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ARTICLE

Improved lifestyle and decreased diabetes risk over 13 years: long-term follow-up of the randomised Finnish Diabetes Prevention Study (DPS)

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Abstract
Aims/hypothesis This study aimed to determine whether lifestyle intervention lasting for 4 years affected diabetes incidence, body weight, glycaemia or lifestyle over 13 years among individuals at high risk of type 2 diabetes.

Methods Overweight, middle-aged men (n=172) and women (n=350) with impaired glucose tolerance were randomised in 1993–1998 to an intensive lifestyle intervention group (n=265), aiming at weight reduction, dietary modification and increased physical activity, or to a control group

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(n=257) that received general lifestyle information. The primary outcome was a diagnosis of diabetes based on annual OGTTs. Secondary outcomes included changes in body weight, glycaemia, physical activity and diet. After active intervention (median 4 years, range 1–6 years), participants still free of diabetes and willing to continue their participation (200 in the intervention group and 166 in the control group) were further followed until diabetes diagnosis, dropout or the end of 2009, with a median total follow-up of 9 years and a time span of 13 years from baseline.

Results During the total follow-up the adjusted HR for diabetes (intervention group vs control group) was 0.614 (95% CI 0.478, 0.789; p<0.001). The corresponding HR during the post-intervention follow-up was 0.672 (95% CI 0.477, 0.947; p=0.023). The former intervention group participants sustained lower absolute levels of body weight, fasting and 2 h plasma glucose and a healthier diet. Adherence to lifestyle changes during the intervention period predicted greater risk reduction during the total follow-up.

Conclusions/interpretation Lifestyle intervention in people at high risk of type 2 diabetes induces sustaining lifestyle change and results in long-term prevention of progression to type 2 diabetes.

Trial registration ClinicalTrials.gov NCT00518167

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Keywords Diet · Lifestyle · Long-term · Physical activity · Prevention · Randomised controlled study · Type 2 diabetes · Weight

Abbreviations

DPS Finnish Diabetes Prevention Study
IGT Impaired glucose tolerance

Introduction

Lifestyle intervention has repeatedly and conclusively been shown to prevent or postpone the development of type 2 diabetes among high-risk individuals [1–5]. The Finnish Diabetes Prevention Study (DPS) was the first individually randomised, controlled clinical trial to show that a relative risk reduction of almost 60% can be achieved with intensive dietary and physical activity counselling [2]. Today several other trials among different populations and ethnic groups have confirmed this finding, with relative risk reduction ranging from 30% to 60% and absolute risk reduction of approximately 15–20% during active intervention [6].

We have previously shown that the effect of lifestyle intervention on diabetes incidence and lifestyle indicators was sustained for a median of 3 years after the end of active lifestyle intervention [7]. The finding was subsequently supported by long-term follow-up results from other major prevention trials from China and the USA [8, 9]. It is not known whether the sustained reduction in diabetes incidence results from permanent lifestyle changes or whether it is the legacy effect of improved glycaemia in the past.

The aim of this study was to investigate whether diabetes incidence was still reduced after a median of 13 years from the initiation of lifestyle intervention. In addition, we explored the changes in lifestyle (diet and physical activity) and clinical variables (body weight, fasting and 2 h plasma glucose after an OGTT) during and after the active intervention period and analysed the association between lifestyle changes and diabetes risk.

Methods

Study design The DPS was a randomised study aimed at preventing type 2 diabetes through intensive lifestyle intervention, carried out at five study clinics in Finland. The randomised trial started in November 1993, the recruitment period lasted until June 1998 and the intervention period lasted until the end of 2001. This article presents the follow-up results until the end of 2009, corresponding to a median time span of 13 years from baseline (Fig. 1). Detailed descriptions of the randomisation and study procedures have been published elsewhere [2, 10, 11]. Randomisation was stratified by clinic, sex and baseline 2 h plasma glucose to ensure a balanced study design. The study protocol was approved by the ethics committees of the National Public Health Institute in Helsinki, Finland (intervention phase), and of the North Ostrobothnia Hospital District (follow-up period). All study participants gave written informed consent at baseline and again at the beginning of the post-intervention follow-up.

Participants and interventions Participants (172 men and 350 women), aged 40–64 years at baseline, were overweight and had impaired glucose tolerance (IGT) based on the mean of two 75 g OGTTs. At baseline, mean (±SD) age was 55±7 years, mean BMI was 31.2±4.5 kg/m^2, mean fasting plasma glucose was 6.1±0.7 mmol/l and mean 2 h plasma glucose was 8.9±1.5 mmol/l.

