age and sex (r = 0.29, p = 0.018). Change in self-reported walking activity was significantly correlated with change walking self-efficacy after adjustment for age and sex (r = 0.38, p = 0.002). No other significant associations were observed between change in measures of physical activity and change in measured psychological variables.
Table 6.6: Partial correlation coefficients, adjusted for age and sex, showing the strength of the association of change in measures of physical activity and diet with selected biochemical variables at 12-months.

<table>
<thead>
<tr>
<th>Variable</th>
<th>2-h glucose</th>
<th>Fasting glucose resistance</th>
<th>Insulin resistance</th>
<th>Log$_{10}$ Tumor necrosis factor-α</th>
<th>Interleukin-6</th>
<th>Log$_{10}$ C-reactive protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pedometer counts</td>
<td>-0.196</td>
<td>-0.274</td>
<td>-0.101</td>
<td>-0.278</td>
<td>-0.195</td>
<td>-0.083</td>
</tr>
<tr>
<td>P value</td>
<td>0.089</td>
<td>0.017</td>
<td>0.411</td>
<td>0.020</td>
<td>0.105</td>
<td>0.483</td>
</tr>
<tr>
<td>Self-reported walking activity</td>
<td>-0.024</td>
<td>-0.043</td>
<td>-0.072</td>
<td>-0.243</td>
<td>-0.162</td>
<td>-0.005</td>
</tr>
<tr>
<td>P value</td>
<td>0.843</td>
<td>0.725</td>
<td>0.571</td>
<td>0.050</td>
<td>0.193</td>
<td>0.970</td>
</tr>
<tr>
<td>Total moderate- to vigorous-intensity physical activity</td>
<td>0.033</td>
<td>-0.104</td>
<td>-0.193</td>
<td>-0.047</td>
<td>0.143</td>
<td>-0.004</td>
</tr>
<tr>
<td>P value</td>
<td>0.785</td>
<td>0.393</td>
<td>0.127</td>
<td>0.708</td>
<td>0.255</td>
<td>0.974</td>
</tr>
<tr>
<td>Total fat score</td>
<td>-0.117</td>
<td>0.251</td>
<td>0.202</td>
<td>-0.034</td>
<td>0.027</td>
<td>0.033</td>
</tr>
<tr>
<td>P value</td>
<td>0.307</td>
<td>0.028</td>
<td>0.092</td>
<td>0.778</td>
<td>0.819</td>
<td>0.779</td>
</tr>
<tr>
<td>Total Fibre score</td>
<td>0.000</td>
<td>0.120</td>
<td>-0.085</td>
<td>-0.129</td>
<td>0.045</td>
<td>0.198</td>
</tr>
<tr>
<td>P value</td>
<td>0.999</td>
<td>0.299</td>
<td>0.481</td>
<td>0.280</td>
<td>0.707</td>
<td>0.087</td>
</tr>
</tbody>
</table>
Discussion

This study found that group-based structured education aimed at promoting increased physical activity is effective at positively influencing efficacy beliefs surrounding walking activity and perceived knowledge of IGT and increasing ambulatory activity compared to control conditions after 12 months. Furthermore, participants who were enabled, as part of the education programme, to set personalised step per day goals and to self-monitor their daily ambulatory activity with a pedometer had significant improvements in glucose tolerance.

Glucose control and markers of chronic low-grade inflammation

At 12 months, 2-h glucose had decreased by -1.75 mmol/l (95% CI -2.57 to -0.94) in the EP group compared to -0.47 mmol/l (95% CI -1.28 to 0.34) in the control group. The resulting intervention effect, after adjustment for age, sex and baseline value, of -0.94 mmol/l (95% CI -1.79 to -0.10) is similar to that reported in previous diabetes prevention programmes. For example, the Finnish Diabetes Prevention Study reported a mean decrease in 2-h glucose of -0.9 mmol/l in the intervention group compared to a mean decrease of -0.3 mmol/l in the control group (Lindström et al. 2003) and a diabetes prevention trial in the Netherlands reported a mean decrease of -0.8 mmol/l in the intervention group compared to a mean increase of 0.2 mmol/l in the control group (Mensink et al. 2003b). A recent meta-analysis of eight diabetes prevention studies promoting lifestyle change reported an overall intervention effect in 2-h glucose of -0.84 mmol/l (95% CI -1.29 to -0.39) at 12 months and a reduction in relative risk of developing type 2 diabetes of 0.55 (95% CI 0.44 to 0.69) (Yamaoka & Tango 2005). However, the PREPARE programme is the only lifestyle intervention study in free-living adults with prediabetes in the United Kingdom to show significant improvements in glucose tolerance. A study in Newcastle Upon Tyne testing the efficacy of lifestyle counselling in those with IGT found that whilst the intervention was successful at improving insulin sensitivity after 12 months, this did not translate into significant improvements in either fasting or 2-h glucose (Oldroyd et al. 2006). Similarly, a multi-centred study investigating the effect of reinforced healthy-living in France and England in those with impaired fasting glucose did not find improvements in glucose control or insulin sensitivity after 12 months (Dyson et al. 1997).
Although small reductions were seen in all markers of chronic low-grade inflammation in the EP group compared to the control group at 12-months, these differences failed to reach significance and were smaller than that predicted by the cross-sectional study detailed in Chapter Three and other epidemiological studies (Panagiotakos et al. 2005, Pischon et al. 2003, Wannamethee et al. 2002). However, other exercise training studies have also tended to show that the effect of exercise training on inflammatory markers is weaker than predicted by epidemiological research (Giannopoulou et al. 2005, Lakka et al. 2005, Marcell et al. 2005, Niebauer et al. 2005, Polak et al. 2006, Rokling-Andersen et al. 2007), suggesting that the results from epidemiological research may be biased by confounding factors that were either not measured or controlled for. The largest exercise training study to date to investigate the effect of exercise on markers of chronic low-grade inflammation found that exercise training did not result in reductions in CRP after 20 weeks, although sub-group analysis found that the intervention resulted in significant reductions in CRP in those with high (> 3.0 mg/l) baseline levels (Lakka et al. 2005). Another recent, well designed study found that exercise training with or without dietary intervention actually resulted in significant increases in TNFα when compared to control conditions (Rokling-Andersen et al. 2007). However, whilst no intervention effect was detected in the present study at any follow-up time point, there was a significant and inverse association of change in steps per day and change in self-reported walking activity with change in TNFα after 12 months, suggesting small decreases in TNFα may be obtained through increased walking activity. As intervention studies investigating the effect of exercise on markers of chronic low-grade inflammation are characterised by small sample sizes, more research is needed from adequately powered trials.

**Physical activity**

The PREPARE programme study reported a significant increase in ambulatory activity of 1039 steps per day (95% CI 135 to 1943) in the EP and an increase of 573 (95% CI -304 to 1449) steps per day in the E group compared to a decrease of 965 steps per day (95% CI -1680 to -251) in the control group at 12-months. The resulting intervention effect in the EP group, after adjustment for age, sex and baseline value, of 1952 steps per day (95% CI 953 to 2951) equates to around 140 minutes of walking activity per week (Tudor-Locke & Bassett 2004); the intervention effect in the E group of 1480 (95% CI 436 to 2522) equates to around 100 minutes of walking activity per week (Tudor-Locke & Bassett 2004).
The increase in pedometer counts and self-reported physical activity seen in the EP group was greater than that reported in other lifestyle interventions in those with IGT (see chapter 2). For example, the Diabetes Prevention Program reported an intervention effect in self-reported leisure time physical activity of around 7 MET-hours/week (equivalent to 127 minutes of walking activity per week) at 12-months (Knowler et al. 2002), while the Finnish Diabetes Prevention Study did not find a significant difference in overall self-reported leisure time physical activity change at 12 months (Lindström et al. 2003). Similarly, most — although not all (Kirk et al. 2004) — lifestyle or physical activity intervention studies in the United Kingdom in a variety of different settings have been unsuccessful at promoting increased physical activity and/or cardiovascular fitness after 12 months (Dyson et al. 1997, Harland et al. 1999, Hillsdon et al 2002, Kinmonth et al. 2008, Oldroyd et al. 2006). For example, a recent randomized controlled trial in a community setting in those with a family history of type 2 diabetes found that one-to-one counselling designed to promote self-regulatory skills was no more effective at promoting physical activity than providing participants with an information leaflet (Kinmonth et al. 2008); another study concluded that motivational interviewing or direct advice were ineffective at promoting physical activity in a health care setting (Hillsdon et al 2002).

All groups in this study experienced a decline in ambulatory activity after 3-months, with the control group achieving a significant decrease in ambulatory activity at 12 months compared to baseline. Although physical activity levels are known to decrease with age (Department of Health 2004a), the pattern of change in ambulatory activity observed in this study may have also been influenced by a decline in the behavioural reactivity associated with wearing a measurement pedometer over time (Clemes et al. 2008). An earlier free-living physical activity intervention study conducted over 12 months in individuals with type 2 diabetes which also included the objective measurement of physical activity and multiple follow-up time points also found a significant decline in physical activity in the control group over the course of the study (Kirk et al. 2004).

**Mechanisms**

Previous diabetes prevention programmes such as the Diabetes Prevention Program and the Finnish Diabetes Prevention Study used diet and physical activity with the aim of achieving
weight loss. In contrast the primary aim of the PREPARE programme was to promote physical activity. This is an important distinction because it has been hypothesised that the success of previous diabetes prevention programmes have been largely attributable to weight loss (see Chapter Two). For example, a recent study concluded that weight loss was the dominant determinant of the reduced risk of developing type 2 diabetes observed in the Diabetes Prevention Program and therefore interventions designed to reduce the risk of type 2 diabetes should primarily target weight loss (Hamman et al. 2006). However, the PREPARE programme study suggests that interventions that successfully promote physical activity to levels that are consistent with the current physical activity recommendations in inactive individuals with IGT are as effective at improving glucose tolerance as holistic lifestyle interventions aimed at weight loss. This is important because it has been recommended that around 60 minutes per day of moderate-intensity physical activity is needed to achieve long-term energy balance (Brooks et al 2004), an amount which most individuals are likely to be unable or unwilling to achieve. Therefore the PREPARE programme study further emphasises the importance of promoting physical activity for its own sake, regardless of whether it results in weight loss.

This conclusion is consistent with numerous proven mechanisms linking physical activity to reduced insulin resistance and improved glucose control that are independent of body weight. For example, acute exercise and exercise training result in increased insulin-dependant and insulin-independent GLUT4 translocation, which facilitates the diffusion of circulating glucose into muscle cells, and an improved ability to oxidise fatty acid in skeletal muscle (Hawley 2004, Ivy et al. 1999, Zierath 2002). The latter mechanism is important because insulin resistance is characterised by an accumulation of intra-muscular fat and an inability to oxidize fatty acids (Bruce & Hawley 2004).

In the present study, change in 2-h glucose was inversely correlated with change in steps per day at 12 months, although this association just failed to reach significance (r = 0.20, p = 0.089). However, given the poor repeatability of 2-h glucose (Ko et al. 1998, Mooy et al. 1996), this study was insufficiently powered to detect an association between change in 2-h glucose and change in physical activity. Change in fasting glucose, which has superior repeatability compared to 2-h glucose (Mooy et al. 1996), was significantly correlated with change in steps per day at 12 months (r = -0.27, p = 0.017).
Results for insulin resistance seen in this study preclude the deduction of a clear mechanism linking increased physical activity to improvements in glucose tolerance in the EP group. Change in insulin resistance in the EP group at 12 months failed to reach significance (p = 0.087), whereas the E group achieved a reduction in insulin resistance despite no significant change in glucose tolerance. Given the positive association of fat intake with fasting glucose and insulin resistance and the fact that self-reported fat intake decreased in the E group, it is likely that the decrease in insulin resistance seen the E group was caused, at least in part, by dietary modification. However, caution needs to be applied when interpreting the results for insulin resistance. Although the method of measuring insulin resistance used in this study, HOMA-IR, has been shown to correlate well with gold standard methods of measuring insulin resistance in those with normal glucose control and those with type 2 diabetes (Bonora et al. 2000, Stumvoll et al. 2000, Yokoyama et al. 2003), several studies have shown it to be a poor marker of insulin resistance in those with IGT (Anderson et al. 1995, Kang et al. 2005), particularly in the elderly (Ferrara & Goldberg 2001). One of the key assumptions underlying the HOMA-IR model is that fasting glucose and insulin concentrations reflect the normal insulin secretory response after a glucose challenge, which may not necessarily be true for those with IGT (Ferrara & Goldberg 2001, Kang et al. 2005).

**Pedometer**

As sustained increases in physical activity and improvements in glucose tolerances were only seen in the EP group, the PREPARE programme study proved the stated secondary hypothesis by showing that structured education programmes aimed at increasing physical activity is more effective if individuals are enabled to use a pedometer. It is therefore likely that the pedometer plays an important role in helping promote the self-regulatory strategies needed to convert the motivational impact of an education programme into sustained physical activity behaviour change. This is unsurprising considering the primary focus of structured education is to enable participants to actively self-manage their behaviour using self-monitoring and goal-setting strategies, for which the pedometer is ideally suited.

Although pedometer intervention studies have consistently proven effective at promoting physical activity over the short-term (Bravata et al. 2007), only two other studies have directly compared the effect of adding pedometer use to standard physical activity counselling strategies (Bjørgaas et al. 2008, Engel & Lindner 2006). One found that although
one-to-one counselling that included pedometer use resulted in increased cardiovascular fitness and improved glycaemic control after 6 months when compared to one-to-one counselling only, these results were not significant (Bjørgaas et al. 2008). However, the statistical analysis used in this study was limited by failure to control for baseline values in the analysis despite the fact that those in the pedometer group had higher levels of cardiovascular fitness and better glycaemic control than those in the non-pedometer group at baseline, making it likely that regression to the mean was responsible, at least in part, for the non-significant result (Vickers & Altman 2001). Another study in individuals with type 2 diabetes also found that intensive one-to-one lifestyle coaching that included pedometer use and step per day goals was no more effective than lifestyle coaching only at improving glycaemic control or increasing the distance walked in a shuttle test (Engel & Lindner 2006). Although the negative results in these studies may be due to the fact that they were inadequately powered to detect differences between two intervention conditions, it could also be the result of the type of intervention used. The beneficial effect of pedometers in helping participants self-regulate their physical activity behaviour may be less pronounced when regular one-to-one support is provided. However, given the equivocal nature of the evidence for the efficacy of one-to-one physical activity counselling in the United Kingdom and its resource-intensive nature (see Chapter Four), it is unlikely that this method of promoting physical activity is viable in a health setting in the United Kingdom. A recent review by NICE concluded that evidence for the efficacy of pedometer use from randomized controlled trials in a health care setting is lacking, particularly over the longer-term (National Institute of Health and Clinical Excellence 2006); the PREPARE programme study therefore adds to this body of literature by highlighting the potential utility of pedometer use in the promotion of physical activity in a health care setting, when incorporated into a theory-driven structured group education programme.

It is also possible that differences inherent in the exercise recommendations associated with the two intervention conditions contributed to the success of the EP group at improving glucose tolerance. Unlike time-based goals, step per day recommendations emphasise the importance of accumulating steps throughout the day. Consequently, participants in the EP group may have been more likely to have accumulated their physical activity levels in small bouts. A recent study found that 5 weeks of exercise training in individuals with type 2 diabetes resulted in improved glucose tolerance when individuals undertook three 10 minute sessions of moderate intensity exercise per day (Eriksen et al. 2007), but the same study
found no improvement in glucose tolerance when individuals carried out one 30 minute exercise session per day. The authors hypothesise that, when compared to a single bout, multiple bouts of physical activity result in greater metabolic activity and energy expenditure which, through unspecified mechanisms, may have lead to the observed differences in glucose tolerance between groups. It has also been shown that breaks in sedentary time are associated with improved glucose control, independent of light- to vigorous-intensity physical activity (Healy et al. 2008a), suggesting that for the same energy expenditure those who carry out small bouts of physical activity throughout the day will have improved glucose control compared to those who undertake a single exercise session followed by sedentary behaviours. However, given that the present study did not measure physical activity patterns throughout the day this hypothesis is purely speculative and needs further investigation.

Structured education

The PREPARE programme study is the first study to test the efficacy of group-based structured education at promoting health behaviour change and improving glucose tolerance in those with IGT. Previous diabetes prevention programmes and theory-driven physical activity interventions have tended to use one-to-one counselling strategies that have focused on determinants directly associated with the promoted health behaviours (Kinmonth et al. 2008, Knowler et al. 2002, Lindström et al. 2003, Mensink et al. 2003a). In contrast, the PREPARE programme curriculum focused equally on participants’ knowledge and perceptions surrounding their diagnosis of IGT and the promotion of physical activity. Results at 12 months found the PREPARE programme positively targeted perceived knowledge of IGT, perceived effectiveness of exercise as a treatment for IGT and efficacy beliefs. This is consistent with the DESMOND structured education programme for individuals with type 2 diabetes, which also found increases in perceived knowledge of diabetes in those with newly diagnosed type 2 diabetes 12-months after receiving the programme (Davies et al. 2008). In the present study overall change in perceived knowledge of IGT was also correlated with change in self-reported physical activity after 12 months. Given that pilot data from the PREPARE programme, described in Chapter 5, found that perceived knowledge of IGT changed immediately after participants received the PREPARE programme, this finding suggests that perceived knowledge of IGT is an important pre-condition for the effective promotion of physical activity in this at-risk group. Future lifestyle interventions in individuals with prediabetes should therefore consider targeting perceptions
and knowledge surrounding the condition along with the traditional motivational and volitional determinants of physical activity behaviour change.

Interventions using group-based education, as opposed to one-to-one counselling, may also offer other specific advantages. Vicarious learning has been identified by Bandura’s self-efficacy theory as one of the main sources of self-efficacy (Bandura 1997); therefore group-based education may increase the success of the programme by allowing individuals to interact and learn from others who share the same illness/condition. Although this hypothesis has not been tested in those with IGT, it is supported by several studies in those with type 2 diabetes. For example, a systematic review of self-management strategies for those with type 2 diabetes concluded that lifestyle interventions were generally more effective in group-based settings (Norris et al. 2001) and a later randomized controlled trial found that greater improvements in glycaemic control were observed when individuals were given group-based education compared to education delivered in an individual setting (Rickheim et al. 2002).

**Limitations**

This study has several important limitations, which are listed below.

Firstly, the small sample size precluded meaningful sub-group analysis, which is important given the heterogeneity of the study sample.

Secondly, the study was conducted in a single centre by a dedicated research team; this limits the generalizability of the findings to other settings.

Thirdly, as this study did not include an additional comparison group given an open pedometer, step count goals and a log book without the framework of the PREPARE programme, it was not possible to determine whether the success of the pedometer group was solely or partly due to the reactivity of wearing a pedometer and keeping a daily step count log or whether the framework of the PREPARE programme was needed in addition to pedometer use for sustained behaviour change. However, studies have shown that the reactivity of wearing an open pedometer in adults is minimal and likely to be temporary (Clemes et al. 2008, Eastep et al. 2004, Matevsky et al. 2006), suggesting that some form of additional support is required in order to initiate sustained behaviour change. Additionally, as
qualitative research has shown that individuals suffer negative emotions and affect when they are diagnosed with prediabetes and not given sufficient information surrounding their condition (Troughton et al. 2008), it could be argued that clinicians giving a diagnosis of prediabetes or IGT have a duty of care to that individual which includes providing clear and accurate information on key aspects of prediabetes. Structured education, which provides a clear and reproducible framework for targeting both illness perceptions and health behaviour, is likely to be a feasible method of meeting this need.

Fourthly, as all education programmes were delivered by the same individuals it is possible that the educators introduced bias when delivering the pedometer version of the PREPARE programme compared to the standard version of the PREPARE programme. It is also possible that the educators’ attitudes and characters, rather than the content of the PREPARE programme, were primarily responsible for affecting behaviour change.

Fifthly, whilst pedometers provide a valid and reliable objective measure of total ambulatory activity, they cannot be used to estimate exercise intensity or bout duration. Therefore it is not possible to accurately determine the relative effectiveness of the PREPARE programme at: promoting light-, moderate- or vigorous-intensity physical activity; the number of exercise bouts lasting a set length of time; or the relative importance of these factors in improving glucose tolerance. The fact that pedometers were used as a measurement tool as well as being a central part of one of the interventions also potentially limits the findings of this study in terms of physical activity.

Sixthly, this study had multiple outcome, follow-up and group comparisons. As adjustment was not carried out for these multiple comparisons there is an increased risk of a type 1 error occurring in any one measurement. However, it has been pointed out that adjustment for multiple comparisons is not necessary in studies which have a single primary outcome accompanied by multiple supportive secondary outcomes (Altman 1991, Bender & Lange 2001, Schulz & Grimes 2005). Furthermore, it has been argued that adjustment for multiple comparisons when several intervention conditions are compared against a single control condition is inappropriate if the interventions are based on the same treatment (Schulz & Grimes 2005). Given these factors, adjustment for multiple comparisons in the PREPARE programme study is likely to have diminished, rather than enhanced, the informativeness of
the results. Nonetheless, the results of this study should be considered as exploratory, rather than definitive.

**Strengths**

Along with the limitations listed in the above section, this study also has some important strengths, which are listed below.

Firstly, the development, design and evaluation of the PREPARE programme followed a systematic framework proposed by the Medical Research Council (Medical Research Council 2001), which is particularly important given that educational programmes are complex interventions that are hard to describe, and therefore hard to replicate.

Secondly, the randomization procedure used in this study worked well and produced similar groups.

Thirdly, despite its intensive nature, this study had a relatively low drop-out rate, which increases the utility of the results.

Fourthly, in contrast to most previous physical activity interventions aimed at promoting walking activity (Ogilvie et al. 2007), the participants in this study were robustly phenotyped using numerous biochemical markers of metabolic and vascular health. This enabled the clinical effectiveness, as well as the efficacy, of the PREPARE programme to be quantified.

**Conclusion**

In conclusion, this study suggests that structured education with pedometer use is effective at promoting increased physical activity and improved glucose tolerance in those with IGT. This has important implications for primary health care providers willing to invest in lifestyle interventions for those identified with an increased risk of developing a chronic disease. However, given the aforementioned limitations of this study, this approach to patient care for those identified with a high risk of developing type 2 diabetes needs to be tested further in a multi-centred randomized controlled trial with progression to type 2 diabetes as the main outcome.
Chapter Summary

The PREPARE programme study demonstrated that structured group education aimed at the promotion of physical activity is successful at improving glucose tolerance in those with IGT if it includes pedometer use. The improvements in glucose tolerance observed in this group were comparable to previous multi-factor diabetes prevention programmes. This study also demonstrated that structured education can be used to positively influence efficacy beliefs surrounding walking activity and perceived knowledge of IGT and increase ambulatory activity compared to control conditions after 12 months.
Chapter Seven

Discussion and future directions
The preceding chapters have: reviewed the role of physical activity in the prevention of diabetes; investigated the associations of walking activity with novel risk markers for type 2 diabetes; detailed the development of a new approach to promoting physical activity in individuals identified with a high risk of developing type 2 diabetes; and described a randomized controlled trial investigating the effect of the developed physical activity intervention on physical activity levels and glucose tolerance in individuals with impaired glucose tolerance.

This chapter summarizes the main findings reported within this thesis, contextualises the importance of these findings and highlights areas for future research.
Evidence from palaeoanthropological, epidemiological, intervention and mechanistic studies suggests that the human phenotype has evolved to function optimally in the presence of high energy expenditures due to physical activity and that physical inactivity is associated with metabolic dysfunction and an increased risk of developing many chronic diseases (Booth et al. 2000, Chakravarthy & Booth 2004), of which type 2 diabetes is one of the most serious and prevalent. The main aim of this thesis was to further our understanding of the role of physical activity in the prevention of type 2 diabetes. This has been achieved by using a systematic review to highlight some important gaps in and limitations of the current evidence and by designing research studies to address some of these limitations (specific research aims are listed in Chapter One).

**Chapter Two**

Chapter Two of this thesis comprises a systematic review investigating whether physical activity change is associated with a reduced risk of developing diabetes in individuals identified with IGT. IGT is a form of prediabetes and is associated with a significantly increased risk of developing type 2 diabetes and cardiovascular disease (Unwin et al. 2002). The systematic review, which included one exercise training study and seven multi-component lifestyle intervention studies, found that previous free-living lifestyle interventions, such as the Diabetes Prevention Program and the Finnish Diabetes Prevention Study, were unsuccessful at initiating meaningful increases in free-living physical activity as measured by self-report. Therefore the review concluded that, based on evidence from controlled trials, the evidence for the efficacy of physical activity behaviour change at prevention or delaying the progression to type 2 diabetes in those with IGT is equivocal. In June 2007, this review was published in Diabetologia (Yates et al. 2007a), where its conclusions were broadly supported in an accompanying editorial (Carnethon 2007), challenged in a subsequent letter to the editor from the investigators of the Finnish Diabetes Prevention Study (Laaksonen et al. 2007) and defended by a published response from the authors of the review (Yates et al. 2007b); differences of opinion notwithstanding, there was broad agreement that more research using objective methods of measuring physical activity was needed. This debate in the scientific press emphasizes the fact that there is not enough evidence from well designed physical activity intervention trials to enable definitive statements to be made about the independent role of physical activity behaviour change in the prevention of type 2 diabetes. Accordingly, as discussed in Chapter Two, the advice given by
ADA recommends that individuals with IGT should include 150 minutes per week of moderate- to vigorous-intensity exercise as part of a weight management programme (Sigal et al. 2006), inferring that the main aim of interventions in individuals with prediabetes should be weight management. This position understates the numerous epidemiological and mechanistic studies that have demonstrated the independent importance of physical activity in the prevention of type 2 diabetes (Bassuk & Manson 2005). Therefore the lack of data from experimental trials is the important missing link in the evidence chain needed to definitively quantify the role of physical activity in preventing or delaying the progression to type 2 diabetes in those with prediabetes.

**Chapter Three**

Chapter Three adds to the epidemiological evidence linking physical activity to metabolic and vascular health by detailing a cross-sectional study that found important reductions in key markers of chronic low-grade inflammation in individuals screened for type 2 diabetes who achieve at least 30 minutes of self-reported walking activity on at least five days per week compared to those reporting less than that. Although markers of chronic low-grade inflammation, which are thought to play an important role in the pathogenesis of type 2 diabetes (Pickup & Crook 1998), have previously been inversely associated with physical activity (Panagiotakos et al. 2005, Pischon et al. 2003, Wannamethee et al. 2002), the effect of walking, independent of more vigorous forms of exercise, has not been described; this is an important limitation as walking has been shown to be the preferred choice of physical activity for the majority of individuals (Booth et al. 1997, Crespo et al. 1996, Vaz de Almeida et al. 1999), including those with IGT (Laaksonen et al. 2005).

This study therefore further emphasizes the clinical importance of promoting walking activity to levels that are consistent with the current physical activity recommendations in sedentary populations, particularly in those identified with an increased risk of developing a chronic disease, and supports the findings from other studies that have shown that walking activity is independently, and inversely, associated with the risk of developing type 2 diabetes (Hu et al. 1999, Laaksonen et al. 2005).

The findings reported in this study have were published in Preventive Medicine (Yates et al. 2008a).
Chapters Four, Five and Six

Chapters Four, Five and Six of this thesis detail the development and testing of the PREPARE programme, a structured education programme aimed at increasing physical activity and improving glucose tolerance in those with IGT. These chapters were informed by internationally recognized criteria for developing, evaluating and reporting complex interventions (Medical Research Council 2000, Moher et al. 2001).

The equivocal nature of the evidence surrounding the effectiveness of previous diabetes prevention programmes at increasing levels of physical activity (see Chapter Two) is mirrored in physical activity and lifestyle interventions studies in the UK, where the majority have proven ineffective at initiating clinically significant increases in physical activity, particularly over the longer term (Dyson et al. 1997, Harland et al. 1999, Hillsdon et al. 2002, Kinmonth et al. 2008). These physical activity and lifestyle interventions, like previous diabetes prevention programmes, utilized one-to-one counselling strategies, which is the most widely researched method of promoting behaviour change. However, an alternative to one-to-one counselling in the promotion of health behaviour change is structured education which has recently been emphasized in government agendas (Department of Health 2005). Structured education, which refers to group-based education, with a theory-driven written curriculum, is widely used as a method of promoting self-management skills and behaviour change in those with type 2 diabetes. It is typically used as part of patients’ usual care and delivered in a primary health care or community setting. Structured education has proven to be an acceptable and successful self-management strategy for those with type 2 diabetes, compatible with the infrastructure of the National Health Service and likely to be more cost-effective than other methods of promoting patient self-management (Davies et al. 2008, Heller et al. 1988, National Institute for Clinical Excellence 2003 and 2008, Rickheim et al. 2002). However, it has not been tested in at-risk individuals or utilized specifically as a method of promoting physical activity behaviour change (see Chapter Four for a further discussion of structured education).

The PREPARE programme, which is a theory-driven structured education programme for those with IGT, was developed in response to this need. The primary aim of the PREPARE programme study was to improve glucose tolerance in individuals with IGT through increased physical activity, predominately walking activity. A detailed description of the
background, rationale and content of the PREPARE programme is detailed in Chapter Four and the results of initial pilot studies testing the efficacy of the programme are detailed in Chapter Five.

The efficacy of the PREPARE programme at increasing levels of physical activity and improving glucose control and other markers of metabolic health was tested in a randomized controlled trial, detailed in Chapter 6. Two versions of the PREPARE programme were developed and tested against control conditions: a standard version and a pedometer version. The standard version encouraged participants to set time-based goals based on generic exercise recommendations, whereas the pedometer version enabled participants to set personalized steps-per-day goals and to objectively self-monitor their daily physical activity levels using a pedometer. One hundred and three individuals were recruited to the study and follow-up was conducted at 3, 6 and 12 months. The main outcome was 2-h glucose and secondary outcomes included physical activity, as measured objectively by a pedometer and subjectively by self-report, along with markers of chronic low-grade inflammation, illness perceptions and efficacy beliefs. Results at 12 months found that individuals who were randomized to the pedometer version of the PREPARE programme significantly decreased their 2-h glucose levels by -0.94 mmol/l (95% CI -1.79 to -0.10) compared to control conditions, despite no significant change in body weight or waist circumference. However, there was no significant improvement in glucose tolerance in those given the standard education programme. Although in the pedometer group all markers of chronic low-grade inflammation were decreased at 12 months compared to the control group, these differences failed to reach significance. Objectively measured ambulatory activity and overall self-reported moderate- to vigorous-intensity physical activity increased in the pedometer group compared to the control at all follow-up time points. At 12 months the increase in pedometer counts, compared to the control group, was 1952 steps per day (95% CI 953 to 2951); this equates to around 140 minutes ambulatory activity per week (Tudor-Locke & Bassett 2004), which is similar to the current minimum physical activity recommendations for inactive individuals (Department of Health 2004b, Haskell et al. 2007). In contrast, although those who received the standard PREPARE programme had a significant increase in pedometer counts compared to the control group at 12 months, there was no significant difference at the intermediary follow-up time points. As was hypothesised in this thesis (see Chapter Four) it is therefore likely that the pedometer played an important role in promoting the self-
regulatory strategies needed to convert the motivational impact of the education programme into sustained physical activity behaviour change.

Despite being a single-factor physical activity intervention which did not result in weight loss, the pedometer group achieved improvements in glucose tolerance that are comparable to multi-factor diabetes prevention programmes (Yamaoka & Tango 2005). Indeed, the decrease in 2-h glucose observed in the pedometer group relative to the control group has been associated with around a 40-50% reduction the relative risk of developing type 2 diabetes (Tuomilehto & Wareham 2006, Yamaoka & Tango 2005). The PREPARE programme study therefore provides new evidence surrounding the efficacy of structured education at increasing physical activity in those with IGT and serves to emphasize the key importance of physical activity in the prevention of type 2 diabetes.

As well as addressing many of the limitations identified by the systematic review in Chapter Two, the detailed description of the development of PREPARE programme and the subsequent randomized controlled trial described in Chapters Four, Five and Six has some important strengths and adds to the evidence in two other key ways. Firstly, by providing a detailed description of the theoretical underpinning and rationale behind the PREPARE programme and by measuring several of the key determinants associated with these theories, this thesis addresses some important limitations of previous lifestyle and physical activity intervention studies. Namely, few randomized controlled trials have adequately described or evaluated the theoretical underpinning of the intervention being tested (Brug et al. 2005, Michie & Abraham 2004). This has curtailed advances in our understanding of the key determinants associated with physical activity behaviour change, resulting in few innovative advances being made in the health professional’s ability to change targeted behaviours (Rothman 2004). The PREPARE programme provided a novel insight into the potential importance of perceived knowledge of IGT and pedometer use in the initiation and maintenance of physical activity behaviour change after a lifestyle intervention designed to treat or control IGT through increased physical activity.

Secondly, by investigating the effectiveness of an intervention designed to promote walking activity at improving relevant biochemical markers of metabolic health in those with IGT and by including a long-term follow-up at 12 months the PREPARE programme study addressed several of the key limitations recently identified in a systematic review of walking
intervention studies (Ogilvie et al. 2007). This systematic review concluded that, in terms of public health, most walking studies have, to date, been conducted over the short-term in non-patient groups and have investigated the efficacy rather than the clinical effectiveness of walking interventions.

An article detailing the rationale, design and baseline data for the PREPARE programme study was published in Patient Education and Counseling (Yates et al. 2008b) and findings from intermediary follow-up time points have been presented at several national and international conferences including the annual meeting of the European Association for the Study of Diabetes, Rome, 2008 (Yates et al. 2008e).

**Future directions**

The findings presented in this thesis could have important implications for clinical practice regarding the efficacy of structured education with pedometer use at increasing physical activity levels and improving glucose control in individuals identified with an increased risk of developing type 2 diabetes; however, in the light of the important limitations of the PREPARE programme study (see Chapter Six), more trials are needed in multi-centred settings to further test the efficacy of structured education at promoting health behaviour change and reducing the risk of type 2 diabetes in community and health care settings. As successful strategies for identifying those at risk of developing type 2 diabetes are vital if prevention initiatives are to be implemented in a wider health care setting, it is also important that the feasibility and acceptability of screening for type 2 diabetes and prediabetes are established. In particular, screening for those with prediabetes is likely to subject thousands of individuals who previously thought they were healthy to the stigma or worry of being labelled “ill” or “unhealthy”, therefore it is crucial that the cost-effectiveness of identifying and educating those at risk of developing type 2 diabetes is clearly ascertained before such initiatives are considered for implementation on a national level.

Further research is also needed from randomized controlled trials investigating the efficacy of physical activity behaviour change at promoting metabolic health. In particular, the relative importance of physical activity mode, bout length and intensity in reducing the
incidence of type 2 diabetes and improving traditional and novel markers of metabolic health require further investigation. Other areas not considered in this thesis that are also likely to play an important role in shaping future research agendas and policy decisions also deserve a brief mention: firstly, recent research using objective measures of physical activity has shown that time spent in sedentary pursuits is inversely associated with glucose control and metabolic health, independent of light-, moderate- or vigorous-intensity physical activity (Healy et al. 2008a, Healy et al. 2008b). Therefore it is likely that sedentary time and behaviours, as distinct from physical activity, will be the target of future research interventions and diabetes prevention programmes. Secondly, the impact of certain genotypes on the association between physical activity and health is an emerging and relevant area of research that is likely to have important implications in the future (Roth 2008). Although controversial, it is conceivable that future lifestyle recommendations for those identified with an increased risk of developing type 2 diabetes may be tailored to specific genotypes, meaning the end of universal exercise, diet and weight loss recommendations.

The success of the PREPARE programme study was key in helping the Diabetes Research Unit, hosted by the University of Leicester and the University Hospitals of Leister NHS Trust, secure funding to enable further investigation into the efficacy of structured education at reducing the risk of type 2 diabetes in those with prediabetes along with continued investigation into the importance of different aspects of physical activity in determining metabolic health. To date, over four million pounds has been committed to this research from various funding bodies, including the National Institute for Health Research. The results of these studies should be known within the next five years.
References


Hillsdon M, Foster C & Thorogood M, 2005a. Interventions for promoting physical activity, *Cochrane Database of Systematic Reviews*, 1, CD003180.


Marcell TJ, McAuley KA, Traustadóttir T & Reaven PD, 2005. Exercise training is not associated with improved levels of C-reactive protein or adiponectin, Metabolism: Clinical and Experimental, 54, 533-541.


Skinner TC, Davies MJ, Heller S, & Khunti K, 2005. To determine the effects of a structured educational programme on illness beliefs, quality of life and physical activity in individuals with newly diagnosed type 2 diabetes: results from the DESMOND (Diabetes education and self management for ongoing and newly diagnosed) pilot study, Diabetic Medicine, 22, A 15.


Yates T, Khunti K & Davies MJ, 2007c. Can structured education be used to promote physical activity in primary care? Diabetes and Primary Care, 9, 252-260


Appendix One

Journal publications and conference presentations relating to work detailed in this thesis
**Journal Articles**

(Copies of articles are presented below in the listed order)


**Conference Presentations**

Annual Diabetes UK professional conference 2007 and 2008


Yates T, Khunti K, Bull F, Gorely T, Mandalia P & Davies M, 2008. Three-month follow-up data from the PREPARE (Pre-diabetes Risk Education and Physical Activity Recommendation and Encouragement) programme study, *Diabetic Medicine*, 25, A48 (this abstract was short-listed for the Education Award)


Accepted for the annual meeting of the European Association for the Study of Diabetes, 2008

The role of physical activity in the management of impaired glucose tolerance: a systematic review

T. Yates • K. Khunti • F. Bull • T. Gorely • M. J. Davies

Received: 15 December 2006 / Accepted: 23 January 2007
© Springer-Verlag 2007

Abstract Although physical activity is widely reported to reduce the risk of type 2 diabetes in individuals with prediabetes, few studies have examined this issue independently of other lifestyle modifications. The aim of this review is to conduct a systematic review of controlled trials to determine the independent effect of exercise on glucose levels and risk of type 2 diabetes in people with prediabetes (IGT and/or IFG). A detailed search of MEDLINE (1966-2006) and EMBASE (1980-2006) found 279 potentially relevant studies, eight of which met the inclusion criteria for this review. All eight studies were controlled trials in individuals with impaired glucose tolerance. Seven studies used a multi-component lifestyle intervention that included exercise, diet and weight loss goals and one used a structured exercise training intervention. Four studies used the incidence of diabetes over the course of the study as an outcome variable and four relied on 2-h plasma glucose as an outcome measure. In the four studies that measured the incidence of diabetes as an outcome, the risk of diabetes was reduced by approximately 50% (range 42–63%); as these studies reported only small changes in physical activity levels, the reduced risk of diabetes is likely to be attributable to factors other than physical activity. In the remaining four studies, only one reported significant improvements in 2-h plasma glucose even though all but one reported small to moderate increases in maximal oxygen uptake. These results indicate that the contribution of physical activity independent of dietary or weight loss changes to the prevention of type 2 diabetes in people with prediabetes is equivocal.

Keywords Exercise • IFG • Impaired fasting glucose • IGT • Impaired glucose tolerance • Physical activity • Prediabetes • Prevention • Type 2 diabetes

Abbreviations
ADA American Diabetes Association
DPP Diabetes Prevention Program
FDPS Finnish Diabetes Prevention Study
MeSH medical subject headings

Introduction
Given the growing prevalence of diabetes and the high economic cost of treating the condition and its comorbidities, it is important to find effective ways of targeting those who are most at risk of developing the disease [1]. Prediabetes is the collective term for people with IGT and/or IFG [2]. Prediabetes is associated with an increased risk of development of type 2 diabetes [3] and cardiovascular disease [4-6]. There is good evidence from cross-sectional and longitudinal studies for a link between levels of physical activity and the risk of type 2 diabetes [7-9]. However,
Evidence from intervention studies in high-risk populations is limited, making it difficult to quantify the effectiveness of physical activity in reducing the risk of type 2 diabetes in individuals with prediabetes. Lifestyle intervention studies that have encouraged weight loss through a combination of dietary change and increased physical activity have reduced the risk of type 2 diabetes in individuals with IGT [10-14]. However, because physical activity was not usually analysed independently of other variables, such as weight loss, it is difficult to determine the effectiveness of physical activity at protecting against the risk of diabetes in individuals with prediabetes. Therefore, the aim of this systematic review is to establish the effectiveness of physical activity independent of other variables at reducing the risk of diabetes or improving glucose parameters in people with prediabetes.

Materials and methods

Search strategy MEDLINE (1966 to February week 4, 2006) and EMBASE (1980 to week 8, 2006) were searched for articles examining the effect of an exercise or lifestyle intervention on individuals with prediabetes. The search was carried out using medical search headings (MeSH) and by searching titles and abstracts for relevant words. For example, studies including individuals with prediabetes were found by using the MeSH 'prediabetic state,' 'insulin resistance,' 'glucose intolerance' and 'diabetes mellitus' (subheading 'prevention and control'), and by searching titles and abstracts for 'prediabetes,' 'impaired glucose tolerance,' 'IGT,' 'impaired fasting glucose' and 'IFG.' Studies that included an exercise intervention were found by using the MeSH 'lifestyle,' 'sports,' 'exercise therapy' and 'physical fitness,' and by searching titles and abstracts for 'exercise,' 'physical activity,' 'physical fitness,' 'resistance training,' 'endurance training' and 'aerobic training.' In addition, the reference lists of relevant published original articles and reviews were hand-searched.

One reviewer (T. Yates) performed the electronic and hand-searches and reviewed the results. Studies that clearly did not meet the inclusion criteria were rejected during the initial review. Where uncertainty existed, the full text of the article was obtained and reviewed. Two reviewers (T. Yates and K. Khunti) independently assessed all potentially relevant studies and performed data extraction. Disagreement was resolved by discussion and, where necessary, third party adjudication.

Subjects Participants were adults (age ≥ 18 years) diagnosed with prediabetes. Prediabetes was defined as IGT and/or IFG using one of the sets of criteria previously recommended by the WHO [15, 16] or the American Diabetes Association (ADA) [17, 18]. Studies that defined IGT or IFG using other criteria were included if the mean value of the participants' plasma glucose fell within the range of IGT or IFG as defined by the WHO or ADA criteria (2-h plasma glucose ≥ 7.8 mmol/l and < 11.1 mmol/l, and fasting glucose < 7.8 mmol/l for IGT; 2-h plasma glucose < 7.8 mmol/l and fasting glucose ≥ 5.6 mmol/l and < 7.0 mmol/l for IFG).

Interventions Interventions that included an exercise programme were included. 'Exercise programme' was taken to mean any intervention that actively promoted and supported physical activity or a structured exercise training regimen. Studies that only provided individuals with brief written or verbal physical activity advice were excluded. Studies investigating the effect of a single or acute episode of exercise were also excluded.

Outcome measures Only studies with an outcome measure of physical activity and a relevant clinical measure were included. A relevant clinical measure was defined as progression to diabetes or a suitable measure of plasma glucose (2-h plasma glucose for IGT, or fasting glucose for IFG).

Type of study Randomised and non-randomised controlled trials were included.

Analysis As the heterogeneity of the type of exercise interventions and outcome measures did not lend itself to quantitative methods of analysis, a systematic narrative review was undertaken. Baseline and follow-up exercise, body mass and glucose parameters were reported using mean ± SEM, or median (interquartile range). Results reporting the SD or the 95% CI were converted to SEM using the formula SEM = SD/√n and SEM = (Chigh - Cherch) /t (where t is the t distribution value for a 95% CI), respectively. Where the SEM, SD or 95% CI for the change from baseline to follow-up was not reported, only the mean value is reported because of the potential error involved in calculating SEM for this figure. When available, the relative risk of diabetes in the intervention group compared with the control group was also reported.

Results

The search produced 307 hits, from which 279 potential studies were identified, of these, eight trials met the criteria for inclusion (see Fig. 1). Study details for the eight included studies are shown in Table 1; the main outcomes are presented in Table 2.
Diabeticologia

Fig. 1 Flow diagram of the literature search. Duplicates: where several studies reported on the same trial and cohort, only one published study for each trial was included for the purposes of this review; the included studies were those that reported on the cohort as a whole, had relevant follow-up measures and included the most recently published data. Where a relevant study was identified in more than one publication, the study was included only once.

Study design Seven of the eight trials involved randomisation of subjects to a treatment group or control group [10, 12, 19–23]. The non-randomised trial identified control participants by using individuals who, for various (unstated) reasons, were not enrolled in the intervention programme [24].

Sample size Sample size ranged from 62 to 2,161. Two studies reported a power calculation based on the expected difference in the incidence of diabetes between groups [12, 21], and one reported a power calculation based on the expected difference between groups in the proportion of individuals with IGT at the end of the study [19]. In the latter study, O'Driscoll et al. calculated that a total of 100 participants were required to detect a 0.6 mmol/l difference in fasting glucose and a 20% difference in the number of individuals with IGT, allowing for a 90% power at a significance of 0.05. Three studies had sample sizes of fewer than 100 participants at follow-up [19, 20, 22].

Inclusion criteria All studies examined in this review included individuals with IGT and excluded those with isolated IFG [10, 12, 19–24].

Sex Except for one trial that involved only men (n=188) [24], all trials included both men and women. In the included studies a total of 40% of participants were men.

Intervention conditions Seven of the eight included studies used a multi-component lifestyle intervention [10, 12, 19, 21–24], and one used a structured gym-based exercise training intervention [20].

Six of the lifestyle intervention studies were based on encouraging individuals to increase their physical activity to approximately 150 min of exercise of moderate to vigorous intensity per week whilst also encouraging weight loss through a healthy energy-restricted diet [10, 12, 19, 21, 22, 24]. Participants in all six studies received regular encouragement and counselling from a trained dietitian at least once every 3 months throughout the duration of the intervention. Two of the six studies also provided participants with the option of attending supervised exercise classes for some or all of the study duration [21, 24] and one provided discounted access to local gyms [19]. One study determined the effect of diet and exercise separately and in combination [10].

One lifestyle intervention included an initial 1-month stay at a wellness centre where individuals were provided with healthy dietary options and encouraged to take part in 2.5 h/day of light to moderate intensity exercise using the leisure facilities provided [23]. After the stay at the wellness centre, participants were encouraged to make plans about how they could incorporate healthier habits into everyday life and then received no further contact until follow-up.

The structured exercise intervention study used a training protocol of 180 min per week of aerobic exercise at 70% of heart rate reserve [20]. Exercise training was supervised for the first 6 months and both groups were encouraged to eat a healthy energy-balanced diet, with those in the exercise training group also being encouraged to eat a diet with a high percentage of energy from carbohydrate [20].

Outcomes Four studies included the incidence of diabetes as the main outcome [10, 12, 21, 24], and four used 2-h plasma glucose levels as a direct measure of glucose control [19].
Table 1 Characteristics of included studies

<table>
<thead>
<tr>
<th>Authors, year</th>
<th>Study location/ name</th>
<th>Study design</th>
<th>Intervention duration</th>
<th>Number of subjects (men/women)</th>
<th>Inclusion criteria</th>
<th>Type of intervention</th>
<th>Type of dietary intervention</th>
<th>Type of exercise intervention</th>
<th>Method of physical activity measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindstrom et al. 2003 [21]</td>
<td>Finland/ Finnish Diabetes Prevention Study</td>
<td>RCT</td>
<td>3 years</td>
<td>522 (172/350)</td>
<td>KGT (WHO criteria, 1985), age 40–64 years, BMI ≥ 25 kg/m²</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy diet</td>
<td>Participants individually encouraged to increase their overall level of physical activity. Circuit-type exercise sessions were also offered.</td>
<td>Self-report— Kuopio 12 month leisure time physical activity questionnaire</td>
</tr>
<tr>
<td>Knowler et al. 2002 [12]</td>
<td>USA/ Diabetes Prevention Research Group</td>
<td>RCT</td>
<td>Average follow-up</td>
<td>2.8 (range 1.8–4.6)</td>
<td>KGT (ADA criteria, 1997), age ≥ 25 years, BMI ≥ 24.4 kg/m² (≥ 22 kg/m² if Asian), fasting plasma glucose ≥ 5.5 mmol/L</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet</td>
<td>Participants individually encouraged to accumulate at least 150 min/week of moderate intensity exercise.</td>
<td>Self-report— Modified Activity Questionnaire and activity log</td>
</tr>
<tr>
<td>Pan et al. 1997 [10]</td>
<td>China/The Da Qing KIT and Diabetes Study</td>
<td>RCT</td>
<td>6 years</td>
<td>530 (283/247)</td>
<td>KGT (WHO criteria, 1985), age ≥ 25 years</td>
<td>1. Exercise and diet</td>
<td>Weight maintenance for those with a BMI &lt; 25 kg/m² through a healthy energy-balanced diet; weight reduction for those with a BMI ≥ 25 kg/m² through reduced energy intake</td>
<td>Participants were encouraged to increase their physical activity to 1 unit per day7. Those who were aged &lt; 50 years and were able to were encouraged to accumulate 2 units per day.</td>
<td>Self-report— type not reported</td>
</tr>
<tr>
<td>Eriksson and Lindgarde 1991 [24]</td>
<td>Sweden/The Malmö feasibility study</td>
<td>Non-randomised controlled trial</td>
<td>5 years</td>
<td>260 (260/0)</td>
<td>KGT (2-h post-challenge glucose values 7–11 mmol/L, and fasting plasma glucose &lt; 7.8 mmol/L)</td>
<td>Exercise and diet</td>
<td>Healthy dietary advice given</td>
<td>Participants were encouraged to increase their physical activity levels. Participants given the option of training in organised groups for a</td>
<td>VO2max— heart rate response to submaximal workload using bicycle ergometer</td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Study Type</td>
<td>Duration</td>
<td>Sample Size</td>
<td>Inclusion Criteria</td>
<td>Intervention</td>
<td>Outcomes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------</td>
<td>------------</td>
<td>------------</td>
<td>----------</td>
<td>-------------</td>
<td>-------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oldroyd et al.</td>
<td>England</td>
<td>RCT</td>
<td>2 years</td>
<td>69 (3930)</td>
<td>KIT (WHO criteria, 1985)</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-month period in the first year</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Participants were encouraged to undertake 20-30 min of aerobic activity 2-3 days/week</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Menink et al.</td>
<td>Netherlands</td>
<td>RCT</td>
<td>2 years</td>
<td>114 (64/50)</td>
<td>KIT (2-h post-challenge glucose values 7.8-12.5 mmol/l, and fasting plasma glucose &lt;7.8 mmol/l), age &gt;40 years, BMI ≥25.0 kg/m²</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-month period in the first year</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Participants were encouraged to undertake 20-30 min of aerobic activity 2-3 days/week</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Self-report—type not reported</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lindahl et al.</td>
<td>Sweden</td>
<td>RCT</td>
<td>1 year</td>
<td>186 (69/117)</td>
<td>KIT (WHO criteria, 1985), age 30-60 years, BMI ≥27 kg/m²</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-month period in the first year</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Participants were encouraged to increase their physical activity.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Supervised exercise sessions were available</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>in the first month</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Walking/jogging at &gt;70% of heart rate reserve for 1 h on 3 days/week</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carr et al.</td>
<td>USA</td>
<td>RCT</td>
<td>2 years</td>
<td>62 (29/33)</td>
<td>KIT (WHO criteria, 1995)</td>
<td>Structured exercise and diet</td>
<td>Participants were encouraged to follow the energy-balanced American Heart Foundation Step 2 diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6-month period in the first year</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Participants were encouraged to undertake 20-30 min of aerobic activity 2-3 days/week</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Self-report—type not reported</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1O₂max—maximal oxygen consumption
2Data for lifestyle and control groups only
31 unit = 30 min of mild exercise, or 20 min of moderate exercise, or 10 min of strenuous exercise, or 5 min of very strenuous exercise
<table>
<thead>
<tr>
<th>Authors, year</th>
<th>Baseline ( F_U ) (min)</th>
<th>Change in ( F_U ) (min)</th>
<th>Baseline selfreported physical activity levels</th>
<th>Change in selfreported leisure time physical activity</th>
<th>Baseline body mass (kg)</th>
<th>Change in body mass (kg)</th>
<th>Baseline 2-h plasma glucose (mmol/l)</th>
<th>Change in 2-h plasma glucose from baseline (mmol/l)</th>
<th>Baseline fasting glucose (mmol/l)</th>
<th>Change in fasting plasma glucose (mmol/l)</th>
<th>Relative risk of diabetes in the intervention group vs the C: group (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindström et al. 2003 [21]</td>
<td>N/A</td>
<td>N/A</td>
<td>L: 156 (62 to 288) min/week</td>
<td>L: 61 (-33 to 168) min/week²</td>
<td>L: 86.7±0.8</td>
<td>Results at 3 year</td>
<td>L: 8.9±0.1</td>
<td>Change at 1 year</td>
<td>L: 6.1±0.05</td>
<td>Change at 1 year</td>
<td>0.4 (0.3-0.7)</td>
</tr>
<tr>
<td>Knowler et al. 2002 [12]</td>
<td>N/A</td>
<td>N/A</td>
<td>L: 15.5±0.7 MET-h/week</td>
<td>L: 94.1±0.6</td>
<td>L: -5.6±0.1</td>
<td>L: 9.1±0.03</td>
<td>NR</td>
<td>NR</td>
<td>L: 5.9±0.01</td>
<td>NR</td>
<td>0.4 (0.3-0.5)</td>
</tr>
<tr>
<td>Pan et al. 1997 [10]</td>
<td>N/A</td>
<td>N/A</td>
<td>E: 1.3±0.02 min²</td>
<td>E: 0.6±0.00</td>
<td>DAE: -2.5</td>
<td>E: 8.8±0.1</td>
<td>E: 1.7</td>
<td>E: 5.6±0.1</td>
<td>E: 1.3</td>
<td>E: 0.5 (0.2-0.9)</td>
<td></td>
</tr>
<tr>
<td>Eriksson and Lindström 1991 [21]</td>
<td>L: 2.4±0.04</td>
<td>C: 2.29±0.1</td>
<td>L: 85.3±2.9</td>
<td>L: -1.3±1.3</td>
<td>C: 8.3±0.1</td>
<td>C: 0.1±0.1</td>
<td>L: 9.2±0.1</td>
<td>Change at 1 year</td>
<td>L: 6.1±0.1</td>
<td>Change at 1 year</td>
<td>N/A</td>
</tr>
<tr>
<td>Okboyd et al. 2006 [19]</td>
<td>N/A</td>
<td>N/A</td>
<td>L: 85.5±2.5</td>
<td>L: -0.6±0.3</td>
<td>C: 8.2±0.2</td>
<td>Change at 2 years</td>
<td>L: 0.3±0.3</td>
<td>Change at 2 years</td>
<td>L: 0.3±0.1</td>
<td>Change at 2 years</td>
<td>C: 0.1±0.2</td>
</tr>
<tr>
<td>Study</td>
<td>L: 2.15±</td>
<td>L: 0.09±</td>
<td>N/A</td>
<td>N/A</td>
<td>L: ±6±</td>
<td>L: ±2.4±</td>
<td>L: ±8.9±0.3</td>
<td>Change at 1 year</td>
<td>L: ±5.9±0.1</td>
<td>Change at 1 year</td>
<td>N/A</td>
</tr>
<tr>
<td>------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----</td>
<td>-----</td>
<td>---------</td>
<td>-----------</td>
<td>-------------</td>
<td>-----------------</td>
<td>-------------</td>
<td>-----------------</td>
<td>-----</td>
</tr>
<tr>
<td>Menink et al. 2003 [22]</td>
<td>0.1</td>
<td>1.59b</td>
<td></td>
<td></td>
<td>1.9</td>
<td>0.7</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C: 2.33±</td>
<td>C: ±0.01±</td>
<td>0.1</td>
<td>2.77</td>
<td>C: ±0.3±</td>
<td>C: ±0.1±</td>
<td>C: ±8.6±0.2</td>
<td>C: ±0.9±0.3b</td>
<td>C: ±5.8±0.1</td>
<td>C: ±0.1±0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lindahl et al. 1999 [21]</td>
<td>0.1a</td>
<td>0.1±a</td>
<td></td>
<td></td>
<td>1.1</td>
<td>0.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>C: ±0.02±</td>
<td>C: ±0.02±</td>
<td>0.1</td>
<td>0.1</td>
<td>C: ±0.5±</td>
<td>C: ±0.5±</td>
<td>C: ±8.0</td>
<td>C: ±0.3±0.3</td>
<td>C: ±6.1</td>
<td>C: ±0.3±0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carr et al. 2005 [20]</td>
<td>0.1</td>
<td>0.06b</td>
<td></td>
<td></td>
<td>2.9</td>
<td>0.3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>E: ±1.9±</td>
<td>E: ±0.16±</td>
<td>0.1</td>
<td>0.04±</td>
<td>C: ±0.6±</td>
<td>C: ±0.6±</td>
<td>C: ±9.1±0.2</td>
<td>E: ±0.7</td>
<td>C: ±5.4±0.1</td>
<td>C: ±0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data are presented as means±SD or medians (interquartile range).

- Change from baseline values reflect results at final follow-up, unless stated otherwise.
- \(^b^p<0.05\) vs C.
- \(^a^Value estimated from graph.
- \(^b^1\) unit=20 min of moderate E; or 10 min of strenuous E.
- \(^1^Fitness measurement taken in a randomly selected subgroup, n=45.

C control, D diet, E exercise, L lifestyle, MET-h/week metabolic equivalent per week, NR not reported, N/A not applicable.
All the studies using the incidence of diabetes as their main outcome were based on a multi-component lifestyle intervention (see intervention conditions).

**Incidence of diabetes and physical activity** All four of the intervention studies that measured the incidence of diabetes as their primary outcome found a significant reduction in the incidence of type 2 diabetes in the intervention group. Diabetes incidence was reduced by 42-63% in this group compared with the control group (see Table 2). The study that investigated the effect of diet and physical activity both separately and in combination found a greater reduction in the incidence of diabetes (46% reduced risk) in the physical activity-only group than in either the combined physical activity and diet group (42% reduced risk) or the diet-only group (13% reduced risk), although the difference between groups was not statistically significant [10]. Three of these four studies relied on self-reported measures of physical activity [10, 12, 21], and of these, only the Diabetes Prevention Program (DPP) [12] and the Finnish Diabetes Prevention Study (FDPS) [21] reported using a validated physical activity questionnaire. All three of the studies relying on self-reported physical activity levels reported non-significant to small changes in physical activity levels in the intervention group. For example, the DPP reported a mean increase in energy expenditure due to leisure time physical activity of around six metabolic equivalent hours per week [12], which is approximately equivalent to walking at a moderate pace for 15 min/day [25]. The FDPS reported no significant change in total physical activity levels compared with the control group and an increase of 9 min/day in moderate to vigorous physical activity [21], and the Da Qing IGT and Diabetes Study reported no significant change in physical activity levels compared with the control group [10]. The Malmö Feasibility Study, which used an objective outcome measure (cardiovascular fitness), reported an 8% increase in maximal oxygen uptake [24].

**2-h post-challenge plasma glucose and physical activity** Three of the studies that used the incidence of diabetes as their primary outcome measure also measured 2-h plasma glucose before and after the intervention [10, 21, 24]. The FDPS reported a 0.9 mmol/l decrease in 2-h plasma glucose after 1 year, but no significant change after 3 years [21]; the Da Qing IGT and Diabetes Study found that 2-h plasma glucose increased in all groups, but the increase in the control group was over twice that in either of the intervention groups [10]; and the Malmö Feasibility Study reported a 1.1 mmol/l reduction in 2-h plasma glucose in the intervention group [24]. Of the three lifestyle intervention studies that used 2-h plasma glucose levels rather than the incidence of diabetes as the primary indicator of improved glucose tolerance [19, 22, 23], only one reported a significant difference between the groups in terms of 2-h plasma glucose at follow-up [22]. Two of the studies used a measure of cardiovascular fitness as an indicator of physical activity levels [22, 23], and one [19] used distance walked in a shuttle test [26] as a measure of physical activity. Two studies found a small to moderate increase in cardiovascular fitness (<10% increase compared with baseline value) [22, 23], and the study using the shuttle test reported no change in the distance walked during the test [19]. Similarly, the moderate increases in cardiovascular fitness observed in the structured exercise training study were not associated with significant improvements in 2-h plasma glucose compared with the control group [20].

**Fasting glucose** None of the included studies reported a significant change in fasting glucose in the intervention group compared with the control group at follow-up. One study did not report fasting glucose values [24].

**Discussion**

Eight controlled trials in individuals with IGT were included in this review. Four studies measured the incidence of diabetes as a primary outcome measure, and found that the risk of diabetes was reduced by approximately 50% (range 42-63%) in individuals who were encouraged to reduce their body mass through changes in diet and physical activity [10, 12, 21, 24]. Although the promotion of physical activity was an important component of these studies, the effect of exercise independent of other factors on the risk of diabetes in individuals with IGT is still unclear. All but one [10] of the studies included in this review reported significant weight loss among participants. Given that weight loss is known to improve many of the factors associated with IGT, including insulin sensitivity and glycaemic control [27], and considering only modest increases in physical activity were found in these studies, the success of these interventions is likely to be largely explained by weight loss. The apparent success of the exercise-only intervention in the Da Qing IGT and Diabetes Study [10] is likely to be attributable, at least in part, to the significantly higher levels of physical activity at baseline in the exercise intervention group compared with the control group. The separation of physical activity and weight loss may seem an over-correction given that increased physical activity may encourage weight loss through increased energy expenditure; however, several meta-analyses of controlled trials investigating the effect of physical activity on glycaemic control in individuals with diabetes found that exercise training was not associated with weight loss [28, 29]. Furthermore it is increasingly recognised that at least 60 min/
day of moderate intensity exercise should be undertaken for the effective management of body mass [30], an amount that none of the interventions included in this review achieved.

Three of the four studies that investigated the effect of a lifestyle intervention in individuals with IGT on the incidence of type 2 diabetes [10–12] relied on self-reported measures of physical activity. Given the limitations of subjective measures of physical activity, particularly when measuring non-structured forms of moderate physical activity such as walking activity [31], these lifestyle intervention studies provide uncertain information about the effect of physical activity in individuals with IGT.

Results from the lifestyle intervention studies that relied on changes in 2-h plasma glucose rather than the incidence of diabetes as the primary measure of glucose control were inconclusive [19, 20, 23]. Two of the three studies were unsuccessful at improving glucose tolerance [19, 23]. Similarly, the one study that used an aerobic exercise training protocol found no improvements in glucose tolerance as measured by 2-h plasma glucose [20]. However, it did find a significant improvement in insulin sensitivity at both 6 and 24 months. This suggests that, although the intervention goal of 3 h/week of moderate intensity exercise was enough to improve insulin sensitivity, it was not long enough and/or of sufficient intensity to elicit the necessary magnitude of change in insulin sensitivity for this to be translated into a significant reduction in 2-h plasma glucose.

Overall, non-significant results were seen in all but two of the studies that measured 2-h plasma glucose before and after the intervention. However, despite the link between 2-h plasma glucose and diabetes risk, it does not follow that the risk of diabetes was unchanged in these studies, as demonstrated by the FDPS, which reported a non-significant change in 2-h plasma glucose over the course of the intervention but a >50% reduction in the risk of diabetes [21]. One reason for this discrepancy is likely to be the poor repeatability of 2-h plasma glucose values [32], and given the relatively small sample sizes in most of these studies, it is possible that improvements in glucose tolerance were not detected using 2-h plasma glucose. The glucose AUC has been identified as a more reliable measure of glucose tolerance than 2-h plasma glucose [33], and is therefore a more sensitive measure of glucose tolerance. One study included in this review measured both 2-h plasma glucose and glucose AUC at baseline and follow-up [20]. It reported that, although 2-h plasma glucose did not change significantly at any of the follow-up time points, there was a significant reduction in the glucose AUC at 6 months, and a trend towards significance at 24 months.

Given the failure of the lifestyle interventions to substantially increase physical activity levels, and the inconclusive result of the structured exercise training study, the role of physical activity independent of other lifestyle changes in the treatment of prediabetes remains equivocal. However, statistical analysis of the independent effects of physical activity, which has been carried out on some of the lifestyle intervention studies included in this review, show interesting results. For example, the conclusion of the Malmö Feasibility Study that cardiovascular fitness and weight loss were equally correlated to improved glucose tolerance is supported by data from the Study on Lifestyle Intervention and Impaired Glucose Tolerance Maastricht (SLIM) [34], and a recent analysis of data from the FDPS found a 49% difference in the risk of diabetes, after adjustment for changes to body mass and diet, when comparing those in the highest and lowest tertiles of moderate to vigorous leisure time physical activity change [35]. Thus, although the overall evidence for the independent effect of physical activity in the management of prediabetes is equivocal, encouraging evidence is starting to emerge in support of the importance of exercise.

Given the limitations of the studies included in this review it is not possible to make any recommendations as to the intensity and duration of exercise needed to improve glucose tolerance and/or reduce the risk of diabetes in individuals with IGT, independently of other lifestyle changes. The equivocal nature of the evidence is reflected in the advice given by the ADA, which recommends that individuals with IGT should include 150 min/week of moderate to vigorous intensity exercise as part of a weight management programme [36]. However, the aforementioned analysis of the change in physical activity in the FDPS found that a difference of 246 min/week in median values between those in the lowest and the highest tertiles of moderate to vigorous physical activity change was associated with a significant reduction in the risk of diabetes, after adjusting for changes in diet and body mass. However, the difference of 120 min/week in median values between the lowest and middle tertiles was not associated with a reduced risk of diabetes [35]. Although this result was obtained by analysing the pooled cohort, and therefore provides little information about the effectiveness of the intervention itself, it does suggest that 150 min/week of moderate to vigorous intensity exercise is unlikely to be enough to significantly reduce the risk of type 2 diabetes in individuals with IGT, independently of other lifestyle changes. However, given that this analysis relied on self-reported physical activity levels, further rigorous studies are needed to confirm this.

All studies included in this review selected individuals using IGT as an inclusion criteria. Therefore, any conclusions from this review can only be applied to individuals with IGT and it is impossible to determine whether or not exercise may be effective in treating individuals with isolated IFG. However as individuals with isolated IFG account for a
minority of individuals with prediabetes [37], conclusions about the effect of exercise on IGT drawn from this review will apply to the majority of individuals with prediabetes.

In summary, the majority of studies identified for this review used interventions that encouraged dietary and physical activity to initiate and maintain weight loss in individuals with IGT. Analysis of these studies found that the independent effect of physical activity in reducing the risk of type 2 diabetes in individuals with prediabetes is equivocal. Furthermore, given the limited evidence, no definite conclusion can be drawn either as to the amount of physical activity needed to reduce the risk of diabetes in individuals with prediabetes or the effectiveness of a single-component physical activity intervention compared with more conventional multi-component interventions.

Thus, more evidence from rigorously designed randomised controlled trials with objective measures of physical activity is needed. As the majority of studies promoting lifestyle changes included in this review failed to substantially increase physical activity levels, strategies for effecting increased physical activity in this population also need to be researched thoroughly. Further investigation is also needed into whether exercise is equally effective in treating individuals with prediabetes or the efficacy of a single-component physical activity intervention compared with more conventional multi-component interventions.

Duality of Interest There was no duality of interest in the writing of this review.

References

Increased physical activity is a cornerstone in the prevention of type 2 diabetes in high-risk individuals. Reply to Laaksonen DE, Lindström J, Tuomilehto J, Uusitupa M [letter]

T. Yates • K. Khunti • F. Bull • T. Gordy • M. Davies

We agree that the success of the Finnish Diabetes Prevention Study (FDPS) in achieving a large decrease in the number of sedentary individuals is encouraging; however, the benefits of this decrease have yet to be quantified. Laaksonen et al. [1] do not agree with our conclusion that the 9 min/day increase in physical activity found in the FDPS was clinically insignificant. To our knowledge, no randomised-controlled trial has found substantive clinical benefit from an increase in physical activity of this magnitude, independent of other lifestyle changes. Indeed, data from the FDPS found that a difference of twice this magnitude in physical activity change (2 h/week) was not associated with a reduced risk of diabetes after adjusting for other lifestyle variables [4].

Laaksonen et al. [1] question our conclusion that current exercise recommendations may be of insufficient magnitude to reduce the risk of diabetes, independent of other factors. Specifically, they go on to state that at least 2.5 h/week of walking or moderate-to-vigorous exercise is associated with a decrease in the risk of diabetes. However, 2.5 h/week of moderate-to-vigorous intensity exercise in the FDPS was not associated with a decreased risk of developing diabetes after adjustment for likely confounders [4]. Furthermore, these results were gained by analysing the association of diabetes risk with physical activity levels at follow-up, rather than physical activity change. We accept that the epidemiological evidence for the association between exercise and risk of development of diabetes is well established. However, the key issues for health professionals are whether physical activity change can affect the risk of diabetes independent of other lifestyle factors, such as weight loss, and whether free-living physical activity can
be successfully promoted in at-risk individuals using cost-effective methods. It is these questions that we feel the current evidence from randomised controlled trials is unable to answer.

Laaksonen et al. [1] also question our extrapolation of data from exercise intervention trials in individuals with diabetes to infer that modest increases in physical activity in individuals with pre-diabetes are unlikely to result in weight loss. However, as we highlighted [2], there is growing consensus among national and global health agencies, including the World Health Organization [5], that about 60 min of moderate intensity exercise on most days of the week is needed to achieve long-term energy balance in the general population [6]. On this basis, although we agree that physical activity can be used to initiate weight loss, we are uncertain whether this can be achieved with the current exercise recommendations of at least 30 min of moderate intensity exercise on most days of the week. Laaksonen et al. go on to cite unreported data from the FDPS showing that the weight-loss difference between those in the highest and lowest tertiles of moderate-to-vigorous exercise change was 2.1 kg [1]. In fact, there is no disagreement here. Published data from the FDPS show that the difference in median values of moderate-to-vigorous physical activity change between those in the lowest and highest tertiles was more than 4 h/week [4], a value that is substantially higher than the current exercise recommendations. Thus the unreported data from the FDPS do not provide evidence that levels of physical activity that are in line with the current exercise recommendations are sufficient to initiate weight loss.

We agree that there is a need to distinguish between change in 2 h and fasting glucose values and the risk of developing diabetes, particularly over the longer term, as treatment strategies for diabetes are likely to confound any difference in glucose levels between groups. As we pointed out [2], the area under the glucose curve may be a more sensitive outcome measure than 2 h and fasting glucose. However, the oral glucose tolerance test is recognised as the gold standard method for assessing glucose control status and 2 h glucose values have been shown to be a strong predictor of cardiovascular disease risk [7]. We therefore feel that 2 h glucose is an appropriate main outcome in smaller and/or shorter-term trials on the effect of exercise in those with pre-diabetes.

Publications from the FDPS have made a major contribution to our understanding of the impact of physical activity in reducing the risk of developing diabetes in high-risk populations. The purpose of our review [2] and this response to the letter by Laaksonen et al. [1] was not to cast doubt on the link between physical activity and risk of type 2 diabetes, but to highlight some gaps in the current evidence. We welcome the comments by Laaksonen et al. and hope that continued debate and research into some of the issues identified in our review [2], highlighted in the commentary by Carnethon [8] and challenged in the letter from Laaksonen et al. [1], will help lead to a greater understanding of how physical activity can be utilised to its full potential in helping to out-run the diabetes epidemic.

References

Evidence shows that successful lifestyle modification programmes are fundamental to slowing and/or stopping the progression to diabetes in high-risk populations and preventing and/or reducing the complications associated with type 2 diabetes. This article will explore the issues surrounding the promotion of physical activity in primary care. Although the main focus of the article is prevention, the issues and strategies described in this article are equally applicable to individuals with type 2 diabetes, especially those who are newly diagnosed.

The prevalence of diabetes is reaching epidemic proportions and the costs associated with its treatment are set to represent a serious clinical and financial challenge to the UK’s health system (Bagust et al, 2002). Individuals with pre-diabetes have a significantly increased risk of developing diabetes and cardiovascular disease compared with those with normal glucose tolerance (Unwin et al, 2002) and are therefore likely to form a significant proportion of the healthcare burden associated with diabetes in the future. Pre-diabetes is the collective term for people with impaired glucose tolerance and/or impaired fasting glucose (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2003). It is of primary importance to counter this worrying trend by identifying strategies that are appropriate to local primary healthcare services and infrastructure (Davies et al, 2002).

Although the increasing prevalence of diabetes and its co-morbidities does have a genetic component, deleterious effects are only seen in environments where energy-dense food is plentiful and the link between physical activity and food procurement has been broken. Such environments have been termed ‘toxic’ in both the scientific press and mainstream media. The recent technological revolution and its associated plethora of labour-saving devices, and the reduction in jobs requiring manual labour have lead to a physically inactive society far removed from the high energy expenditures on which the human phenotype evolved (Cordain et al, 1998). Consequently, physical inactivity is one of the most important factors contributing to the
Can structured education be used to promote physical activity in primary care?

Page points

1. There is good evidence that increased physical activity, even without weight loss, can improve glycaemic control substantially in individuals with type 2 diabetes.

2. Clinicians and researchers need to develop successful ways of promoting physical activity, not only in individuals with type 2 diabetes.

3. Physical activity interventions need to promote forms of physical activity that are appropriate and acceptable to their target populations.

4. Data from epidemiological and intervention studies in the UK and other developed countries have consistently shown walking to be the preferred choice of physical activity in the general as well as diseased populations.

5. A simple and cheap way of promoting walking activity is to use pedometers.

Walking – the best exercise

Physical activity interventions need to promote forms of physical activity that are appropriate and acceptable to their target populations. It is of little practical benefit to promote gym-based physical activity interventions if the majority of the individuals with the most to gain are unable or unwilling to access their local gyms. This is likely to explain the poor take up of and adherence to some exercise on prescription schemes (Thurston & Green, 2004). Data from epidemiological and intervention studies in the UK and other developed countries have consistently shown walking to be the preferred choice of physical activity in the general as well as diseased populations (Crespo et al, 1996; Booth et al, 1997; Cooper et al, 2000; Di Loreto et al, 2003). Interventions that promote walking activity have been shown to improve glycaemic control and cardiovascular risk markers in individuals with diabetes (Di Loreto et al, 2003) and epidemiological data have shown that as little as 30 minutes of walking activity per day has a significant impact on the risk of diabetes compared with being sedentary, even after adjustment for body mass and other likely confounding variables (Hu et al, 1999). Walking would therefore seem to be an appropriate mode of exercise to use when promoting physical activity in at-risk individuals. It is also likely that walking will be associated with fewer barriers than other forms of physical activity in black and minority ethnic populations (Johnson, 2000).

A simple and cheap way of promoting walking activity is to use pedometers. Pedometer interventions have been successful at initiating physical activity behaviour change in individuals with diabetes (Tudor-Locke et al, 2004) and those at risk of diabetes (Swartz et al, 2003). When using pedometers in the promotion of physical activity, it is important to work with patients to set realistic and attainable goals. For example, promoting the popular 10000 steps-per-day target in someone who normally takes only 3000 steps per day is likely to be inappropriate and demotivating; it is important that individual goals are based on normal activity levels. Table 1 gives an overview of activity categories based on the number of steps per day. The immediate goal of the clinician or healthcare professional should be to help individuals move up an activity category. For example, a sedentary individual taking 3000 steps per day should be encouraged to increase their activity levels to over 5000 steps per day. This should be achieved gradually, by increasing activity levels in small weekly increments until the target amount is reached. Along with setting realistic goals, it is important that individuals keep a daily log of their steps per day. This should be reviewed with the clinician or health professional at subsequent appointments. Clinicians should also consider using pedometers with proven reliability and validity (Schneider et al, 2004), as patients may become demotivated if the pedometer they are
Can structured education be used to promote physical activity in primary care?

Interventions to increase physical activity need to take into account the current healthcare climate and make use of existing strategies that have already been used to successfully promote self-management in people with chronic disease. Interventions that use a patient-centred approach to education are increasingly being recognised as both appropriate and successful in a UK primary healthcare setting (DoH and Diabetes UK, 2005). Structured educational programmes delivered to small groups of participants are also likely to be a cost-effective method of health promotion (NICE, 2003); this is important given that the resource-intensive methods used in the Diabetes Prevention Programme and other successful diabetes prevention programmes are unlikely to be cost effective in a real world primary healthcare setting (Ikels et al, 2007).

Health behaviour theory

It is important that structured educational programmes aimed at health promotion are based on known learning techniques and health behaviour theory (DoH and Diabetes UK, 2005). Therefore, educational programmes that are designed around physical activity promotion need to be grounded in appropriate healthcare theory and delivered using patient-centred learning techniques. Physical activity research has typically failed to consider or adequately describe a theoretical justification for their chosen approach, which has made it more difficult to understand why a given approach may fail or succeed.

The PREPARE programme

In order to address some of the issues highlighted in this article and to target some of the gaps in the current evidence around physical activity and diabetes prevention (Yates et al, 2007), we have designed a programme called the Pre-diabetes Risk Education and Physical Activity Encouragement (PREPARE) programme, which is a theory-driven, structured educational programme designed to promote increased levels of walking activity in individuals identified as having pre-diabetes using methods appropriate for a primary health care setting.

The PREPARE programme is based on the approach to patient education that was developed for the Diabetes Education and Self Management for Ongoing and Newly Diagnosed (DESMOND) programme, which is recognised by the DoH (DoH and Diabetes UK, 2005) as being the only national structured educational programme for individuals with type 2 diabetes that meets the key criteria identified by NICE for effective patient education (NICE, 2003). The DESMOND programme has been shown to be successful at targeting illness perceptions (Skinner et al, 2006) and promoting physical activity (Skinner et al, 2005).

Drawing on the knowledge and expertise of the DESMOND collaborative, the PREPARE programme aims to promote physical activity by targeting perceptions and knowledge of pre-diabetes, self-efficacy beliefs, and perceived barriers surrounding walking activity. As self-regulation is the key to success in any structured educational programme, the PREPARE programme also helps participants to: form realistic personalised goals; develop strategies for success by planning when, where and how active they want to be; and take into account the current healthcare climate and make use of existing strategies that have already been used to successfully promote self-management in people with chronic disease.

Table 1. Physical activity categories based on steps per day (adapted from Tudor-Locke & Bassett, 2004).

<table>
<thead>
<tr>
<th>Category</th>
<th>Steps per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedentary</td>
<td>&lt;5000</td>
</tr>
<tr>
<td>Low (typical of daily activity excluding volitional activity)</td>
<td>5000–7499</td>
</tr>
<tr>
<td>Moderate (likely to incorporate the equivalent of around 30 minutes per day of moderate intensity physical activity)</td>
<td>7500–7599</td>
</tr>
<tr>
<td>High (likely to incorporate the equivalent of around 45 minutes per day of moderate intensity physical activity)</td>
<td>10000–12499</td>
</tr>
<tr>
<td>Very high (likely to incorporate the equivalent of over 45 minutes per day of moderate intensity physical activity)</td>
<td>&gt;12500</td>
</tr>
</tbody>
</table>
Can structured education be used to promote physical activity in primary care?

Page points

1. Pilot data suggest that the PREPARE programme is successful at increasing perceived knowledge of pre-diabetes and initiating physical activity behaviour change in individuals with pre-diabetes.

2. The PREPARE programme is currently being tested in a randomised, controlled trial funded by Diabetes UK.

3. If we are to stem the rising tide of diabetes and its associated complications, it is essential that physical activity is recognised as a lifestyle variable of primary importance and promoted using strategies that are applicable and cost effective in a primary healthcare setting, and appropriate across a wide range of abilities and cultures.

and how they will achieve their goals; and monitor their behaviour using pedometers. Total contact time for the PREPARE programme is 3 hours. Pilot data suggest that the PREPARE programme is successful at increasing perceived knowledge of pre-diabetes and initiating physical activity behaviour change in individuals with pre-diabetes (Yates et al, 2007a).

The PREPARE programme is currently being tested in a randomised, controlled trial funded by Diabetes UK. The trial is powered to detect a 1 mmol/l difference, over 1 year, in 2-hour glucose levels in individuals with impaired glucose tolerance. Physical activity levels will be assessed using self-report and medical-grade piezoelectric pedometers. Additional outcomes will include blood lipids and standard anthropometric measurements. Taken together, these outcomes will help inform clinicians and health professionals as to whether or not physical activity can be promoted successfully using structured education and if any observed increase in physical activity leads to changes in traditional markers of diabetes or cardiovascular disease risk.

Conclusion

If we are to stem the rising tide of diabetes and its associated complications, it is essential that physical activity is recognised as a lifestyle variable of primary importance and promoted using strategies that are applicable and cost effective in a primary healthcare setting, and appropriate across a wide range of abilities and cultures. It is for this purpose that we have designed the PREPARE programme, which we hope will provide a successful and appropriate method of promoting increased walking activity in usual healthcare practice.


Hillston M, Foster C, Thorogood M (2009) Interventions for promoting physical activity. Cochrane Database of Systematic Reviews CD003180


Diabetes & Primary Care Vol 9 No 5 2007
Can structured education be used to promote physical activity in primary care?


Skinner TC, Davies MJ, Heller S, Khunti (2005) To determine the effects of a structured education programme on illness beliefs, quality of life and physical activity in individuals newly diagnosed with Type 2 diabetes results from the DESMOND (Diabetes Education and Self Management for Ongoing and Newly Diagnosed) pilot study. Diabetic Medicine 22(Suppl 1): 15


Diabetes & Primary Care Vol 9 No 5 2007

Page points

1. XXX

2. XXX

3. XXX

4. XXX
Prevention of diabetes: A reality in primary care?

Type 2 diabetes mellitus is a growing health problem, with the prevalence of the disease set to rise dramatically in westernised societies [1]. Individuals with diabetes have a life expectancy that can be shortened by as much as 15 years with the majority dying of cardiovascular disease [2]. The excess mortality, morbidity and the substantial healthcare costs associated with its rising prevalence make diabetes a major global health issue. Intervening to delay or prevent type 2 diabetes has the potential to improve health and to reduce the burden of healthcare costs. The total direct medical cost of managing diabetes in eight European countries is estimated at 29 billion euros a year (1999 value) [3]. This cost is projected to substantially increase in line with the number of individuals identified with diabetes in the coming decades. It is therefore of primary importance to identify strategies that can successfully counter this worrying trend. Several successful diabetes prevention trials [4,5] that have captured the attention of clinicians, governments and the media over the past decade have highlighted the importance of promoting lifestyle change in the fight against diabetes.

Individuals diagnosed with impaired glucose tolerance have a high risk of developing type 2 diabetes and many trials of interventions for prevention of type 2 diabetes have focused on such individuals. A recent systematic review concluded that lifestyle and pharmacological interventions reduce the rate of progression to type 2 diabetes in individuals with impaired glucose tolerance [6]. However, despite promising findings from these research studies, the gap between what is known about diabetes prevention and what is commonly practiced in primary health care settings is considerable. Bridging that gap is essential if we are to stem the rising tide of diabetes. We believe that there are several areas that researchers and clinicians need to address in order to achieve this and to encourage the continued shift in national policy strategies towards prevention.

1. Regional applicability

It has been widely reported that the Diabetes Prevention Programme (DPP) is a cost effective method of reducing the risk of diabetes in high-risk individuals in the United States and other developed countries [3,7]. The problem with these analyses is that not only do they suffer from the inherent problems of projecting findings from research populations onto the general population, they also assume that the programme would be equally effective across diverse cultural and regional populations. The inherent flaws in this assumption have been highlighted in the United Kingdom, where interventions similar to the DPP have not resulted in meaningful lifestyle or clinical change [8,9], a trend that may be explained by several locally specific factors and issues [10]. Furthermore, it has been shown that even if programmes like DPP are shown to be effective, they may not be cost-effective in "real world" European routine health care settings [11]. There is therefore a need to design and test programmes tailored to the specific needs of local communities that are appropriate for local primary health care services and infrastructure. To this end, more research is needed to test interventions in multiple and representative settings.

2. Identifying those at risk

Successful diabetes prevention studies have tended to include participants based on 2-h glucose values. However, oral glucose tolerance tests are not routinely carried out in most health care settings and their inclusion would represent a significant burden on health care resources and patient time. Indeed it has been pointed out that a true primary prevention programme would identify and treat those at risk of diabetes before glycaemic control becomes impaired [12], an aim that is all the more important given the increased risk of cardiovascular disease seen in individuals with pre-diabetes. Validated methods that utilise data routinely gathered in primary care are therefore needed to identify individuals at risk of diabetes. The validated FINRISK score developed in Finland [12] is a good example of how diabetes risk can be assessed using non-invasive data from measurements that could easily be incorporated into primary care without putting an additional burden on overstretched health care resources. The continued
development and testing of such tools is needed in ethnically diverse settings.

3. Self-regulation—the key to maximising behaviour change

Even interventions that have successfully reduced the risk of diabetes by means of lifestyle change have been unsuccessful at effecting clinically significant changes in some important lifestyle variables such as physical activity [13] and little attempt has been made to identify which health behaviours have the greatest impact on the risk of diabetes. Therefore continued research following clearly defined national frameworks is needed to develop complex interventions that are designed to identify how lifestyle change can be maximised. Over recent decades, substantial advances have been made in our understanding of the link between lifestyle factors and the biological processes that determine health and illness, yet our understanding of health behaviours and behaviour change has seen little significant innovation over the same period [14]. This is likely to be due to several factors, including the sheer number of health behaviour theories and the plethora of competing determinants and models that an interventionist must choose from when designing a behaviour change intervention. However, it is becoming increasingly clear that promoting self-management in a patient centred framework is of primary importance in effecting behaviour change [15, 16]. The key to self-management is self-regulation, what this means in practice is that clinicians should help patients to make lifestyle change action plans that are realistic and tailored to the individual. Individuals should also be encouraged to self-monitor their behaviour on a regular basis. For example, an individual wanting to increase their physical activity levels should be helped to: form realistic goals that are individual to them, develop strategies to meet their goals by planning when, where and how their daily goals will be met; and where possible to objectively monitor their behaviour, such as by using a pedometer, in order to assess how successful they are in achieving their goals. The importance of self-regulation in promoting a healthy lifestyle is likely to be applicable across diverse cultural groups. However, self-regulatory skills should be promoted using culturally sensitive methods.

4. Conclusion

Although good quality randomised controlled trials have demonstrated that stopping and/or slowing the progression to diabetes is possible in high risk populations, we believe that successfully translating these results into diabetes prevention programmes that can be used in a primary care setting requires that: (i) at risk individuals are identified using methods that are appropriate to local primary care settings, and (ii) interventions are tailored to local health care and population norms and (iii) are grounded in appropriate health behaviour theory. Only then will the gap between research and health care delivery be successfully bridged.

REFERENCES

Walking and inflammatory markers in individuals screened for type 2 diabetes

Thomas Yates a,*, Melanie Davies b, Emer Brady b, David Webb b, Trish Gorely a, Fiona Bull c, Duncan Talbot c, Naveed Sattar d, Kamlesh Khunti e

* School of Sport and Exercise Sciences, Loughborough University, Ashby Road, Loughborough, Leicestershire, LE11 3TB, UK
b University Corporate Research, Colworth Park, Bedfold, UK
c Department of Vascular Biochemistry, University of Glasgow, UK
d Department of Health Sciences, University of Leicester, UK

A R T I C L E I N F O
Available online xxx
Keywords:
Adipocytokine
C-reactive protein
Exercise
Inflammation
Insulin
Interleukin-6
Physical activity
Tumor necrosis factor-α
Walking

A B S T R A C T
Objective. To investigate the association of walking activity with inflammatory markers and fasting insulin in a bi-ethnic population screened for type 2 diabetes in Leicester, United Kingdom, between 2005 and 2006.

Method. Physical activity, adipocytokine, high-sensitivity C-reactive protein and fasting insulin measurements were available for 400 individuals screened for type 2 diabetes. Of the 400 participants, 56% were diagnosed with normal glucose control, 36% with prediabetes and 8% with diabetes.

Results. Multivariate analysis showed that those who reported walking for as little as 30 min on at least 5 days per week had lower levels of C-reactive protein, interleukin-6, and tumor necrosis factor-α compared to those who reported lower walking activity levels, after adjustment for other modes of moderate-to-vigorous intensity physical activity, age, ethnicity, sex, social deprivation and smoking status. Further adjustment for waist circumference attenuated the association of walking with tumour necrosis factor-α.

Conclusion. Walking activity, independent of other forms of physical activity, is associated with lower levels of circulating pro-inflammatory markers.

© 2008 Elsevier Inc. All rights reserved.

Introduction

Markers of chronic low-grade inflammation, such as tumor necrosis factor-α (TNFα), interleukin-6 (IL-6), and C-reactive protein (CRP), have been shown to predict the risk of developing type 2 diabetes and cardiovascular disease and are thought to be directly involved in the pathogenesis of these chronic diseases (Libby et al., 2002; Pickup and Cook 1998; Xu et al. 2003). TNFα and IL-6 are cytokines which are predominantly secreted from adipose tissue while CRP is the principal downstream mediator of the acute phase response and is secreted by the liver in response to TNFα and IL-6 stimuli (Du Clos 2000). Circulating levels of these markers of chronic low-grade inflammation are therefore largely influenced by levels of adiposity and have been proposed as an important mediating link between obesity and chronic disease (Berg and Scherer 2005). However, recent evidence has shown that levels of physical activity are also inversely and independently associated with TNFα, IL-6, and CRP (Panagiotakos et al. 2005; Pischon et al. 2003; Wannamethee et al. 2002). As levels of physical activity have also consistently been shown to be inversely associated with the risk of developing both type 2 diabetes and cardiovascular disease (Bassuk and Manson 2005), markers of chronic low-grade inflammation could be a mediating link between levels of physical inactivity and chronic disease. However, the effect of walking, independent of more vigorous forms of exercise, on inflammatory markers is not well documented. This is an important limitation as walking has been shown to be the preferred choice of physical activity for the majority of individuals (Crespo et al. 1996), and walking for as little as 150 min/week has been associated with a reduction in the relative risk of developing type 2 diabetes and cardiovascular disease (Huet al. 1995; Laaksonen et al. 2005; Manson et al. 2002). The aim of this study is to investigate the effect of walking on key markers of chronic low-grade inflammation associated with the development of type 2 diabetes and cardiovascular disease, along with fasting insulin, in a bi-ethnic population screened for type 2 diabetes. Our hypothesis was that walking, at levels that are consistent with the current exercise recommendations, would be independently associated with reduced chronic low-grade inflammation.

Methods

The ADDITION study is a Europe-wide screening and treatment programme for type 2 diabetes (Sandbaek et al. 2008). Between 2005 and 2006, 573 (m = 304, f = 269) individuals screened as part of the Leicester ADDITION study also consented for a sub-study which...
additional blood samples were taken for the analysis of inflammatory biomarkers. The average age of the participants was 62 ± 9 years and 24% were from a South Asian ethnic background.

Physical activity

Physical activity was measured using the short last-seven-days self-administered format of the International Physical Activity Questionnaire (IPAQ) (Craig et al., 2003). IPAQ measures the frequency and duration of the moderate-to-vigorous intensity physical activity undertaken for more than 10 continuous minutes across all contexts (e.g. work, home and leisure) over a seven day period. Importantly, IPAQ distinguishes walking activity from other forms of moderate-to-vigorous intensity physical activity across all contexts. It also enables the calculation of metabolic equivalents (MET-hours/week). IPAQ has been UK. In this case, the reliability was calculated with a coefficient (p = 0.8) with accelerometer data in the United Kingdom (Craig et al., 2003a). Walking activity categories were formed by distinguishing between those who reported walking for at least 30 min on at least 5 days/week and those who reported walking for less than this.

Biochemical, clinical and demographic measurements

Venous blood samples were collected in the morning following an overnight fast. Participants were not asked to avoid vigorous intensity physical activity before attending. Prediabetes and diabetes were defined according to the American Diabetes Association’s 1997 criteria (The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 1997). Diagnosis of diabetes was confirmed on a repeat oral glucose tolerance test.

All assays were measured by individual blinded to the patients’ identity. Plasma glucose was measured using a glucose oxidase method on the Beckman Auto Analyzer (Beckman, High Wycombe, UK). High-sensitivity C-reactive protein (hsCRP) was analysed on an ABX Pentra clinical chemistry analyser using a latex-enhanced immunoturbidimetric assay (Horiba Group, Montpellier, France). TNFα and IL-6 were analysed using quantitative high-sensitivity enzyme linked immunosorbant assays (LISA; R&D Systems, Abingdon, UK). Insulin was analysed using a Perkin Elmer time-resolved fluoro-immunoassay on the AutoDELFIA. The intraassay and interassay coefficients of variation for the included assays did not exceed 16.7.

Arterial blood pressure was measured in the sitting position (Omron Healthcare, Henfield, UK); three measurements were obtained and the average of the last two measurements was used. Body weight (Tanita TBF 611, Tanita, West Drayton, UK); waist circumference (midpoint between the lower costal margin and iliac crest) and height were also measured to the nearest 0.1 kg and 0.5 cm respectively.

Current smoking status and medication history were obtained by an interview-administered protocol. For the purposes of this study participants were defined as non-smokers, past smokers, or current smokers (≥ 1 cigarette/day). Blood pressure and statin medication status was defined by whether or not participants were currently taking these medications.

Social deprivation was determined by assigning an Index of Multiple Deprivation (IMD) score to each participant’s postcode (Office for the Deputy Prime Minister 2004). IMDs are publicly available continuous measures of compound social and material deprivation, and are calculated using a variety of data including current income, employment, health, education, and housing.

Statistical analysis

Variables are presented as mean ± standard deviation. Chi-squared tests were used to analyse categorical variables, independent t-tests were used to analyse normally distributed continuous variables and Mann-Whitney tests were used to analyse non-parametric variables. Multivariate analysis of variance was used to analyse the associations between walking activity categories and hsCRP, IL-6, TNFα, and insulin. Due to their skewed distribution, all dependant variables in the multivariate analysis were log-transformed. Multivariate analysis models were adjusted for non-walking physical activity levels and measured demographic variables (age, sex, ethnicity and social deprivation). Further adjustment was carried out for medication and smoking status if the inclusion of these factors as covariates in the multivariate analysis changed the coefficient for walking status by 10% or more for any of the included dependant variables (Maldonado and Greenland 1993). In addition, further adjustment was also made for waist circumference in order to establish the independent effect of walking activity status. All analyses were two sided; p < 0.05 was considered significant. Analysis was carried out on SPSS 12.0 for Windows (SPSS, Chicago, USA).

Results

Complete physical activity data was available for 400 (70%) participants. Of these participants, 142 (36%) had prediabetes, 33 (8%) had diabetes and 15 (4%) had a previous history of myocardial infarction, stroke or angina. Those that completed the questionnaire

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample (n=400)</th>
<th>Low walking activity (n=191)</th>
<th>High walking activity (n=209)</th>
<th>P for difference between groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walking activity (MET-hours/week)</td>
<td>15.4 (42.9)</td>
<td>3.3 (9.8)</td>
<td>35.8 (52.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Moderate to vigorous physical activity (including walking activity) (MET-hours/week)</td>
<td>0 (3.6)</td>
<td>0 (8.0)</td>
<td>8.2 (44.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>209 (52)</td>
<td>93 (49)</td>
<td>116 (55)</td>
<td>0.17</td>
</tr>
<tr>
<td>Female</td>
<td>191 (48)</td>
<td>98 (51)</td>
<td>93 (45)</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White European</td>
<td>307 (77)</td>
<td>135 (71)</td>
<td>172 (82)</td>
<td>0.01</td>
</tr>
<tr>
<td>South Asian</td>
<td>93 (23)</td>
<td>56 (29)</td>
<td>37 (18)</td>
<td></td>
</tr>
<tr>
<td>Index of Multiple Deprivation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Score</td>
<td>192 (1.18)</td>
<td>186 (1.18)</td>
<td>210 (1.18)</td>
<td>0.23</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure medication</td>
<td>61.8 ± 9.3</td>
<td>52.2 ± 9.0</td>
<td>61.4 ± 9.3</td>
<td>0.64</td>
</tr>
<tr>
<td>(mg/dl)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(medication and no medication)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Betablockers</td>
<td>52 (13)</td>
<td>27 (14)</td>
<td>25 (12)</td>
<td>0.12</td>
</tr>
<tr>
<td>Angiotensin converting enzyme inhibitors</td>
<td>24 (6)</td>
<td>10 (5)</td>
<td>14 (7)</td>
<td></td>
</tr>
<tr>
<td>Statin medication</td>
<td>59 (14.8)</td>
<td>32 (16.8)</td>
<td>27 (12.9)</td>
<td>0.27</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>140 ± 21</td>
<td>143 ± 22</td>
<td>142 ± 29</td>
<td>0.83</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>86 ± 11</td>
<td>87 ± 12</td>
<td>85 ± 11</td>
<td>0.64</td>
</tr>
<tr>
<td>Current smokers</td>
<td>45 (11)</td>
<td>16 (8)</td>
<td>29 (14)</td>
<td>0.08</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.3 ± 6.6</td>
<td>29.4 ± 4.4</td>
<td>29.3 ± 4.6</td>
<td>0.91</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>98.8 ± 12.7</td>
<td>98.5 ± 11.3</td>
<td>98.5 ± 11.3</td>
<td>0.39</td>
</tr>
<tr>
<td>Fasting insulin (μU/ml)</td>
<td>78 (6.1)</td>
<td>7.9 (6.5)</td>
<td>7.2 (5.8)</td>
<td>0.05</td>
</tr>
<tr>
<td>Tumor necrosis factor-α (pg/ml)</td>
<td>19 (1.3)</td>
<td>21 (1.4)</td>
<td>17 (1.3)</td>
<td>0.01</td>
</tr>
<tr>
<td>Interleukin-6 (pg/ml)</td>
<td>19 (1.8)</td>
<td>2.2 (2.4)</td>
<td>1.8 (1.4)</td>
<td>0.01</td>
</tr>
<tr>
<td>High-sensitivity C-reactive protein (mg/dl)</td>
<td>2.3 (4.7)</td>
<td>2.3 (5.1)</td>
<td>2.3 (4.9)</td>
<td>0.16</td>
</tr>
</tbody>
</table>

Participants were screened for type 2 diabetes in Leicester, United Kingdom, between 2003 and 2006. Categorical variables as number (column percentage) continuous parametric results as mean±SD and continuous non-parametric results as median (interquartile range).

were more likely to have normal glucose control and were more likely to come from less deprived areas than non-completers. No significant differences in inflammatory markers, age or ethnicity was observed between completers and non-completers. Data is reported for the subset of 400 participants who completed the physical activity questionnaire.

There was no significant difference between groups in the incidence of diabetes, prediabetes or those with a history of myocardial infarction, stroke or angina. Table 1 shows the characteristics of the study participants overall and according to their walking status. Compared to White Europeans, those from a South Asian ethnic background were less likely to report walking for 30min on at least 5 days a week.

Multivariate statistical analysis found that those who reported walking for at least 30 min on at least 5 days a week had lower IL-6, hsCRP and TNFα levels compared to those who reported lower walking activity; after adjustment for other modes of moderate-to-vigorous intensity physical activity, age, ethnicity, sex, social deprivation and smoking status (Fig. 1). Further adjustment for waist circumference attenuated the association with TNFα, although the association with IL-6 and hsCRP remained significant. There was no association between fasting insulin and walking status. All results were unaffected by the inclusion of statin or blood pressure medication status as covariates in the statistical analysis.

Discussion

In this cross-sectional study of individuals screened for type 2 diabetes, walking on at least 5 days/week for at least 30 min/day was associated with lower circulating IL-6, TNFα and hsCRP levels after adjustment for other modes of physical activity, demographic variables and smoking status. Further adjustment for waist circumference attenuated the association of walking categories with TNFα, although the association with IL-6 and hsCRP remained significant.

Although other studies have shown that walking for around 150 min/week is associated with a reduced risk of developing type 2 diabetes and cardiovascular disease (Hu et al. 1999, Laaksonen et al. 2005, Manson et al. 2002), to our knowledge this is the first study to investigate the effect of walking on inflammatory markers after adjustment for other forms of physical activity. As chronic low-grade inflammation is thought to play an important role in the pathogenesis of type 2 diabetes and cardiovascular disease (Libby et al. 2002, Pickup and Crook 1998, Xu et al. 2003), this study suggests that increased walking activity may reduce the risk of developing a debilitating chronic disease through reduced systemic inflammation. This is clinically important as, for the majority of individuals, walking is the most accessible form of physical activity. The findings reported in this study are consistent with other studies which have shown that...
overall levels of moderate-to-vigorous intensity physical activity are inversely associated with markers of chronic low-grade inflammation (Panagiotakos et al., 2005, Fischer et al., 2003, Wannamethee et al., 2002).

This study further emphasizes the importance of promoting walking activity to levels that are consistent with the current physical activity recommendations in sedentary populations, particularly in those identified with an increased risk of developing a chronic disease. However, there is little evidence that traditional methods of promoting health behaviour change in at-risk populations, such as established diabetes prevention programmes (Carnethon et al., 2007, Yates et al., 2007), have been successful at promoting clinically significant increases in physical activity. Therefore there is a continuing need to develop and test innovative strategies for promoting physical activity, in particular walking activity, in health care and community settings.

The exact mechanisms linking physical activity to reduced inflammation have not been well described. However several studies have shown that exercise training does not affect cytokine production from adipose tissue (Klimcakova et al., 2006, Polak et al., 2006), although it may alter cytokine production from mononuclear cells, another important source of cytokines (Smith et al., 1999). It has been hypothesized that the release of cytokines (cytokines released from muscle, such as IL-6) from exercising muscle may, when performed regularly, cause adaptations to the immune system resulting in lower levels of cytokines being released from mononuclear cells (You and Nicklas 2008), which in turn would reduce the production of CRP from the liver. However, as the interactive effects of exercise, muscle, body fat, and markers of chronic low-grade inflammation and the development of metabolic and vascular dysfunction are poorly defined (Telford 2007), more research is needed to quantify the overall significance of the findings from this study.

The finding that walking activity status was not associated with fasting insulin levels is in contrast to other studies (Hounard et al., 2004, Mayer-Davis et al., 1998). However, studies in individuals with type 2 diabetes have shown that ambulatory activity is generally accumulated at a low intensity and that exercise intensity, but not exercise volume, predicts glycemic control (Boule et al., 2003, Johnson et al., 2005). Therefore, given that almost half the participants in this study had prediabetes or diabetes, it is possible that participants accumulated their walked activity at an intensity lower than that needed to improve insulin sensitivity.

The main limitations of this study are that it was not possible to determine causality and the high percentage of missing physical activity data. The small sample size precluded meaningful sub group analysis (Boule et al., 2003). Johnson et al., 2005). Therefore, given that almost half the participants in this study had prediabetes or diabetes, it is possible that participants accumulated their walked activity at an intensity lower than that needed to improve insulin sensitivity.

In the main limitations of this study are that it was not possible to determine causality and the high percentage of missing physical activity data. The small sample size precluded meaningful sub group analysis (Boule et al., 2003). Johnson et al., 2005). Therefore, given that almost half the participants in this study had prediabetes or diabetes, it is possible that participants accumulated their walked activity at an intensity lower than that needed to improve insulin sensitivity.

Conclusions

This study suggests that greater walking is associated with lower inflammatory markers independent of a range of confounders and of other forms of physical activity.
Rationale, design and baseline data from the Pre-diabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme study: A randomized controlled trial

Thomas Yates a,*, Melanie Davies b, Trish Gorely c, Fiona Bull d, Kamlesh Khunti c

a School of Sports and Exercise Sciences, Loughborough University, UK
b Department of Cardiovascular Sciences, University of Leicester, UK
c Department of Health Sciences, University of Leicester, UK

ABSTRACT

Objective: The PREPARE programme study is a randomized controlled trial which aims to determine whether structured education can be used to increase physical activity and improve glucose tolerance in individuals with impaired glucose tolerance (IGT). This paper outlines the rationale, design and baseline data from the PREPARE programme study.

Methods: Individuals with IGT were recruited from ongoing diabetes screening programmes. Outcomes included an oral glucose tolerance test, physical activity (pedometer pedometer) and psychological determinants.

Results: 103 individuals (male n = 65; female n = 38) were recruited. 28% of whom were from a South Asian ethnic background. At baseline the participants’ mean age and BMI were 64 ± 9 years and 29.4 ± 4.5 kg/m² respectively. Steps per day were associated with 2-h glucose (p = 0.022, p = 0.031) fasting glucose (p = 0.022, p = 0.04), HDL-cholesterol (p = 0.23, p = 0.02), triglycerides (p = 0.22, p = 0.03) and body fat percentage (p = 0.026, p = 0.01). Mean self-efficacy scores were significantly (p<0.01) higher for walking than for any other form of exercise. Participants reported high levels of concern about their IGT status but were confident that exercise would help treat control IGT.

Conclusion: This study demonstrates the importance of developing effective physical activity and self-management programmes for individuals with IGT.

Practice implications: This study provides a detailed framework for the promotion of physical activity in a population identified with an increased risk of developing type 2 diabetes which, if successful, could feasibly be implemented in a primary care or community setting.

© 2008 Elsevier Ireland Ltd. All rights reserved.

1. Introduction

1.1. Background

The prevalence of type 2 diabetes is reaching epidemic proportions and the costs associated with its treatment are set to represent a serious clinical and financial challenge to national health systems [1]. It is therefore of primary importance to develop diabetes prevention strategies in high risk populations to counter this worrying trend. Individuals with impaired glucose tolerance (IGT) have an increased risk of developing type 2 diabetes and cardiovascular disease compared to those with normal glucose tolerance [2] and are therefore a suitable population for diabetes prevention initiatives.

Although physical activity has consistently been associated with a reduced risk of developing diabetes [3], there is no evidence that traditional multi-factor diabetes prevention programmes have been successful at initiating clinically significant increases in physical activity [4]. More broadly, it has also been reported that interventions aimed at promoting physical activity make use of methods that would be difficult to deliver in usual health care practice [5], and that there is a gap between physical activity intervention research and the delivery of evidence-based practice [6]. Furthermore, physical activity interventions that have been delivered in primary care have met with limited success, particularly over the longer term [7,8]. Therefore there is a need...
to develop successful physical activity interventions that are appropriate for a primary health care or community setting. This conclusion is also true of diabetes prevention programmes in general where it has been pointed out that such initiatives will have limited feasibility and success unless they are tailored to the specific requirements of national health care services [9,10]. Whilst traditional diabetes prevention programmes are based on multi-factor lifestyle interventions [11,12], it has been shown that single-factor physical activity interventions are more effective at initiating physical activity behaviour change in a health care setting [13] and improving glycaemic control in individuals with type 2 diabetes [14]. Therefore, given the limited success of diabetes prevention programmes at promoting physical activity [4], robustly tested single-factor physical activity interventions are needed [15].

The Pre-diabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme study is a randomized controlled trial designed to test the efficacy of structured education at promoting physical activity and improving glucose tolerance in individuals identified with IGT. The aim of this paper is to describe the rationale, design and baseline data from the PREPARE programme study and to describe the relationships between measured psychological, behavioural and clinical variables. This detailed examination of the study's theoretical underpinning, recruited participants and correlations between measured variables will help contextualize future outcomes.

1.2. Rationale for the PREPARE programme

Patient education has been advocated as a fundamental part of patient care for individuals diagnosed with diabetes in the UK [16]. Whilst established structured education programmes for individuals with diabetes, such as the DESMOND programme [17], have been successful at initiating behaviour change in individuals with diabetes [18,19], structured education has not been tested as a method of promoting health behaviour and self-management in individuals identified with an increased risk of developing diabetes. As structured education is compatible with the infrastructure of many national health services, it is important to test whether this approach to patient care can be utilized to promote physical activity and improve health outcomes in at-risk populations.

In order to be effective it is important that interventions aimed at promoting physical activity and self-management are based on known learning techniques and health behaviour theory [16,20]. However, considering that there are more than 20 health behaviour theories and that many of these theories lack empirical evidence, choosing an appropriate theory on which to ground an intervention is problematic [11]. However, successful physical activity and multi-factor intervention programmes in individuals with IGT and diabetes, regardless of their theoretical underpinning, have consistently utilized methods that are central to Bandura's social cognitive theory [22], such as targeting barriers, self-efficacy and self-regulatory skills [11,23-26]. In particular it is increasingly recognized by Bandura and others that self-regulation is likely to be fundamental to the success of any health promotion intervention [20,27,28]

Self-regulatory models, such as Colwiller's implementation intentions [29], have been shown to be successful at initiating and predicting physical activity behaviour change [30-32]. Therefore in order to maximize physical activity behaviour change it is important that physical activity interventions are successful at promoting self-regulatory and volitional skills as well as traditional motivational components, such as self-efficacy.

Along with traditional social cognitive constructs, perceptions and beliefs about identified illnesses or health conditions may also be important in interventions promoting health behaviours. Leventhal's common sense model postulates that individuals conceptualize any identified health threat in terms of the cause, consequences, identity, control, treatment and timeline associated with the threat and that these domains will influence subsequent coping behaviour [33]. Although illness perceptions have typically been overlooked in physical activity research, Leventhal's common sense model has been demonstrated across a wide range of patient groups [34] and recent findings have shown that illness perceptions and beliefs are closely linked to health behaviour change, including physical activity, in individuals with type 2 diabetes [19]. Although IGT differs from diabetes and other chronic diseases, in that it is not a recognized disease, individuals identified with IGT are nevertheless likely to form a set of perceptions and beliefs about IGT that may influence how they cope with the condition in the future. Therefore, any intervention aimed at increasing physical activity in individuals with IGT should target perceptions and beliefs around IGT.

1.3. Walking and the pedometer

Physical activity interventions need to promote forms of physical activity that are appropriate and acceptable to their target populations. Walking has consistently been shown to be the preferred choice of physical activity in a wide range of populations and patient groups [23,35-37], including those with IGT [38]. Walking is also associated with fewer barriers than other forms of physical activity in black and minority ethnic populations [39]. It is therefore important that walking activity is promoted in interventions aimed at increasing physical activity in individuals with IGT.

The pedometer is widely recognized as an inexpensive tool which can aid the promotion of walking activity through its use as an objective self-monitoring tool. Pedometer intervention studies have consistently been shown to be successful at initiating physical activity behaviour change [40]. However, despite these promising findings the National Institute of Health and Clinical Excellence (NICE) has concluded that, whilst pedometers may be a useful tool in the promotion of physical activity, the success of pedometer intervention studies remains equivocal in a health-care setting [41].

2. Methods

2.1. Research design

The PREPARE programme study is a three-armed randomized controlled trial. The primary purpose of the study is to test the hypothesis that structured education can be effectively utilized to promote physical activity and improve glycaemic control in individuals identified with IGT. A secondary aim of the study is to test the hypothesis that providing participants with a pedometer and step per day goals will increase the effectiveness of structured education at promoting physical activity. We will measure the effectiveness of the pedometer version of the PREPARE programme against control conditions to test our primary hypothesis.

The study was powered to detect a 1 mmol/L difference in post-challenge 2-hour glucose levels between the primary intervention and control group. Using a power of 80%, a significance level of 0.05, a standardized difference of 1 and allowing for a 50% drop-out rate, two groups of 34 individuals were required to test our primary hypothesis [42]. After including a third group of the same size to test our secondary hypothesis, a total of 102 participants was required. Given the relatively small sample size, participants were randomized using a
block design and stratified by age and sex in order to increase the likelihood of randomization producing equivalent groups. Randomization was conducted using opaque envelopes and a randomly generated number sequence (SFSS, Chicago, USA) by a member of our research team with no prior knowledge of recruited individuals, other than their age and sex. Participants will be followed up at 3 months, 6 months and 12 months.

2.2. Treatment regimens

Participants were randomized to receive either usual care, the PREPARE programme with pedometer use or the PREPARE programme without pedometer use. The PREPARE programme is a single session group educational programme designed to promote increased physical activity, primarily walking activity, by targeting perceptions and knowledge of impaired glucose tolerance, physical activity self efficacy, barriers, and self regulatory skills. The programme is group based and delivered to between 5 and 10 participants, is 3 hours long and uses a person-centred approach to patient education, based on Chaiken's dual process theory [43]. The PREPARE programme is divided into four modules. Table 1 gives a broad overview of the theoretical underpinning and weighting of each module. A brief dietary session was included as pilot work had revealed that diet is strongly linked to illness perceptions surrounding IGT. However, participants were not encouraged to set dietary goals or action plans.

The two versions of the PREPARE programme are identical, except that in the pedometer version participants are given, and shown how to use, a pedometer (SW-200, Yamax Corporation, Tokyo, Japan) and encouraged to set personalized steps per day goals based on their baseline ambulatory activity levels and step per day categories proposed by Tudor-Locke and Bassett [44]; whereas in the alternative version participants are encouraged to set physical activity goals based on generic exercise recommendations, such as 30 min of moderate intensity exercise on most days of the week [45]. Participants in both groups are provided with physical activity diaries. A comprehensive written curriculum was developed for each version of the PREPARE programme. Each PREPARE programme session was delivered by two educators.

Educators held an undergraduate degree in a relevant discipline (dietician, sports scientist) and were trained to deliver the DESMOND curriculum [17], which is an established structured educational programme with a similar philosophy and theoretical underpinning to the PREPARE programme. In addition, all educators completed at least two pilot sessions of the PREPARE programme and received instructive feedback from an experienced and accredited DESMOND educator before delivering the PREPARE programme in the randomized controlled trial.

Individuals randomized to the two intervention groups also receive brief (10 min) one-to-one follow up counselling with a trained educator at their 3 month and 6 month clinical measurement session. There is no additional contact with the research team.

Participants randomized to the control group were sent a brief information sheet detailing the likely causes, consequences, symptoms and timeline associated with IGT, along with information about how physical activity can be used to treat/control the condition. No additional advice or encouragement is given to the control group.

2.3. Recruitment

Participants were recruited from ongoing population-based diabetes screening programmes between September 2006 and March 2007. Individuals were invited to take part in the study if, at initial screening, they had IGT (2 h glucose of $>7.8 \text{ mmol/l}$ and $<11.1 \text{ mmol/l}$ and fasting glucose $<7.0 \text{ mmol/l}$) [46] and had a body mass index (BMI) of 25 kg/m$^2$ or greater (23 kg/m$^2$ or greater for those from a South Asian ethnic background) [47]. Individuals who reported taking steroids or who were unable to take part in moderate physical activity were excluded.

2.4. Measures

The PREPARE study will be evaluated using biochemical variables, anthropometric and demographic variables, physical activity measures, as well as psychological variables.

2.4.1. Biochemical

Participants arrived at their appointment for an oral glucose tolerance test after a 12-h fast and 24 h of avoiding vigorous-

---

Please cite this article in press as: Yates T, et al. Rationale, design and baseline data from the Pre-diabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme study: A randomized controlled trial, Patient Educ Couns (2008), doi:10.1016/j.pec.2008.06.010
intensity exercise. Those who had a fasting or 2-h glucose level in the diabetes range [46] were called back for a second oral glucose tolerance test: if the participant had a fasting or 2-h glucose level in the diabetes range at the second test, a diagnosis of diabetes was confirmed and participants were referred to a specialist clinician for treatment.

Plasma glucose was measured using a glucose oxidase method on the Beckman Auto Analyzer (Beckman, High Wycombe, UK). Serum cholesterol was analyzed using the cholesterol enzymatic assay (Abbott Clinical Chemistry, IL, USA). High-density lipoprotein (HDL) cholesterol was analyzed using the ultra HDL assay (Abbott Clinical Chemistry, IL, USA). Low-density lipoprotein (LDL) was calculated using the Friedewald formula [48]. Serum triglyceride was analyzed using the triglyceride glycerol phosphate oxidase assay (Abbott Clinical Chemistry, IL, USA).

2.4.2. Anthropometric and demographic

Arterial blood pressure was measured in the sitting position (Omron, Healthcare, Henfield, UK); three measurements were obtained and the average of the last two measurements was used. Body weight and body fat percentage (Tanita TBF 611, Tanita, West Drayton, UK), waist circumference (midpoint between the lower costal margin and iliac crest) and height were also measured. Information about current medication and smoking status along with ethnicity were also measured by questionnaire.

2.4.3. Physical activity

Physical activity was measured objectively using a pedometer and subjectively with a questionnaire. Sealed piezoelectric pedometers with a 7-day memory (NL 800, New lifestyles, USA) were used for this study. These pedometers have been shown to be one of the most accurate and reliable instruments on the market and are more accurate than traditional spring-loaded pedometers for use on overweight and obese individuals [49]. At baseline, all participants were fitted with a pedometer and instructed to wear it for 7 consecutive days during waking hours and to keep a daily log of the time the instrument was worn. At the end of the 7-day period participants returned the pedometers by post to the research centre where the data was extracted from the instrument and matched to the time the pedometer was worn. For the purposes of this study at least 3 valid days of data were required; a valid day constituted at least 12 000 steps per day. It has been shown that the average steps per day of any weekly 3-day combination is highly correlated with the average steps per day taken over the full 7-day period; consequently, 3 or more days of data provides an acceptable measure of walking activity levels over 7 consecutive days [50]. For the purposes of this study, individuals were classified as sedentary (<5000 steps per day) or active (>5000 steps per day) based on preliminary pedometer indices proposed by Tudor-Locke and Bassett [44]. The long last 7 days self-administered format of the International Physical Activity Questionnaire (IPAQ) was also used to measure physical activity [51]. This questionnaire provides a comprehensive measure of walking and other moderate-to-vigorous intensity activities carried out at work, in the home, as transport and during leisure time. The IPAQ questionnaire has been shown to correlate adequately (p = 0.4) with accelerometer data in the United Kingdom [52]. Participants were classified as sedentary or active based on IPAQ guidelines [51]; these categories correspond to distinguishing those who achieve the current exercise recommendations [45] and those who do not.

2.4.4. Psychological determinants

2.4.4.1. Health-related quality of life

Health-related quality of life was measured using the EQ-5D [53], which is a standardized questionnaire that was developed for use as a measure of health outcomes and defines health in terms of five dimensions: mobility; self-care; usual activities; pain or discomfort; and anxiety or depression. Data from the EQ-5D can be reported either as a health profile (EQ-5D health status) or a health index (EQ-5D index) based on time trade-off data from England, UK, which was used to elicited utility weights for the EQ-5D.

2.4.4.2. Perceptions and perceived knowledge of ICT

Perceptions and perceived knowledge of ICT were measured with the validated brief illness perceptions questionnaire [54]. This instrument uses a 11-point Likert scale to measure five cognitive illness representations (consequences, timeline, personal control, treatment control, and identity), two emotional representations (concern and emotion) and illness comprehensibility.

2.4.4.3. Walking and exercise self-efficacy

Self-efficacy was measured using the 100% confidence rating scale (from 0 = no confidence to 100% = complete confidence) [55]. This self-efficacy questionnaire measures participants' confidence in their ability to walk for 10 min time periods increasing from 10 min to 1 h each day. The same scale was also used to measure participants' confidence in their ability to undertake any other form of exercise. An overall score for walking and exercise self-efficacy was calculated by summing the efficacy scores for each time period divided by the number of time periods. Exercise self-efficacy measures using the 100% confidence rating scale have been shown to have good (a > 0.8) internal reliability [56-59].

2.4.4.4. Exercise self-regulatory efficacy

Participants' confidence in their ability to self regulate their exercise behaviour in the face of five commonly identified barriers (tired, bad mood, bad weather, lack of time and holiday) was measured [60]. This questionnaire used the 100% confidence rating scale; an overall score for self-regulatory efficacy was calculated by summing the efficacy scores for each barrier divided by the number of barriers.

2.5. Data analysis

Differences between groups at baseline were analysed using analysis of variance procedures, nonparametric Kruskal-Wallis, and chi-square tests for, respectively, normally distributed continuous data, nonparametric continuous data and categorical data. Associations between variables measured at baseline were analysed using Spearman correlation coefficients.

3. Results

In total, 326 individuals were invited to take part in the study of whom 103 individuals (32%) consented to take part. The most common reason given for not wanting to take part in this study was a perceived lack of time or physical disability. Those who took part in the study were of a similar age and ethnicity compared to those who declined the invitation; however, relatively more men than women agreed to take part in the study (63% of men participants were male compared to 55% of those who were invited to take part; p = 0.03).

Table 2 presents the clinical and demographic baseline characteristics of the study participants. The randomization procedure produced equivalent groups. The age of the participants was 64 ± 9 years, just under two thirds were male and almost a third were from a South Asian Ethnic background. Ten percent of the participants were current smokers and over half of the participants were taking medication for high blood pressure or cholesterol levels.
were significantly correlated with 2h glucose and walking.

Steps per day ranged from -0.003 to 1. and the median value was 1.

EQ 5D score followed: mobility 26%, self care 9%, usual activities 18%, pain/discomfort 41%, and anxiety/depression 17%. EQ 5D pain scores in each of the five health domains in the EQ SD were as follows: mobility 26%, self care 9%, usual activities 18%, pain/discomfort 41%, and anxiety/depression 17%. EQ 5D pain scores ranged from -0.003 to 1 and the median value was 1.

Table 4 shows the correlations between steps per day, 2h glucose and psychological determinants. Steps per day were significantly correlated with 2h glucose and walking.

Exercise and self-regulatory efficacy. There was also a significant correlation between efficacy beliefs and some illness perceptions. In addition, steps per day were significantly correlated with fasting glucose (r = 0.22, p = 0.04), HDL cholesterol (r = 0.23, p = 0.03), triglycerides (r = 0.22, p = 0.01), body fat percentage (r = 0.26, p = 0.01) and waist circumference (r = 0.25, p = 0.02). There was also a significant correlation between steps per day and four of the EQ-5D items; mobility (r = 0.40, p < 0.01), self-care (r = 0.42, p < 0.01), usual activities (r = 0.38, p < 0.01) and pain/discomfort (r = 0.24, p = 0.02).

There was no significant correlation between steps per day and illness perceptions.

4. Discussion and conclusion

This randomized trial is designed to test whether structured education can be used to increase physical activity in individuals identified with a high risk of developing diabetes. Whilst structured education has been widely used for the treatment of diabetes, this approach has not been utilized to target a single health behaviour in at-risk individuals. In terms of promoting physical activity structured education could provide a feasible alternative to other recently developed theory driven programmes, such as the ProActive trial [62] and the Groningen Active Living Model [63].
Another important aspect of this study is that it will investigate whether providing participants with a pedometer, personalized steps per day goals and a steps per day log will promote physical activity behaviour change to a greater extent than simply providing participants with general time-based goals and a physical activity diary. This study will therefore address many of the limitations identified by NICE in other pedometer intervention studies [41].

At baseline participants took an average of 6346 ± 3444 steps per day. This is around 40% lower than the average steps per day reported previously in normal weight, overweight and obese individuals in the United Kingdom [64], however this level of ambulatory activity is similar to that reported in other industrialised countries [65-67], including in individuals diagnosed with type 2 diabetes [68,69].

We also found that self-efficacy scores were significantly (p < 0.001) higher for walking than for any other form of exercise. Given that self-efficacy levels have been shown to be a mediator of physical activity behaviour change [70], promoting walking activity, which is the primary aim of the PREPARE programme, is highly appropriate for this study population.

Along with the key determinants of social cognitive theory, such as self-efficacy, we hypothesized that illness perceptions are important mediators of physical activity behaviour change in individuals identified with IGT. At baseline, this study did not find a link between illness perceptions and physical activity levels. However, as several illness perceptions were associated with walking, exercise and self-regulatory efficacy beliefs, illness perception may form important preconditions to physical behaviour change in individuals with IGT.

This paper has revealed some limitations with the PREPARE programme study making it likely that future results will have limited generalizability. However, despite these limitations, this study will provide important new evidence on whether structured education can be used to promote physical activity in a multiethnic population identified with IGT in a health care setting. Future analysis will examine the effectiveness of the PREPARE programme at increasing physical activity and improving glucose tolerance with follow-up in the short-term (3 months and 6 months) and longer term (12 months). Analysis will also investigate whether any of the key determinants on which the PREPARE programme was grounded are mediators of behaviour change.

### Table 3

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>Total</th>
<th>Control</th>
<th>PREPARE minus pedometer</th>
<th>PREPARE plus pedometer</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Illness perceptions for IGT</td>
<td>92</td>
<td>0.0 [2.0]</td>
<td>29</td>
<td>0.0 [2.0]</td>
<td>29</td>
<td>0.2 [2.5]</td>
</tr>
<tr>
<td>Consequences (0 = no consequences, 10 = severe consequences)</td>
<td>90</td>
<td>3.0 [4.3]</td>
<td>28</td>
<td>3.0 [4.0]</td>
<td>29</td>
<td>3.0 [4.5]</td>
</tr>
<tr>
<td>Timeline (0 = a very short time, 10 = forever)</td>
<td>90</td>
<td>5.0 [5.0]</td>
<td>28</td>
<td>6.5 [4.8]</td>
<td>29</td>
<td>5.0 [4.5]</td>
</tr>
<tr>
<td>Control (0 = no control, 10 = complete control)</td>
<td>93</td>
<td>8.0 [5.0]</td>
<td>31</td>
<td>8.0 [5.0]</td>
<td>30</td>
<td>8.0 [5.0]</td>
</tr>
<tr>
<td>Treatment (physical activity) control (0 = not at all effective, 10 = extremely effective)</td>
<td>90</td>
<td>0.0 [3.0]</td>
<td>31</td>
<td>0.0 [2.0]</td>
<td>28</td>
<td>0.0 [3.0]</td>
</tr>
<tr>
<td>Symptoms/identity (0 = no symptoms, 10 = many symptoms)</td>
<td>92</td>
<td>6.5 [5.0]</td>
<td>31</td>
<td>5.0 [4.3]</td>
<td>29</td>
<td>6.0 [7.5]</td>
</tr>
<tr>
<td>Concern (0 = not at all concerned, 10 = extremely concerned)</td>
<td>95</td>
<td>5.0 [6.0]</td>
<td>32</td>
<td>4.0 [6.0]</td>
<td>30</td>
<td>5.0 [6.0]</td>
</tr>
<tr>
<td>Comprehension (perceived understanding) (0 = no understanding, 10 = complete understanding)</td>
<td>91</td>
<td>2.0 [5.0]</td>
<td>28</td>
<td>2.0 [3.0]</td>
<td>30</td>
<td>1.5 [6.0]</td>
</tr>
<tr>
<td>Emotional affect (0 = no emotional affect, 10 = extreme emotional affect)</td>
<td>97</td>
<td>90 [48]</td>
<td>32</td>
<td>64 [97]</td>
<td>30</td>
<td>90 [30]</td>
</tr>
<tr>
<td>Walking self-efficacy (0 = no confidence, 10 = complete confidence)</td>
<td>93</td>
<td>50 [67]</td>
<td>31</td>
<td>34 [85]</td>
<td>30</td>
<td>56 [74]</td>
</tr>
<tr>
<td>Exercise self-efficacy (0 = no confidence, 10 = complete confidence)</td>
<td>93</td>
<td>45 ± 28</td>
<td>32</td>
<td>40 ± 28</td>
<td>28</td>
<td>44 ± 28</td>
</tr>
<tr>
<td>Barrier self-efficacy (0 = no confidence, 10 = complete confidence)</td>
<td>92</td>
<td>8.0 [5.0]</td>
<td>31</td>
<td>8.0 [5.0]</td>
<td>30</td>
<td>8.0 [5.0]</td>
</tr>
</tbody>
</table>

Data presented as number (percent) or mean ± SD or median [interquartile range]. The number of complete data sets for each variable is also shown.

### Table 4

<table>
<thead>
<tr>
<th>Variables</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steps per day</td>
<td>-0.22*</td>
<td>-0.00</td>
<td>0.04</td>
<td>0.20</td>
<td>0.12</td>
<td>-0.11</td>
<td>0.02</td>
<td>0.19</td>
<td>-0.02</td>
<td>0.41**</td>
<td>0.28**</td>
<td>0.22**</td>
<td></td>
</tr>
<tr>
<td>2-h glucose</td>
<td>-0.16</td>
<td>-0.05</td>
<td>-0.14</td>
<td>-0.09</td>
<td>0.12</td>
<td>0.12</td>
<td>-0.09</td>
<td>0.16</td>
<td>-0.26*</td>
<td>-0.05</td>
<td>0.01</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Perceived affect</td>
<td>0.45**</td>
<td>0.04</td>
<td>-0.22*</td>
<td>0.46**</td>
<td>0.34**</td>
<td>-0.15</td>
<td>0.45**</td>
<td>-0.23*</td>
<td>0.24*</td>
<td>-0.12</td>
<td>0.12</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Perceived timetable</td>
<td>0.18</td>
<td>-0.15</td>
<td>0.18</td>
<td>0.32**</td>
<td>0.09</td>
<td>0.21</td>
<td>-0.13</td>
<td>0.02</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Perceived confidence</td>
<td>0.40**</td>
<td>0.03</td>
<td>0.04</td>
<td>0.17</td>
<td>-0.05</td>
<td>0.05</td>
<td>0.01</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
<td>0.12</td>
<td></td>
</tr>
<tr>
<td>Perceived effectiveness of exercise at treatment</td>
<td>-0.08</td>
<td>0.17</td>
<td>0.31**</td>
<td>-0.28**</td>
<td>0.30**</td>
<td>0.38**</td>
<td>0.34**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived symptom load</td>
<td>0.22*</td>
<td>-0.05</td>
<td>0.42**</td>
<td>-0.15</td>
<td>0.16</td>
<td>-0.16</td>
<td>0.16</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concern</td>
<td>0.14</td>
<td>0.30**</td>
<td>0.02</td>
<td>0.04</td>
<td>0.18</td>
<td>0.18</td>
<td>0.18</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Perceived knowledge</td>
<td>-0.12</td>
<td>0.20</td>
<td>-0.30*</td>
<td>-0.12</td>
<td>0.16</td>
<td>0.16</td>
<td>0.16</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Emotional affect</td>
<td>-0.30*</td>
<td>-0.12</td>
<td>0.16</td>
<td>0.30**</td>
<td>0.12</td>
<td>0.30**</td>
<td>0.12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walking self-efficacy</td>
<td>0.70**</td>
<td>0.65**</td>
<td>0.72**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise self-efficacy</td>
<td>0.72**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barrier self-efficacy</td>
<td>0.72**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* p < 0.05, ** p < 0.01.

Please cite this article in press as: Yates T, et al. Rationale, design and baseline data from the Pre-diabetes Risk Education and Physical Activity Recommendation and Encouragement (PREPARE) programme study: A randomized controlled trial. Patient Educ Couns (2008), doi:10.1016/j.pec.2008.06.010
4.2. Conclusion

This study emphasises the need to develop successful free-living physical activity and self-management programmes for individuals with IGT that are appropriate for implementation in a primary health care or community setting and suggests that structured education, aimed targeting perceptions and knowledge of IGT and promoting increased walking activity, may be one such approach.

4.3. Practice Implications

The PREPARE programme study will provide evidence for the efficacy of structured education at promoting physical activity and improving health outcomes in individuals identified with IGT. This study could have important implications for diabetes prevention initiatives carried out in a primary health care or community setting. Baseline data reported here indicates that walking is the most appropriate form of activity to promote in individuals with IGT. The fact that objectively measured walking activity was associated with glucose control, lipid profile, and markers of adiposity further emphasises the importance of promoting walking activity in this at risk population. This study also shows that individuals become concerned after being informed they have IGT, therefore it is important to provide this patient group with accessible and accurate information about IGT.

Acknowledgement

This study was funded by a grant from Diabetes UK.

Conflict of interest

None.

References


[51] International Physical Activity Questionnaire. Available at: http://www.ipaq.ki.se/!


Appendix Two

Characteristics of studies included in the systematic review
<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Study location/name</th>
<th>Study design</th>
<th>Intervention duration</th>
<th>Number of subjects (Male/Female)</th>
<th>Inclusion criteria</th>
<th>Type of dietary intervention</th>
<th>Type of exercise intervention</th>
<th>Method of physical activity measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindström et al., 2003</td>
<td>Finland/ Finish Diabetes Prevention Study</td>
<td>RCT</td>
<td>3 years</td>
<td>522 (172/350)</td>
<td>IGT (WHO criteria, 1985), age 40 to 64 yrs old, BMI ≥25</td>
<td>Exercise and diet reduction through a healthy diet.</td>
<td>Participants individually encouraged to increase their overall level of physical activity. Circuit-type exercise sessions were also offered</td>
<td>Self-report – Kuopio 12 month leisure time physical activity questionnaire</td>
</tr>
<tr>
<td>Knowler et al., 2002</td>
<td>USA/Diabetes Prevention Research Group</td>
<td>RCT</td>
<td>Average follow-up 2.8 (range, 1.8 – 4.6) yrs</td>
<td>2161 (680/1481)*</td>
<td>IGT (ADA criteria, 1997), Age ≥25 yr, BMI ≥ 24 Kg/m² ≥ 22 Kg/m² if Asian, fasting plasma glucose ≥ 5.3 mmol/l</td>
<td>Exercise and diet reduction through a healthy, low-energy, low-fat diet.</td>
<td>Participants individually encouraged to accumulate at least 150 minutes of moderate intensity exercise per week</td>
<td>Self-report – Modified Activity Questionnaire and activity log</td>
</tr>
<tr>
<td>Authors, Year</td>
<td>Study location/name</td>
<td>Study design</td>
<td>Intervention duration</td>
<td>Number of subjects (Male/Female)</td>
<td>Inclusion criteria</td>
<td>Type of intervention</td>
<td>Type of dietary intervention</td>
<td>Type of exercise intervention</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------------</td>
<td>--------------</td>
<td>-----------------------</td>
<td>----------------------------------</td>
<td>-------------------</td>
<td>----------------------</td>
<td>-----------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Pan et al., 1997</td>
<td>China/ The Qing IGT and Diabetes Study</td>
<td>RCT</td>
<td>6 years</td>
<td>530 (283/247)</td>
<td>IGT (WHO criteria, 1985), Age ≥25</td>
<td>1) Exercise and diet</td>
<td>Weight maintenance for those with a BMI &lt; 25 Kg/m²</td>
<td>Participants were encouraged to increase their physical activity to 1 unit per day². Those who were &lt; 50 yr old and who were able to accumulate 2 units per day.</td>
</tr>
<tr>
<td>Authors, Year</td>
<td>Study location/name</td>
<td>Study design</td>
<td>Intervention duration</td>
<td>Number of subjects (Male/Female)</td>
<td>Inclusion criteria</td>
<td>Type of intervention</td>
<td>Type of dietary intervention</td>
<td>Type of exercise intervention</td>
</tr>
<tr>
<td>--------------</td>
<td>---------------------</td>
<td>--------------</td>
<td>-----------------------</td>
<td>----------------------------------</td>
<td>-------------------</td>
<td>----------------------</td>
<td>-----------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Eriksson and Lindgarde, 1991</td>
<td>Sweden/ The 6-year Malmo feasibility study</td>
<td>Non-randomised controlled trial</td>
<td>5 years</td>
<td>260 (260/0)</td>
<td>IGT (2-h post challenge glucose values of between 7 and 11 mmol/l and fasting plasma glucose of &lt; 7.8 mmol/l)</td>
<td>Exercise and diet</td>
<td>Healthy dietary advice given</td>
<td>Participants were encouraged to increase their physical activity levels. Participants given the option of training in organised groups for a 6-month period in the first year.</td>
</tr>
<tr>
<td>Oldroyd et al., 2006</td>
<td>England</td>
<td>RCT</td>
<td>24 months</td>
<td>69 (39/30)</td>
<td>IGT (WHO criteria, 1985)</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet.</td>
<td>Participants were encouraged to undertake 20-30min of aerobic activity 2-3 d/wk. In addition, all participants were given a discount at local gyms.</td>
</tr>
<tr>
<td>Authors, Year</td>
<td>Study location/name</td>
<td>Study design</td>
<td>Intervention duration</td>
<td>Number of subjects (Male/Female)</td>
<td>Inclusion criteria</td>
<td>Type of intervention</td>
<td>Type of dietary intervention</td>
<td>Type of exercise intervention</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------------</td>
<td>--------------</td>
<td>-----------------------</td>
<td>----------------------------------</td>
<td>-------------------</td>
<td>---------------------</td>
<td>--------------------------</td>
<td>-------------------------------</td>
</tr>
<tr>
<td>Mensink et al., 2003</td>
<td>Netherlands/ Maastricht</td>
<td>RCT</td>
<td>24 months</td>
<td>114 (64/50)</td>
<td>IGT (2-h post challenge glucose values of between 7.8 and 12.5 mmol/l and fasting plasma glucose of &lt; 7.8 mmol/l), age &gt;40 yrs old, BMI ≥ 25.0</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet.</td>
<td>Participants were encouraged through goal setting to undertake 30 min of moderate intensity exercise per day and were given use of free exercise classes.</td>
</tr>
<tr>
<td>Authors, Study Year</td>
<td>Study location/name</td>
<td>Study design</td>
<td>Intervention duration</td>
<td>Number of subjects (Male/Female)</td>
<td>Inclusion criteria</td>
<td>Type of intervention</td>
<td>Type of dietary intervention</td>
<td>Type of exercise intervention</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------</td>
<td>--------------</td>
<td>-----------------------</td>
<td>---------------------------------</td>
<td>-------------------</td>
<td>---------------------</td>
<td>-----------------------------</td>
<td>---------------------------------</td>
</tr>
<tr>
<td>Lindahl et al., 1999</td>
<td>Sweden</td>
<td>RCT</td>
<td>12 months</td>
<td>186 (69/117)</td>
<td>IGT (WHO criteria, 1985), age 30-60 yrs, BMI ≥ 27 kg/m²</td>
<td>Exercise and diet</td>
<td>Weight reduction through a healthy, low-energy, low-fat diet.</td>
<td>Participants were encouraged to increase their physical activity. Supervised exercise sessions were available in the first month</td>
</tr>
<tr>
<td>Carr et al., 2005</td>
<td>USA</td>
<td>RCT</td>
<td>24 months</td>
<td>62 (29/33)</td>
<td>IGT (WHO criteria, 1998)</td>
<td>Structured exercise and diet</td>
<td>Participants were encouraged to follow the isocalorific American Heart Foundation Step 2 diet</td>
<td>Walking/jogging at &gt; 70% of heart rate reserve for 1 hr 3d/wk</td>
</tr>
</tbody>
</table>

* = Data for lifestyle and control groups only, \(^*\) = 1 unit = 30 min mild exercise, or 20 min moderate exercise, or 10 min strenuous exercise, or 5 min very strenuous exercise
Appendix Three

Baseline values and change in main outcomes for the studies included in the systematic review
<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Baseline VO2max (l/min)</th>
<th>Change in VO2max (l/min)^b</th>
<th>Baseline self-reported physical activity levels</th>
<th>Change in self-reported leisure time physical activity^a</th>
<th>Baseline body mass (Kg)</th>
<th>Change in body mass (Kg)^a</th>
<th>Baseline 2-h PC (mmol/l)</th>
<th>Change in 2-h PC (mmol/l)^a</th>
<th>Baseline fasting glucose (mmol/l)</th>
<th>Change in fasting glucose (mmol/l)^a</th>
<th>Baseline fasting plasma glucose (mmol/l)</th>
<th>Change in fasting plasma glucose (mmol/l)^a</th>
<th>Relative risk of diabetes (intervention group vs control group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lindstöm et al., 2003</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>Results at 3 years</td>
<td>8.9±0.1</td>
<td>Lifestyle</td>
<td>Change at 1 year</td>
<td>6.1±0.05</td>
<td>Lifestyle</td>
<td>Change at 1 year</td>
<td>0.4 (0.3, 0.7)</td>
</tr>
<tr>
<td></td>
<td>156 (62 to 288) min/wk</td>
<td>61 (-33 - Control</td>
<td>Lifestyle: Control</td>
<td>Control: Lifestyle</td>
<td>85.5±0.9</td>
<td>-3.5±0.3^b</td>
<td>8.9±0.1</td>
<td>Control</td>
<td>-0.9±0.1^b</td>
<td>Control</td>
<td>6.2±0.04</td>
<td>Control</td>
<td>-0.2±0.04</td>
</tr>
<tr>
<td></td>
<td>169 (65 to 352) min/wk</td>
<td>6 (-91 - 104) min/wk</td>
<td>Control</td>
<td>Control: Lifestyle</td>
<td>0.9±0.4</td>
<td>Change at 3 yrs</td>
<td>Lifestyle</td>
<td>-0.5±0.2</td>
<td>Control</td>
<td>0.0±0.05</td>
<td>Control</td>
<td>-0.1±0.2</td>
<td>0.1±0.05</td>
</tr>
<tr>
<td>Authors, Year</td>
<td>Baseline VO2max (l/min)</td>
<td>Change in VO2max (l/min)</td>
<td>Baseline self-reported physical activity levels</td>
<td>Change in self-reported leisure time physical activity</td>
<td>Baseline body mass (Kg)</td>
<td>Change in body mass (Kg)</td>
<td>Baseline 2-h PC (mmol/l)</td>
<td>Change in 2-h PC from baseline (mmol/l)</td>
<td>Baseline fasting glucose (mmol/l)</td>
<td>Change in fasting plasma glucose (mmol/l)</td>
<td>Relative risk of diabetes (intervention group vs control group)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>--------------------------</td>
<td>--------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>------------------------</td>
<td>------------------------</td>
<td>-------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td>-----------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowler et al., 2002</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle</td>
<td>Lifestyle: 6</td>
<td>94.1±0.6</td>
<td>-5.6</td>
<td>9.1±0.03</td>
<td>NR</td>
<td>NR</td>
<td>5.9±0.01</td>
<td>0.4 (0.3, 0.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>15.5±0.7</td>
<td>MET-hr/wk</td>
<td>Control: 1</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>MET-hr/wk</td>
<td>Control:</td>
<td>MET-hr/wk</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>17.0±0.9</td>
<td>MET-hr/wk</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pan et al., 1997</td>
<td>N/A</td>
<td>N/A</td>
<td>Exercise</td>
<td>Exercise</td>
<td>8.8±0.1</td>
<td>1.7</td>
<td>5.6±0.1</td>
<td>Exercise</td>
<td>Exercise</td>
<td>0.5 (0.2, 0.9)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>3.4±0.2</td>
<td>0.6 units</td>
<td>Diet and:</td>
<td>-2.5</td>
<td>Diet and:</td>
<td>Diet and:</td>
<td>Diet and:</td>
<td>Diet and:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>units</td>
<td>Exercise</td>
<td>Exercise:</td>
<td>Exercise:</td>
<td>Exercise:</td>
<td>Diet and:</td>
<td>Diet and:</td>
<td>Diet and:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Exercise and diet</td>
<td>Diet and Exercise</td>
<td>Exercise</td>
<td>Exercise</td>
<td>Exercise:</td>
<td>Exercise</td>
<td>Exercise</td>
<td>Exercise</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.8 units</td>
<td>Control</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>units</td>
<td>Control</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td>Control:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>0.1 units</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Control</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2.4±0.2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

200
<table>
<thead>
<tr>
<th>Authors, Year</th>
<th>Baseline VO2max (l/min)</th>
<th>Change in VO2max (l/min)*</th>
<th>Baseline self-reported physical activity levels</th>
<th>Change in self-reported leisure time physical activity*</th>
<th>Baseline body mass (Kg)</th>
<th>Change in body mass (Kg)*</th>
<th>Baseline 2-h PC (mmol/l)</th>
<th>Change in 2-h PC from baseline (mmol/l)*</th>
<th>Baseline fasting glucose (mmol/l)</th>
<th>Change in fasting plasma glucose (mmol/l)*</th>
<th>Relative risk of diabetes (intervention group vs control group)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eriksson and Lindgarde, 1991</td>
<td>2.46±0.04</td>
<td>0.20*</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle: 82c</td>
<td>Lifestyle: -3.3b</td>
<td>Lifestyle: 8.2±01</td>
<td>Lifestyle: -1.1b</td>
<td>Control</td>
<td>Control</td>
<td>Control</td>
</tr>
<tr>
<td>Oldroyd et al., 2006</td>
<td>N/A</td>
<td>N/A</td>
<td>NR</td>
<td>NR</td>
<td>Lifestyle: 85.3±2.9</td>
<td>Lifestyle: -1.8±1.1b</td>
<td>Lifestyle: 9.2±0.1</td>
<td>Lifestyle: -0.6±0.3</td>
<td>Control</td>
<td>Control</td>
<td>Control</td>
</tr>
<tr>
<td>Authors, Year</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle</td>
<td>Lifestyle:</td>
<td>Lifestyle</td>
<td>Change at one year</td>
<td>Lifestyle</td>
<td>Change at 1 year</td>
<td>N/A</td>
</tr>
<tr>
<td>--------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-----</td>
<td>-----</td>
<td>-----------</td>
<td>-----------</td>
<td>-----------</td>
<td>-------------------</td>
<td>-----------</td>
<td>-----------------</td>
<td>-----</td>
</tr>
<tr>
<td><strong>Mensink et al., 2003</strong></td>
<td>2.15±0.1</td>
<td>0.09±1.90&lt;sup&gt;b&lt;/sup&gt;</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle</td>
<td>Lifestyle:</td>
<td>Lifestyle</td>
<td>Change at one year</td>
<td>Lifestyle</td>
<td>Change at 1 year</td>
<td>N/A</td>
</tr>
<tr>
<td>Control</td>
<td>Control</td>
<td>2.13±0.1</td>
<td>-0.03±2.77</td>
<td>83.7±1.5</td>
<td>-0.1±0.5</td>
<td>8.6 ±0.2</td>
<td>-0.9±0.3&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Control</td>
<td>0.3±0.3</td>
<td>Change at 2 years</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Control</td>
<td>0.1±0.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.2±0.1</td>
<td>Lifestyle</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.5±0.1</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.8±0.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Lindahl et al., 1999</strong></td>
<td>2.12± 0.1&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.21±0.1&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>N/A</td>
<td>N/A</td>
<td>Lifestyle</td>
<td>Lifestyle:</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>Lifestyle</td>
<td>N/A</td>
</tr>
<tr>
<td>Control</td>
<td>Control</td>
<td>1.89±0.1&lt;sup&gt;e&lt;/sup&gt;</td>
<td>0.02±0.1&lt;sup&gt;e&lt;/sup&gt;</td>
<td>86.4±1.1</td>
<td>-5.4±0.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.5</td>
<td>-0.7±0.2</td>
<td>5.4 Control</td>
<td>-0.5± 0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6.1</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-0.3±0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Year</td>
<td>VO2max (l/min)</td>
<td>Baseline self-reported physical activity levels</td>
<td>Change in leisure time activity</td>
<td>Baseline body mass (Kg)</td>
<td>Change in body mass (Kg)</td>
<td>Baseline 2-h PC glucose (mmol/l)</td>
<td>Change in 2-h PC glucose (mmol/l)</td>
<td>Baseline fasting plasma glucose (mmol/l)</td>
<td>Change in fasting plasma glucose (mmol/l)</td>
<td>Relative risk of diabetes (intervention group vs control group)</td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>----------------</td>
<td>-----------------------------------------------</td>
<td>---------------------------------</td>
<td>------------------------</td>
<td>-------------------------</td>
<td>---------------------------------</td>
<td>-----------------------------------</td>
<td>----------------------------------------</td>
<td>---------------------------------------------</td>
<td>------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Carr et al., 2005</td>
<td>Exercise 1.93±0.1</td>
<td>N/A</td>
<td>N/A</td>
<td>Baseline 66.5±2.9</td>
<td>Change at 6 months 9.2±0.2</td>
<td>Exercise 5.3±0.1</td>
<td>Change at 6 months 5.4±0.1</td>
<td>Control 0.7</td>
<td>Control 0.0</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Exercise 0.16±0.04</td>
<td>N/A</td>
<td>N/A</td>
<td>Baseline -1.8±0.5</td>
<td>Change at 6 months 9.1±0.2</td>
<td>Exercise -0.7</td>
<td>Change at 6 months 0.1</td>
<td>Control -0.1</td>
<td>Control 0.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2005</td>
<td>Control 2.02±0.1</td>
<td>N/A</td>
<td>N/A</td>
<td>Baseline 69.7±2.6</td>
<td>Change at 24 months 0.6</td>
<td>Exercise 0.0</td>
<td>Change at 24 months 0.1</td>
<td>Control 0.0</td>
<td>Control 0.1</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
* Figure estimated from graph Data for lifestyle and control groups only

d 1 unit = 30 min mild exercise, or 20 min moderate exercise, or 10 min strenuous exercise, or 5 min very strenuous exercise

* Fitness measurement taken in a randomly selected sub-group, n = 45
Appendix Four

The PREPARE programme curriculum
PREPARE
Pre-Diabetes Risk Education and Physical Activity
Recommendation and Encouragement

PROGRAMME CURRICULUM
Symbol Key

- **=[Educator says** (this symbol is usually used to identify a question the educator asks to elicit the desired information from the group. These are only suggestions; it is recommended each educator develops their own style)

- **=[Information collected on a flip chart

- **=[Information elicited or imparted verbally

- **=[Further ideas

- **= Prompt (this symbol is usually follows an open question and is accompanied with the phrase “Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer”. This means that when participants do not know the answer or give the wrong answer the aim should be to use further open questions to steer the group towards the desired response.)
On Arrival

When patients arrive, politely welcome them and check that they can stay for the whole session. If they can’t stay, give them the number to call to re-book a session.

Get each person to sign in so that you have an attendance record in case of fire.

Introduction

Start promptly. If you are expecting others who have not yet arrived, explain this to the group.

Introduce yourself and let the other facilitators introduce themselves.

Create a friendly and relaxed atmosphere.

Tell the group about necessary practical issues such as where the toilets are, the location of the fire escape etc.

Explain the format of the session i.e. when the breaks are etc.

In your own words explain the outline of the initial session.

Encourage participants to ask questions at any time throughout the session.
Patient story

Participant activity

- Participants will be encouraged to articulate what they believe the symptoms, causes, consequences, treatments and timeline associated with IGT are.
- Participants will be asked to identify the most important question they want answered by the end of the session
Ask each individual the following questions and write their answers down on individuals flip chart sheets

**Name**

- What is your name?
- Write answers down under heading “Names”

**Signs and symptoms**

- How did you find out you have IGT?
- Are there any symptoms you think you have that might be the result of having IGT?
- Write answers down under heading “Signs and symptoms”.

**Causes and concerns**

- What do you think caused your IGT?
- What have you heard about IGT?
- Write answers down under heading “Causes and concerns”.

**Consequences/treatments**

- What do you think the long term effects of having IGT are?
- What do you think can be done to treat IGT?
- How long do you think you will have IGT?
- Write answers down under heading “Consequences/treatments”.

**Burning issues**

- Do you have any burning issues you want answered by the end of the session?
- Write answers down under heading “Burning issues”
Healthy blood glucose control

Participant leaning opportunities

Participants will
- Understand that IGT is defined by high levels of glucose in the blood after eating a meal
- Know the definition of IGT
- Know what glucose is and how it is used by the body for energy
- Understand the role insulin plays in regulating blood glucose levels

Content covered

- Definition of IGT
- What glucose is and how it is used by the body
- How insulin helps glucose enter the cells
- How IGT effects the pancreas and leads to the development of type 2 diabetes over time
So, you are all here because you have been identified as having IGT within the last 12 months – can anyone tell me what IGT is?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

In IGT something goes wrong in the way the body functions after eating a meal, this makes the glucose levels in the blood too high for good health. That is why you all had to have your glucose levels tested after drinking a sugary drink. IGT is also called pre-diabetes.

Can anyone tell me how much glucose there is in the blood of someone with IGT?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The definition is a blood glucose level of between 7.8 and 11.1 mmol/l 2 hours after eating 75g of sugar, less than 7.8 mmol/l is considered normal and more than 11.1 mmol/l is considered a diagnoses of type 2 diabetes.

In order to understand what goes wrong in individuals who have pre-diabetes it is important to first understand how a healthy body regulates its glucose levels.

The term glucose has been used a lot, what exactly is glucose?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Glucose is a form of sugar that is found in the blood.

Why do you think we need glucose in our bodies in the first place?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Glucose acts as a fuel or energy for cells in our bodies. Cells are small components that make up all the parts of our body, such as our hearts, brains, and muscles.

Where does the glucose in our bodies come from?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Food, typically from sugary or starchy foods.

Build up the story by adding pictures and symbols to the flip chart as you go along. Start by drawing a large picture of the human body.

When you eat some food where does it go?
Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The stomach where, it is churned up and digested into tiny particles, including glucose particles.

What happens to the glucose then?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

It enters the blood stream through the liver.

On the diagram draw blood vessels with particles of glucose inside.

What happens once the glucose has entered the blood stream?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The blood stream acts as a transport system, it delivers glucose and other things to parts of the body that need them.

What parts of the body do you think are most in need of glucose?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Muscles are the main parts of the body that need glucose for energy.

Draw a muscle on the diagram.

How do you think the glucose gets into the cells that need it?

Use this analogy: Imagine this room is the cell and you are the glucose. How did you get in here? (People usually say through the door) Now the door was open when you came in here. Why don’t we usually leave the door open all the time? (People usually say people steal things etc). So to stop these things happening we keep the door locked most of the time. This is the same with our cells. The doors that allow the glucose to get into the cells are usually locked.

So what do we need to open the cell doors?

A key

What do you think the key might be?

Insulin
Whereabouts in the body is insulin produced?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Insulin is made by the pancreas.

What happens to the glucose after it has entered the cell?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

It is used as energy.

In healthy individuals this system of insulin being released from the pancreas to shuttle glucose into your body’s cells keeps a tight control on blood glucose levels.

Summarize the points of this section
IGT consequences and personal risk

Participant learning opportunities

Participants will
- Work out what goes wrong in the body in individuals with IGT
- Understand why individuals with IGT are likely to progress to type 2 diabetes over time and understand how quickly this happens
- Understand that the future risk of diabetes is controllable by the individual
- Know the risks associated with type 2 diabetes
- Know the risks associated with IGT
- Know the general risk factors of cardiovascular disease
- Know the difference between good (HDL) and bad (LDL) cholesterol
- Know the symptoms of IGT
- Know what causes IGT
- Understand their individual risk profile

Content covered

- What happens in IGT
- How IGT affects the pancreas and leads to the development of type 2 diabetes
- The risk of getting type 2 diabetes
- The controllable nature of IGT
- The risks associated with type 2 diabetes
- The risks that are associated with IGT
- Cardiovascular disease risk markers
- IGT symptoms
- IGT causes
- Risk profiles
Refer back to the diagram of the passage of glucose through the body.

Now obviously something is going wrong in individuals with IGT, which means that glucose levels are no longer being regulated optimally - using this diagram can you work out where things might be going wrong in somebody with IGT?

Acknowledge responses and capture correct answers on the diagram, try to elicit the following. If no answers are forthcoming move on and give the correct answer.

IGT could be the result of insulin resistance (something going wrong with the locks) and/or a reduction in insulin production due to damaged pancreas (not enough keys for the locks) as both these problems will lead to increased amounts of glucose in the blood.

In the vast majority of people with IGT the main problem initially is insulin resistance.

Use this analogy for insulin resistance: *Imagine that the locks on the cells in your body start to become rusty. What will happen to the insulin “keys” if this happens* (People usually say they will not work so well).

How do you think the pancreas which produces the insulin “keys” will have to respond if some of the keys it is producing are not working in the rusty locks?

Use this analogy: “think of the pancreas as a factory that is producing keys but because demand is high the nasty factory boss decides to try to up production not by employing more people and buying more machines but by working the existing machines and workers harder. Although this might be a good idea in the short term what will happen in the long term (People normally say it will get tired or break).

Because the pancreas is being forced to work extra hard it will start to get exhausted and damaged, which will result in insulin production falling. In time the pancreas will get so damaged that is beyond the point of repair, this is when type 2 diabetes sets in. Individuals with IGT are at a point were the cell locks are getting rusty and the pancreas is having to work harder and is getting tired, however most of you will not be at a point where the pancreas is damaged beyond a point of repair.

Does anyone know how high the risk of diabetes is in someone with IGT?

Write answers down: If no one has mentioned it write the correct answer down.

Each year up to 14% of you will develop diabetes, which is about ten times the rate in those with normal glucose tolerance. That means that within 12 months X number (insert the right number) of you will have diabetes and over the course of your lifetime around half will develop type 2 diabetes.

Going back to what is happening in the body in terms of the rusty locks and insulin keys, do you think you have control over whether you will eventually get diabetes or do you think that what you do makes no difference?
Collect answers and acknowledge responses, if no one has said it inform the group of the correct answer.

This is one of the key messages you should take home: the risk of diabetes is controllable by you. We will see, later, how you are able to control your insulin resistance (rusty locks), which will therefore reduce the risk of the pancreas getting damaged which in turn reduces the risk of type 2 diabetes.

Without wanting to alarm you too much, does anyone know the health risks that are associated with diabetes?

Write answers down. Try to elicit answers that include the following:

- Heart attacks
- Stroke
- Circulation problems - amputation
- Kidney problems
- Eye problems
- Loss of feeling
- Blood pressure
- Cholesterol
- Depression

How many of the problems associated with diabetes do you think people with IGT might already be at a higher risk of?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

People with IGT are at a higher risk of heart attacks and stroke than people with normal glucose tolerance.

Other than glucose levels, can anyone name anything else that increases the risk of heart attack or stroke?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Blood pressure, cholesterol levels, physical activity, diet and body weight.

Explain the difference between good (HDL) and bad (LDL) cholesterol.

Hand out each individual’s glucose, cholesterol and blood pressure values from their last clinical measurement session and go through the risk profile. Make sure everyone understands how to use their risk profiles.

Do you think there are specific symptoms associated with IGT?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.
There are generally no specific symptoms associated with IGT.

Refer back to the patient story and ask the participants if they can think of any other causes of IGT.

Write correct answers down and try to elicit the following. If no answers are forthcoming move on and give the correct answer:

- Genetics and family history
- Having central obesity
- Being inactive
- Getting older
- Ethnicity
- Depression and stress (although make it clear cause and effect have not been established)

Summarize the main points of this section.

BREAK FOR TEA AND COFFEE.
Healthy eating

Participant learning opportunities

Participants will

- Know where the energy in food goes
- Know the different macronutrients
- Know that fat, rather than sugar, is the main dietary variable linked to diabetes and heart disease
- Know that there are three types of fat
- Know the importance of saturated fat and why saturated fat may be harmful for people with pre-diabetes
- Know common sources of saturated and unsaturated fat

Content covered

- Human energy balance
- The different macronutrients and the relative importance of fat compared to sugar for individuals with prediabetes
- The different types of fat
- Saturated fat and its link to pre-diabetes, diabetes and heart disease
- Why saturated fat may be harmful for people with pre-diabetes
- Different sources of saturated and unsaturated fat
Let's start with the basics, why do we eat?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Food provides us with energy.

What is energy from food used for?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The energy in food is used to keep us alive by keeping our vital organs functioning and it is used to power any form of voluntary movement. If we eat more energy than our body needs for these tasks than the excess is stored as fat.

All energy from food comes from three major components, can anyone tell me what these components are?

Write answers down: Try to elicit the following:
- Fats
- Carbohydrates (sugars)
- Protein

Of these three components, which is the most important in terms of pre-diabetes and the prevention of diabetes?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Fats

If members of the group think sugar is the most important factor, explain clearly that there is no evidence for this, but there is strong evidence that fat intake is a contributing factor to the development of pre-diabetes, diabetes and heart disease.

There are also three kinds of fat, can anyone tell me what these are?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Saturated, monounsaturated, and polyunsaturated.

Can anyone name some foods that are high in saturated, monounsaturated, or polyunsaturated fat?

Write answers down under the headings:
- **Saturated fat** – most animal fats (butter, ghee, cream, whole milk, red meat), coconut milk and palm oil
- **Polyunsaturated fat** – vegetable oil, fish oil
- **Monounsaturated fat** – olive oil, rape seed oil

Are all these types of fats bad for your health?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

In moderation, polyunsaturated fats and particularly monounsaturated fats are actually healthy and vital for life. However, saturated fats, even in relatively small amounts, are considered unhealthy and have been linked to pre-diabetes, diabetes and heart disease.

So why is saturated fat bad for your health?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

High saturated fat intake is unhealthy for people with pre-diabetes because it increases insulin resistance (i.e. makes the cell locks more rusty) and it increases bad (LDL) cholesterol levels in the blood.

To reinforce the different fat sources, play the saturated fat game by laying out the packaging of various oils and fats and asking the participants to group the fat sources into saturated, polyunsaturated, or monounsaturated groups.

Summarize the main points of this section.
Physical activity module

**Participant learning opportunities**
Participants will:

- Understand the impact of physical activity on their glucose tolerance
- Understand the general health benefits of physical activity
- Know the current recommendation for physical activity, and the minimal level required for controlling IGT
- Be able to list practical ways of increasing their physical activity levels
- Understand why it is better to increase their physical activity levels in small incremental steps
- List barriers that are likely to prevent them from increasing their physical activity and understand how these barriers might be overcome
- Understand the importance of forming action plans and keeping a diary

**Content covered**

- How physical activity may improve glucose tolerance
- The effect of physical activity on other risk factors associated with IGT
- The current national exercise recommendations and how the minimum level needed to improve glucose tolerance is likely to exceed these recommendations
- Practical ways of increasing physical activity levels in home, town and work environments
- Reasons for why it is important to increase physical activity levels in small incremental steps
- Individual barriers to becoming more physically active
- Self-regulation skills
Going back to the model of the insulin keys and the cell locks, how do you think exercise might help individuals with IGT given that we know the main problem with IGT is insulin resistance? How do you think exercise will reduce your blood glucose?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Exercise lowers blood glucose by reducing insulin resistance, hence allowing more glucose into the muscle cells.

Use this analogy: Think of exercise as WD40 that lubricates the rusty locks, but the locks are constantly being exposed to rainy and windy weather. Therefore unless more WD40 is applied soon the locks will seize up again. (Then try and get the group to say that this means that exercise needs to be done regularly).

Does anyone know of other ways that exercise might improve your general health?

Write answers down: Try to elicit the following:

*Increased physical activity:*
- Increases HDL
- Helps keep your heart healthy
- Helps in weight management
- Helps reduce anxiety and depression
  - Individuals with diabetes and cardiovascular disease have higher rates of depression, therefore this may an area that is important to the participants

  *Helps maintain strong and healthy joints*
  - As people age, muscles work on a “use it or lose it” basis, therefore for people that don’t do much exercise the muscles around their joints become weaker over time, this puts more pressure on their joints which will lead to joint pain and discomfort. For people who already experience joint pain or have osteoarthritis it is very important that weight bearing (walking etc) exercise be attempted. Although it may be painful to start off with, over time your muscles will become stronger, which will lead to greater joint stability and lessen the pain. For those with joint pain that do no exercise, the pain is likely to continue to get worse over time.

Summarize the points of this section so far and emphasize the key for people with IGT is insulin resistance and blood glucose and that if these factors can be reduced they will automatically reduce their risk of both diabetes and heart disease.
Now we have established the benefits of increased physical activity, can anyone tell me what the current recommendations are on how intense the physical activity should be, and how much and how often it should be done?

Write down all the answers on the flip chart under sections intensity, duration and frequency. Once the suggestions have finished, write down the correct answer.

The government currently recommends 30 minutes of moderate intensity on most days of the week. The World Health Organization recommends 60 minutes of moderate intensity exercise on most days of the week. The 30-minute a day recommendation applies to the population as a whole and will help reduce blood pressure and increase good (HDL) cholesterol as well as improving overall cardiac and vascular function. The 60-minute a day recommendation is designed for people who want to lose or maintain their body weight and to reduce some of the problems associated with being overweight.

Taking into account recent evidence, it would appear that in terms of helping you control your blood glucose levels at least 45 minutes of moderate intensity activity is likely to be needed on most days of the week – which is in-between these two recommendation.

Let’s look at what is meant by a recommendation of 30 minutes of moderate intensity on every day of the week. Lets concentrate on the word moderate. What do you think is meant by moderate?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

Moderate exercise is exercise that makes you slightly breathless and sweaty, but not so breathless that you can’t talk and not so easy that you could sing. A good example is walking.

If there is room demonstrate examples of low-intensity, moderate-intensity and vigorous-intensity activity by walking around the room.

Now let’s move on to the actual time itself. When I say that the minimum needed to reduce your blood glucose level is 45 minutes of moderate intensity physical activity every day, do you think that the 45 minutes has to be done all at once or do you think it can be accumulated in lots of little bits?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The 45 minutes can be broken down into smaller chunks and accumulated throughout the day.
Does anyone know the minimum time that each chunk should be in order for it to count towards your daily goal?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

The minimum time each chunk can be is 10 minutes.

**Pedometer version:** I hope you have all been wearing your pedometers and have some idea about how many steps you take each day. Would anyone like to guess how many steps you would take if you walked at a moderate-intensity for 30 minutes?

**Pedometer version:** Convert the exercise recommendation to pedometer counts; 30 minutes of moderate-paced walking = 3000 steps, 45 minutes of moderate-paced walking = 4500 steps and 60 minutes of moderate paced walking = 6000 steps.

**Pedometer version:** Explain that this means that individuals need to increase their activity levels by 3000 steps in order to reach the 30 minutes a day goal, 4500 in order to reach the 45 minutes goal and 6000 steps in order to reach the 60 minutes goal. Therefore each person’s goal will be different, although anyone already averaging more than 9000 steps/day is already reaching the 30 minute a target, anyone already doing more than 10,500 steps per day is already reaching the 45 minute target anyone already doing more than 12,000 steps per day is already reaching the 60 minute target.

**Pedometer version:** Hand-out each person’s baseline pedometer counts on a card, and help them form realistic step/day goals based on their normal activity levels.

Good, so now we have established what is meant by the physical activity recommendations I want us to think about ways in which we can increase our moderate physical activity levels in home, town, and work environments. Let’s start with home environments. Who can think of ways of increasing our moderate-intensity levels of physical activity in your home environment?

Write participants’ ideas down. Depending on who you have in the group use open questions to try to get the answers to include:

- Encourage your partner or friends to come on walks with you
- Vacuum the house vigorously
- Walk your dog, or offer to walk your neighbour’s dog
- Walk to your neighbour’s or friend’s house instead of ringing them
- Walk to the corner shop to buy a newspaper
- Park your car further along the road from your house
- When on the phone, walk while you talk.
- When watching television, get up and walk around your house when the adverts come on.
- Plan walks into your day
• Use the weekends to plan walks in the countryside: try to visit new areas to stop routes becoming boring and reward yourself at the end of each walk, for example by having lunch in the local pub.
• Go for a walk while your children are playing sport.

Emphasize that household duties need to be done at a high enough intensity in order to count towards daily activity levels and that activities like washing-up are classified as light-intensity.

Good, so we have managed to think of some good ways of increasing our physical activity in our home environments, can anyone think of ways to increase their physical activity when shopping?

Write participants ideas down. Use open questions to try to get the answers to include:
• Avoid lifts and escalators – take the stairs instead.
• In supermarket car parks, park further away from the shop doors
• When unpacking your shopping from the car, make several trips
• Walk briskly between shops – don’t amble
• Walk, don’t drive, for journeys of less than one mile.
• Walk at the airport while waiting for your plane, and avoid the moving walkways.

OK so now we have some good ideas about ways of increasing physical activity levels while out shopping, can anyone think of ways to increase their physical activity in a work environment?

If appropriate acknowledge the fact that not everyone in the group might be working.

Write participants’ ideas down. Use open questions to try to get the answers to include:
• Get off the bus one or more stops early so as to walk further on your way to work.
• Take several 10-minute walks during the day by walking a few laps of your floor during breaks, or by going outside and walking around the block.
• Choose the furthest entrance to your building, then walk the long way to your office.
• Walk to a toilet, water fountain, or photocopier on a different floor.
• Take a longer route to your meeting.
• Walk a few laps of your floor during breaks, or go outside and walk around the block.
• Take a walk during your lunch break.
• Walk to a colleague’s office rather than ringing or sending email.
• Park further away in the morning
• Take the stairs rather than the lift or the escalator.
• Encourage co-workers to go for walks with you
• Walk around while using a speaker or cordless phone.
• Get up and move about at least once every 30 minutes.
Give examples from the list of how you, the educator, would fit 45 minutes of walking activity into your day.

I would now like to go around the group and ask you each to tell us the main reasons you think fitting 45 minutes of exercise into your day will be difficult?

Give an example of one of your barriers to exercise.

Write down each suggestion under barriers.

OK, thank you for sharing those barriers. Can anyone think how some of these barriers might be overcome?

Collect the solutions and write them down. If no solutions are forthcoming, use open questions to elicit appropriate responses.

Hopefully that has provided some helpful ways of getting around these barriers. Remember that there are no easy ways of trying to fit regular physical activity into your daily lives and it will need a lot of conscious effort and commitment on your part, but I hope from what you have learned here today most of you will agree the benefits of being more active are worth the effort.

Let's talk a bit more about increasing your activity levels. Do you think it would be sensible to try and attempt 45 minutes a day straight away, or might it be better to build up to the target slowly?

Collect answers and acknowledge responses and try to elicit the following. If no answers are forthcoming move on and give the correct answer.

It is better to build up to the 45 minutes slowly?

Good, so some of you feel that it would be better to try and build up to the target slowly. Can any of you think why it would be a good idea to build up to the target slowly?

Write answers down. Try and encourage answers such as:
• Making a big change straight away is likely to lead to sore muscles which in turn will make it unlikely the activity will be sustained because it will hurt
• Trying to make such big change all at once will heighten the chance of failure which in turn is likely to lead to de-motivation

Good, so as we have established there are some good reasons for building up your final physical activity targets in small steps. So, for example I might decide to increase my physical activity levels by 5 to 10 minutes (pedometer version 500 to 1000 steps per day for the) every fortnight until I have reached by final target.

Recap the main points so far.

Answer any questions that are asked.
BREAK FOR TEA AND COFFEE.

Go through the action plan and diaries, use the example page to highlight how the diaries are used.

Emphasis the importance of developing action plans and keeping a daily record of physical activity levels.

Emphasise again the importance of increasing physical activity levels incrementally using weekly or fortnightly goal; participants should aim for an increase of around 5 to 10 minutes or 500 to 1000 steps per fortnight until their final goal is reached.

Give participants 10 minutes to fill in their first action plan.

Explain the Lands Ends to John O’ Groats challenge.
Appendix Five

Participant booklet for the PREPARE Programme
The PREPARE Study
Pre-Diabetes Risk Education and Physical Activity Recommendation and Encouragement

Participant Booklet
Impaired glucose tolerance information sheet

What is impaired glucose tolerance?

Impaired glucose tolerance is a condition where the body’s ability to properly regulate its glucose levels becomes impaired. This usually begins as a problem at the cellular level when the body’s cells start to become “rusty” which makes it harder for insulin to help glucose into the cells where it is used as fuel for energy. This puts pressure on the pancreas to produce more insulin. Because of the higher demand on the pancreas, over time, it will start to become tired and damaged, which will lead to less insulin being produced and higher blood glucose levels. Eventually, the pancreas will become damaged beyond a point where it can be repaired and this is usually when diabetes sets in.

What is the risk of diabetes?

Every year up to 14% of individuals with impaired glucose tolerance will get diabetes which means that around 50% of individuals with impaired glucose tolerance will develop diabetes in their lifetime.

Is there anything I can do to lower my risk of diabetes?

Yes, by eating a healthy diet and particularly by being more physically active you can dramatically lower your risk of diabetes. Those that engage in regular physical activity have half the risk of diabetes compared to those who do no exercise.

Are there any other risk associated with impaired glucose tolerance?

Yes. Individuals with impaired glucose tolerance are also at an increased risk of cardiovascular disease. This means that individuals with impaired glucose tolerance have a high risk of having a heart attack, heart disease, stroke or angina.

Are there any symptoms associated with impaired glucose tolerance?

There are generally no symptoms associated with impaired glucose tolerance.
**What causes impaired glucose tolerance?**

The most common causes of impaired glucose tolerance are: a family history of diabetes, being overweight or obese, and not doing enough exercise.

**How does exercise help impaired glucose tolerance:**

Exercise helps impaired glucose tolerance by making it easier for glucose to get into the body's cells to be used as energy. This means that less glucose remains in the blood.

**How much exercise do I need to be doing?**

In order to effectively control your blood glucose levels you need to be doing a minimum of 45 minutes of moderate intensity exercise everyday. The 45 minutes can be accumulated throughout the day in 10 minute chunks and can include such things as brisk walking, swimming, cycling, aerobics or everyday activity such as vacuuming the house, washing the floor, digging the garden or mowing the lawn, as long as these activities are done vigorously enough to make you somewhat breathless. If you do start an exercise programme remember to always start slowly and gradually increase the intensity and length of your sessions over time. Weight lifting is also a good form of exercise and many of the health benefits associated with aerobic exercise can be gained from following a weight lifting program. However the disadvantages of this type of exercise are that you will probably need to buy gym membership which can be expensive, and in order to be effective you will need specialist instruction to start with.

**Can increasing my activity levels be dangerous?**

Doing moderate levels of activity like walking should not normally present a danger to your health. However, if you have a history of heart disease or exercising makes you feel dizzy or gives you pains in your chest you should consult a doctor. If you plan to start doing vigorous forms of exercise that involve running or lifting heavy weights you should always consult your doctor before you start in order to rule out any underlying problems that may become exacerbated by vigorous activity.
# My health profile

<table>
<thead>
<tr>
<th><strong>2-h glucose</strong></th>
<th>7.8 mmol/l</th>
<th>9 mmol/l</th>
<th>11.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal glucose control</td>
<td>Pre-diabetes</td>
<td>Pre-diabetes</td>
<td>Type 2 diabetes</td>
</tr>
<tr>
<td></td>
<td>• High risk of diabetes</td>
<td>• Very high risk of diabetes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Increased risk of cardiovascular disease</td>
<td>• High risk of cardiovascular disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Make necessary lifestyle changes</td>
<td>• Make necessary lifestyle changes</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Fasting glucose</strong></th>
<th>6 mmol/l</th>
<th>7 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal glucose control</td>
<td>Pre-diabetes</td>
<td>Type 2 diabetes</td>
</tr>
<tr>
<td></td>
<td>• High risk of diabetes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Increased risk of cardiovascular disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Make necessary lifestyle changes</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Systolic blood pressure</strong></th>
<th>140 mmHg</th>
<th>160 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Borderline hypertension</td>
<td>Definite hypertension</td>
</tr>
<tr>
<td></td>
<td>• Elevated risk of cardiovascular disease</td>
<td>• High risk of cardiovascular disease</td>
</tr>
<tr>
<td></td>
<td>• Make necessary lifestyle changes</td>
<td>• Make necessary lifestyle changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Diastolic blood pressure</strong></th>
<th>90 mmHg</th>
<th>100 mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Borderline hypertension</td>
<td>Definite hypertension</td>
</tr>
<tr>
<td></td>
<td>• Elevated risk of cardiovascular disease</td>
<td>• High risk of cardiovascular disease</td>
</tr>
<tr>
<td></td>
<td>• Make necessary lifestyle changes</td>
<td>• Make necessary lifestyle changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Total cholesterol</strong></th>
<th>5 mmol/l</th>
<th>6.5 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desirable</td>
<td>Borderline</td>
<td>High</td>
</tr>
<tr>
<td></td>
<td>• Elevated risk of cardiovascular disease</td>
<td>• High risk of cardiovascular disease</td>
</tr>
<tr>
<td></td>
<td>• Make necessary lifestyle changes</td>
<td>• Make necessary lifestyle changes</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>HDL (good) cholesterol</strong></th>
<th>1 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Undesirable</td>
<td>Desirable</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>LDL (bad) cholesterol</strong></th>
<th>3 mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desirable</td>
<td>Undesirable</td>
</tr>
</tbody>
</table>
Physical Activity Diary
Example (standard version): Week 1

Weekly schedule:

<table>
<thead>
<tr>
<th>Action to be taken</th>
<th>When</th>
<th>Where</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weekday action plan</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Walk to the shop to buy the paper</td>
<td>1) First thing in the morning</td>
<td>1) At home</td>
</tr>
<tr>
<td>2) Take a 10 minute walk</td>
<td>2) At lunch time</td>
<td>2) At work</td>
</tr>
<tr>
<td>Saturday action plan</td>
<td>Walk into town</td>
<td>From the car park on the outside of town</td>
</tr>
<tr>
<td>Sunday action plan</td>
<td>Go for 20 minute walk in the countryside</td>
<td>In the morning</td>
</tr>
</tbody>
</table>

Weekly diary:

**Total time spent exercising this week = 135 minutes**

- **Monday**
  - Date: 12/04/06
  - Actions taken:
    - 10 minute walk to buy the paper
    - 10 minute walk at lunch time
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 20 mins

- **Tuesday**
  - Date: 13/04/06
  - Actions taken:
    - 10 minute walk to buy the paper
    - 10 minute walk at lunch time
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 20 mins

- **Wednesday**
  - Date: 15/04/06
  - Actions taken:
    - No exercise today - feeling ill
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 0 mins

- **Thursday**
  - Date: 16/04/06
  - Actions taken:
    - 10 minute walk to buy the paper
    - 20 minute walk at lunch time
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 30 mins

- **Friday**
  - Date: 17/04/06
  - Actions taken:
    - 15 minute walk at lunch time
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 15 mins

- **Saturday**
  - Date: 18/04/06
  - Actions taken:
    - 10 minutes from the car into town
    - 10 minutes from town back to the car
  - Total time spend walking or engaging in moderate or vigorous exercise (only count activities that lasted longer than 10 minutes): 20 mins
**Example: Week 1 (pedometer version)**

**Weekly schedule:**

*My goal for this week is:* Increase my walking activity to a total of 6000 steps per day or 42000 steps per week.

<table>
<thead>
<tr>
<th>Action to be taken</th>
<th>When</th>
<th>Where</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Weekday action plan</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1) Walk to the shop to buy the paper</td>
<td>1) First thing in the morning</td>
<td>1) At home</td>
</tr>
<tr>
<td>2) Take a 10 minute walk</td>
<td>2) At Lunch time</td>
<td>2) At work</td>
</tr>
<tr>
<td><strong>Saturday action plan</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Walk into town</td>
<td>Midmorning</td>
<td>From the car park on the outside of town</td>
</tr>
<tr>
<td><strong>Sunday action plan</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Go for 20 minute walk in the countryside</td>
<td>In the morning</td>
<td>Around the Outwoods</td>
</tr>
</tbody>
</table>

**Weekly diary:**

Total number of steps taken this week = 43086

**Sunday**

*Date: 11/04/06*

Actions taken:
30 minute walk in the countryside

**Monday**

*Date: 12/04/06*

Actions taken:
10 minute walk to buy the paper
10 minute walk at lunch time

Total number of steps taken today: 6579

**Tuesday**

*Date: 13/04/06*

Actions taken:
10 minute walk to buy the paper
10 minute walk at lunch time

Total number of steps taken today: 6321

**Wednesday**

*Date: 15/04/06*

Actions taken:
No exercise today - feeling ill

Total number of steps taken today: 4300

**Thursday**

*Date: 16/04/06*

Actions taken:
10 minute walk to buy the paper
20 minute walk at lunch time

Total number of steps taken today: 7128

**Friday**

*Date: 17/04/06*

Actions taken:
15 minute walk at lunch time

Total number of steps taken today: 5931

**Saturday**

*Date: 18/04/06*

Actions taken:
10 minutes from the car into town
10 minutes from town back to the car

Total number of steps taken today: 6487
The Lands End to John O' Groats challenge:

<table>
<thead>
<tr>
<th>Date</th>
<th>Total walking time</th>
<th>Total distance travelled</th>
</tr>
</thead>
<tbody>
<tr>
<td>23/02/06</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Conversion factor: 1 hour walking = 3 miles
10 minutes walking = 0.5 miles
Appendix Six

Information sheet sent to the control group as part of the

PREPARE Programme Study
Dear Patient,

Thank you for attending Leicester Royal Infirmary recently as part of the PREPARE study. We hope you will continue to participate in the study in the coming year. Please find enclosed some information on pre-diabetes and exercise, which we hope will answer any questions about pre-diabetes you may have at present.

We look forward to seeing you again,

Yours sincerely,

Thomas Yates
Pre-diabetes information sheet

What is pre-diabetes?

Pre-diabetes is a condition where the body’s ability to properly regulate its glucose levels becomes impaired. This usually begins as a problem at the cellular level when the body’s cells start to become “rusty” which makes it harder for insulin to help glucose into the cells where it is used as fuel for energy. This puts pressure on the pancreas to produce more insulin. Because of the higher demand on the pancreas over time it will start to become tired and damaged, which will lead to less insulin being produced and higher blood glucose levels. Eventually the pancreas will become damaged beyond a point where it can repair itself and this is usually when diabetes is diagnosed.

What is the risk of diabetes?

Every year up to 14% of individuals with pre-diabetes will get diabetes which means that around 50% of individuals with pre-diabetes will develop diabetes in their lifetime.

Is there anything I can do to lower my risk of diabetes?

Yes, by eating a healthy diet and particularly by being more physical active you can dramatically lower your risk of diabetes. Those that engage in regular physical activity have half the risk of diabetes compared to those who do no exercise.

Are there any other risk associated with pre-diabetes?

Yes. Individuals with pre-diabetes are also at an increased risk of cardiovascular disease. This means that individuals with pre-diabetes have an elevated risk of having a heart attack, stroke, heart disease, or angina.

Are there any symptoms associated with pre-diabetes?

There are generally no symptoms associated with pre-diabetes.

What causes pre-diabetes?

The most common causes of pre-diabetes are: a family history of diabetes, being overweight or obese, and not doing enough exercise.
**How does exercise help pre-diabetes:**

Exercise helps pre-diabetes by making it easier for glucose to get into the bodies cells to be used as energy. This means that less glucose remains in the blood.

**How much exercise do I need to be doing?**

In order to effectively control your blood glucose levels you need to be doing a minimum of 45 minutes of moderate intensity on most days of the week. The 45 minutes can be accumulated throughout the day in 10 minute chunks and can include such things as brisk walking, swimming, cycling, aerobics or everyday activity such as vacuuming the house, washing the floor, digging the garden or mowing the lawn, as long as these activities are done vigorously enough to make you somewhat breathless. If you do start an exercise programme remember to always start slowly and gradually increase the intensity and length of your sessions over time. Weight lifting is also a good form of exercise and many of the health benefits associated with aerobic exercise can be gained from following a weight lifting program. However the disadvantages of this type of exercise are that you will probably need to buy gym membership which can be expensive, and in order to be effective you will need specialist instruction to start with.

**Can increasing my activity levels be dangerous?**

Doing moderate levels of activity like walking should not normally present a danger to your health. However if you have a history of heart disease or exercising makes you feel dizzy or gives you pains in your chest you should consult a doctor. If you plan to start doing vigorous forms of exercise that involve running or lifting heavy weights you should always consult your doctor before you start in order to rule out any underlying problems that may become exacerbated by vigorous activity.
Appendix Seven

International physical activity questionnaire
We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous and moderate activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal.

**PART 1: JOB-RELATED PHYSICAL ACTIVITY**

The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home. Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. These are asked in Part 3.

1. **Do you currently have a job or do any unpaid work outside your home?**
   - Yes
   - No **→ Skip to PART 2: TRANSPORTATION**

The next questions are about all the physical activity you did in the **last 7 days** as part of your paid or unpaid work. This does not include traveling to and from work.

2. **During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, heavy construction, or climbing up stairs as part of your work?** Think about only those physical activities that you did for at least 10 minutes at a time.
   - ___ days per week
   - No vigorous job-related physical activity **→ Skip to question 4**

3. **How much time did you usually spend on one of those days doing vigorous physical activities as part of your work?**
   - ___ hours per day
   - ___ minutes per day

4. **Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads as part of your work?** Please do not include walking.
   - ___ days per week
   - No moderate job-related physical activity **→ Skip to question 6**
5. How much time did you usually spend on one of those days doing moderate physical activities as part of your work?

______ hours per day
______ minutes per day

6. During the last 7 days, on how many days did you walk for at least 10 minutes at a time as part of your work? Please do not count any walking you did to travel to or from work.

______ days per week
☐ No job-related walking → Skip to PART 2: TRANSPORTATION

7. How much time did you usually spend on one of those days walking as part of your work?

______ hours per day
______ minutes per day

PART 2: TRANSPORTATION PHYSICAL ACTIVITY

These questions are about how you traveled from place to place, including to places like work, stores, movies, and so on.

8. During the last 7 days, on how many days did you travel in a motor vehicle like a train, bus, car, or tram?

______ days per week
☐ No traveling in a motor vehicle → Skip to question 10

9. How much time did you usually spend on one of those days traveling in a train, bus, car, tram, or other kind of motor vehicle?

______ hours per day
______ minutes per day

Now think only about the bicycling and walking you might have done to travel to and from work, to do errands, or to go from place to place.

10. During the last 7 days, on how many days did you bicycle for at least 10 minutes at a time to go from place to place?

______ days per week
☐ No bicycling from place to place → Skip to question 12
11. How much time did you usually spend on one of those days to bicycle from place to place?

___ hours per day

___ minutes per day

12. During the last 7 days, on how many days did you walk for at least 10 minutes at a time to go from place to place?

___ days per week

☐ No walking from place to place → Skip to PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

13. How much time did you usually spend on one of those days walking from place to place?

___ hours per day

___ minutes per day

PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY

This section is about some of the physical activities you might have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family.

14. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, chopping wood, shoveling snow, or digging in the garden or yard?

___ days per week

☐ No vigorous activity in garden or yard → Skip to question 16

15. How much time did you usually spend on one of those days doing vigorous physical activities in the garden or yard?

___ hours per day

___ minutes per day

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, sweeping, washing windows, and raking in the garden or yard?

___ days per week

☐ No moderate activity in garden or yard → Skip to question 18
17. How much time did you usually spend on one of those days doing moderate physical activities in the garden or yard?

____ hours per day
____ minutes per day

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate activities like carrying light loads, washing windows, scrubbing floors and sweeping inside your home?

____ days per week

☐ No moderate activity inside home → Skip to PART 4: RECREATION, SPORT AND LEISURE-TIME PHYSICAL ACTIVITY

19. How much time did you usually spend on one of those days doing moderate physical activities inside your home?

____ hours per day
____ minutes per day

PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY

This section is about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during the last 7 days, on how many days did you walk for at least 10 minutes at a time in your leisure time?

____ days per week

☐ No walking in leisure time → Skip to question 22

21. How much time did you usually spend on one of those days walking in your leisure time?

____ hours per day
____ minutes per day

22. Think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do vigorous physical activities like aerobics, running, fast bicycling, or fast swimming in your leisure time?

____ days per week

☐ No vigorous activity in leisure time → Skip to question 24
23. How much time did you usually spend on one of those days doing vigorous physical activities in your leisure time?

______ hours per day
______ minutes per day

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do moderate physical activities like bicycling at a regular pace, swimming at a regular pace, and doubles tennis in your leisure time?

______ days per week

☐ No moderate activity in leisure time  → Skip to PART 5: TIME SPENT SITTING

25. How much time did you usually spend on one of those days doing moderate physical activities in your leisure time?

______ hours per day
______ minutes per day

PART 5: TIME SPENT SITTING

The last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television. Do not include any time spent sitting in a motor vehicle that you have already told me about.

26. During the last 7 days, how much time did you usually spend sitting on a weekday?

______ hours per day
______ minutes per day

27. During the last 7 days, how much time did you usually spend sitting on a weekend day?

______ hours per day
______ minutes per day
Appendix Eight

DINE food frequency questionnaire
These questions are about some foods which you may eat. Please answer all the questions by ticking the box you think most applies to you.

1. About how many pieces or slices *per day* do you eat the following types of bread, rolls, or chapattis (*Please tick the box you think most applies to you*)

<table>
<thead>
<tr>
<th></th>
<th>None</th>
<th>Less than 1 a day</th>
<th>1 to 2 a day</th>
<th>3 to 4 a day</th>
<th>5 or more a day</th>
</tr>
</thead>
<tbody>
<tr>
<td>White bread rolls, chapattis or parathas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brown bread or rolls, or brown chapattis, or parathas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wholemeal bread, rolls, chapattis, or parathas</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. About how many servings *per week* do you eat of the following types of breakfast cereal or porridge (*Please tick the box you think most applies to you*)

**Sugared type**: e.g. Frosties, Coco Pops, Ricicles, Sugar Puffs

**Rice or Corn type**: e.g. Corn Flakes, Rice Krispies, Special K

**Porridge** or Ready Brek

**Wheat type**: e.g. Shredded Wheat, Weetabix, Fruit 'n Fibre, Puffed Wheat, Nutri-grain, Start

**Museli type**: Alpen, Jordan's

**Bran type**: All-bran, Bran Flakes, Sultana Bran

![Table for breakfast cereals and porridge](https://example.com/table.png)

249
3. How many times a **week** do you eat a serving of the following foods?

**Please tick the box you think most applies to you**

<table>
<thead>
<tr>
<th>None</th>
<th>Less than 1 a week</th>
<th>1 to 2 a week</th>
<th>3 to 5 a week</th>
<th>6 to 7 a week</th>
<th>8 to 11 a week</th>
<th>12 or more a week</th>
</tr>
</thead>
</table>

**Pasta, rice, or dishes made from grains much as millet, semolina and cornmeal**
Rice: include plain boiled rice, rice and peas, pilau and biryani

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Potatoes (excluding chips), yams, cassava, plantains, breadfruit, sweet potatoes or taro/eddo**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Peas, lentils (dal) or beans (including baked beans)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Other types of vegetables (cooked or raw as in salads)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Fruit (including fresh, frozen or canned fruit)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. About how many times a **week** do you eat a serving of the following foods?

**Please tick the box you think most applies to you**

<table>
<thead>
<tr>
<th>None</th>
<th>Less than 1 a week</th>
<th>1 to 2 a week</th>
<th>3 to 5 a week</th>
<th>6 or more a week</th>
</tr>
</thead>
</table>

**Cheese (any except cottage)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Beef, pork or lamb**

*INCLUDE: burgers, sausages, bacon, ham, meat pies, meat curries, casseroles, and processed meat*

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Chicken or turkey (including processed types)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Fish (not including fried fish)**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Any fried food**

*INCLUDE fried fish, fried chicken, chips, fried breakfast, samosas, West Indian soup or stew, fried rice, puris, and bhajis*

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Cakes, pies, puddings, pastries, or Indian sweets**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Sweet or savory snakes such as chocolate, crisps, biscuits, Bombay mix, sev and chanachur**

<p>| | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

250
About how much of the following types of milk do you usually use *per day*, for example in cereal, tea, or coffee (choose one answer from each line)

<table>
<thead>
<tr>
<th>Type</th>
<th>None</th>
<th>Less than a quarter pint</th>
<th>About a quarter pint</th>
<th>About a pint</th>
<th>One pint or more</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full cream (silver top) or Channel Islands (gold top)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Semi-skimmed (green or red striped top)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skimmed (red or blue checked top)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

About how many rounded teaspoons *per day* do you usually use of the following types of spread, for example on bread, chapattis, potatoes, vegetables or dhal? (Choose one answer from each line)

<table>
<thead>
<tr>
<th>Type</th>
<th>None</th>
<th>1 tsp</th>
<th>2 tsp</th>
<th>3 tsp</th>
<th>4 tsp</th>
<th>5 tsp</th>
<th>6 tsp</th>
<th>7 or more tsp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Butter, ghee or margarine (e.g. Flora, Vitalite, Sunflower types, blue band, Golden crown, Olivio etc)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low fat spreads (e.g. Shape, Delight, Flora Lite, Half fat butter, half fat ghee, etc)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

What sort of fat do you usually use for the following purposes (Choose one from each line)

- **Butter ghee or lard**
- **Cooking fat, hard margarine, half butter or ghee**
- **Soft margarine or reduced fat spread**
- **Vegetable oil, olive oil, rape seed oil, peanut oil, corn oil etc**
- **No fat used**

- As a spread on bread, chapattis, vegetables etc
- For frying
- For baking and cooking
Appendix Nine

Brief Illness perception questionnaire
These questions are about what you think about pre-diabetes

On the following questions, please circle the number that best corresponds to your views

How much does pre-diabetes affect your life?

<table>
<thead>
<tr>
<th>It does not affect my life at all</th>
<th>Severely affects my life</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How long do you think you will have pre-diabetes for?

<table>
<thead>
<tr>
<th>A very short time</th>
<th>Forever</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How much control do you feel you have over your pre-diabetes?

<table>
<thead>
<tr>
<th>Absolutely no control</th>
<th>Complete control</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How much do you think exercise can help control your pre-diabetes?

<table>
<thead>
<tr>
<th>Not at all</th>
<th>Extremely helpful</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

Are you experiencing any symptoms from having pre-diabetes?

<table>
<thead>
<tr>
<th>No symptoms at all</th>
<th>Many severe symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How concerned are you about having pre-diabetes?

<table>
<thead>
<tr>
<th>Not at all concerned</th>
<th>Extremely concerned</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How well do you feel you understand pre-diabetes?

<table>
<thead>
<tr>
<th>Don't understand it at all</th>
<th>Understand it very clearly</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>

How much does having pre-diabetes affect you emotionally (e.g. does it make you angry, scared, upset or depressed)?

<table>
<thead>
<tr>
<th>Not at all affected emotionally</th>
<th>Extremely affected emotionally</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
</tr>
</tbody>
</table>
Appendix Ten

Walking self-efficacy questionnaire
These questions are designed to measure your confidence in your ability to walk and exercise for different times every day.

Scale

Please use the scale and example below to help you answer these questions.

<table>
<thead>
<tr>
<th>0%</th>
<th>10%</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
<th>50%</th>
<th>60%</th>
<th>70%</th>
<th>80%</th>
<th>90%</th>
<th>100%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completely certain I could not do it</td>
<td>Moderately certain that I could do it</td>
<td>Completely certain I could do it</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Example

This question is about running

On the following questions, please circle the percentage that best corresponds to your views.

How confident are you that you could run for 20 minutes every day

This answer indicates the person has little confidence in their ability to run for 20 minutes every day.

This set of questions is about walking.

On the following questions, please circle the percentage that best corresponds to your views for each question.

How confident are you that you could walk for 10 minutes every day

How confident are you that you could walk for 20 minutes every day

How confident are you that you could walk for 30 minutes every day

How confident are you that you could walk for 40 minutes every day
How confident are you that you could walk for 50 minutes every day

10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
0%

How confident are you that you could walk for 60 minutes every day

10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
0%
Appendix Eleven

Exercise self-efficacy questionnaire
Please think about any exercise you like doing other than walking. This might be swimming, playing badminton, cycling, golf etc. Please do not include such sports as bowls, darts, snooker etc.

On the following questions, please circle the percentage that best corresponds to your views for each question.

How confident are you that you could exercise for 10 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%

How confident are you that you could exercise for 20 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%

How confident are you that you could exercise for 30 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%

How confident are you that you could exercise for 40 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%

How confident are you that you could exercise for 50 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%

How confident are you that you could exercise for 60 minutes every day

- 0%
- 10%
- 20%
- 30%
- 40%
- 50%
- 60%
- 70%
- 80%
- 90%
- 100%
Appendix Twelve

Exercise self-regulatory efficacy questionnaire
Please use the scale below to help you answer these questions.

<table>
<thead>
<tr>
<th>0%</th>
<th>10%</th>
<th>20%</th>
<th>30%</th>
<th>40%</th>
<th>50%</th>
<th>60%</th>
<th>70%</th>
<th>80%</th>
<th>90%</th>
<th>100%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Completely certain I could not do it</td>
<td>Moderately certain that I could do it</td>
<td>Completely certain I could do it</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

On the following questions, please circle the percentage that best corresponds to your views for each question.

How confident are you that you could exercise in the following circumstances?

When I am feeling tired

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

When I am in a bad mood

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

During bad weather

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

When I feel I don't have the time

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%

When I am on holiday

0% 10% 20% 30% 40% 50% 60% 70% 80% 90% 100%
Appendix Thirteen

Change from baseline and the associated intervention effect for blood pressure, blood lipids and waist circumference at 3, 6 and 12 months
<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Intervention 1 (PREPARE)</th>
<th>Intervention 2 (PREPARE + pedometer)</th>
<th>Adjusted intervention effect (Intervention 1 vs. control)</th>
<th>p</th>
<th>Adjusted intervention effect (Intervention 2 vs. control)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Blood Pressure</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmHg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-months</td>
<td>-0.5 (-5.6 to 4.6)</td>
<td>4.0 (-2.0 to 10)</td>
<td>-0.3 (-5.1 to 4.4)</td>
<td>5.5 (-1.8 to 12.8)</td>
<td>0.136</td>
<td>0.1 (-7.0 to 7.2)</td>
<td>0.789</td>
</tr>
<tr>
<td>6-month</td>
<td>-4.4 (-8.4 to -0.4)</td>
<td>-0.2 (-5.5 to -5.1)</td>
<td>-3.3 (-10.3 to 3.3)</td>
<td>4.6 (-2.5 to 11.7)</td>
<td>0.203</td>
<td>0.5 (-6.5 to 7.6)</td>
<td>0.886</td>
</tr>
<tr>
<td>12-months</td>
<td>-6.8 (-12.6 to -1.0)</td>
<td>-3.3 (-9.3 to 2.7)</td>
<td>0.3 (-4.8 to 5.4)</td>
<td>4.6 (-2.4 to 11.7)</td>
<td>0.194</td>
<td>6.6 (-0.4 to 13.5)</td>
<td>0.065</td>
</tr>
<tr>
<td><strong>Total Cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>-0.00 (-0.34 to 0.33)</td>
<td>-0.03 (-0.17 to 0.11)</td>
<td>-0.12 (-0.38 to 0.14)</td>
<td>-0.01 (-0.35 to 0.33)</td>
<td>0.938</td>
<td>-0.14 (-0.47 to 0.19)</td>
<td>0.413</td>
</tr>
<tr>
<td>6-months</td>
<td>0.05 (-0.28 to -0.37)</td>
<td>0.03 (-0.25 to 0.30)</td>
<td>-0.26 (-0.47 to 0.06)</td>
<td>-0.04 (-0.40 to 0.33)</td>
<td>0.845</td>
<td>-0.34 (-0.70 to 0.03)</td>
<td>0.070</td>
</tr>
<tr>
<td>12-months</td>
<td>0.25 (-0.01 to 0.50)</td>
<td>-0.02 (-0.20 to 0.18)</td>
<td>-0.04 (-0.34 to 0.25)</td>
<td>-0.23 (-0.58 to 0.12)</td>
<td>0.191</td>
<td>-0.28 (-0.62 to 0.07)</td>
<td>0.116</td>
</tr>
<tr>
<td><strong>HDL-cholesterol</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mmol/l)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-month</td>
<td>0.02 (-0.06 to 0.11)</td>
<td>-0.08 (-0.15 to -0.02)</td>
<td>-0.08 (-0.14 to -0.02)</td>
<td>-0.10 (-0.21 to 0.01)</td>
<td>0.069</td>
<td>-0.03 (-0.14 to 0.08)</td>
<td>0.566</td>
</tr>
<tr>
<td>6-months</td>
<td>-0.04 (-0.11 to 0.02)</td>
<td>-0.05 (-0.14 to 0.04)</td>
<td>-0.08 (-0.14 to -0.02)</td>
<td>0.01 (-0.09 to 1.00)</td>
<td>0.898</td>
<td>-0.02 (-0.12 to 0.07)</td>
<td>0.641</td>
</tr>
<tr>
<td>12-months</td>
<td>0.02 (-0.04 to 0.09)</td>
<td>-0.01 (-0.08 to 0.06)</td>
<td>-0.01 (-0.07 to 0.04)</td>
<td>-0.02 (-0.11 to 0.06)</td>
<td>0.634</td>
<td>-0.04 (-0.12 to 0.05)</td>
<td>0.398</td>
</tr>
</tbody>
</table>