The participants were randomised to two treatment groups: a control group that received standard advice at

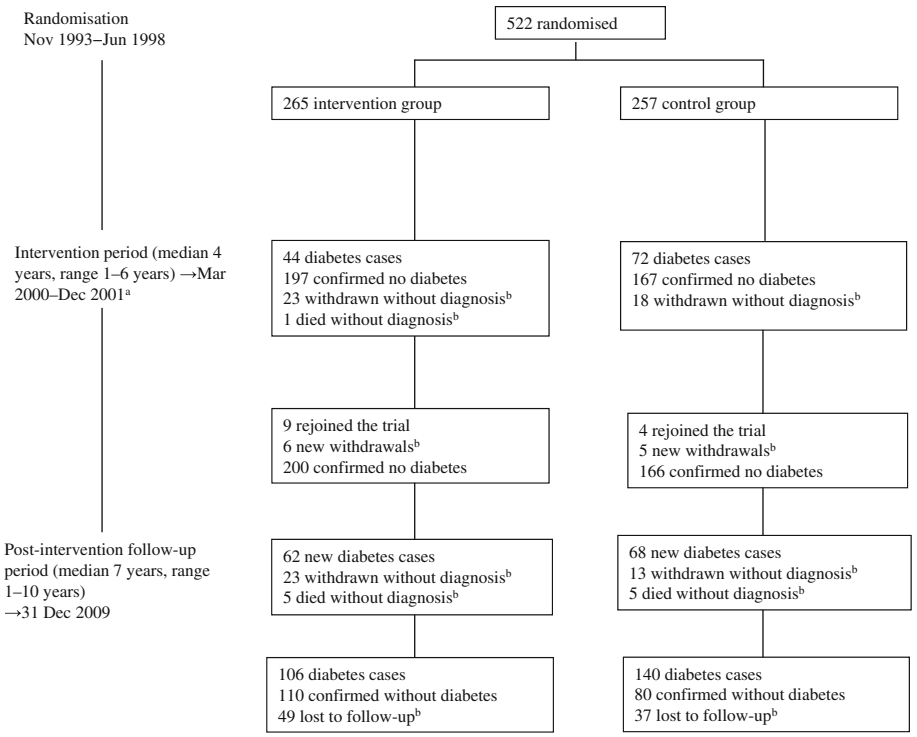

Fig. 1 Trial profile. [a]After the decision to end the intervention period, the intervention was continued until each participant's next scheduled annual clinic visit. The end date thus varied from March 2000 to December 2001. [b]Participants without a diabetes diagnosis who were lost to follow-up due to death or withdrawal were treated as censored observations in the analyses

baseline or an individualised lifestyle intervention group. Individualised lifestyle intervention included seven face-to-face counselling sessions with the study nutritionist during the first year and every 3 months thereafter, as well as voluntary free-of-charge supervised exercise sessions in the gym. The specific intervention goals were weight reduction (5% or more from baseline weight), dietary modification (energy proportion of total fat less than 30% and saturated fat less than 10% of total energy, dietary fibre intake 3.6 g/MJ (15 g/1,000 kcal)) or more and increased physical activity (4 h per week or more) [11].

All participants had an annual follow-up examination including clinical and laboratory measurements and an OGTT to diagnose incident diabetes, which was the endpoint of the study. Originally, the aim was to continue the intervention for 6 years for each participant unless diabetes was diagnosed beforehand. However, the intervention phase of the study was discontinued prematurely after a median follow-up of 4 years as suggested by the independent endpoint committee, based on interim endpoint analyses [2]. Thus, because of the lengthy recruitment period, the duration of intervention varied substantially between study participants (range 1–6 years).

After the intervention period, the post-intervention follow-up was initiated. Of the original 522 participants, 406 had not been assigned a verified diabetes diagnosis at the end of the intervention period. Of these, 35 had withdrawn from the study and five had died. Thus, 366 individuals (200 from the former intervention group and 166 from the former control group) participated in the post-intervention follow-up study at least once. During the post-intervention follow-up, 36 additional participants withdrew and ten died without a verified diabetes diagnosis (Fig. 1).

Clinical measurements Laboratory and clinical visits were completed annually (biennially after 5 years from the beginning of the post-intervention follow-up). During the intervention period, plasma glucose concentrations were determined locally according to standard guidelines.

During the follow-up phase, analyses were done in the central laboratory. Type 2 diabetes was defined according to 1985 WHO criteria (fasting plasma glucose ≥7.8 mmol/l or 2 h plasma glucose ≥11.1 mmol/l in two separate OGTTs) [12]. Once a diabetes diagnosis was verified, the participant had reached the study endpoint.

If a participant had been diagnosed with diabetes between the study visits in primary healthcare, we asked him/her to bring the results of the glucose tests to the study clinic and to attend a confirmatory OGTT. If the participant had been prescribed and was taking blood glucose-lowering drugs, he/she was asked to refrain from the drugs for 3 days before the confirmatory OGTT. If the diabetes diagnosis in primary healthcare had been based on the 1999 WHO criteria [13] and did not fulfil the 1985 criteria (that is, if fasting plasma glucose was ≥7.0 but <7.8 mmol/l), the diagnosis was not verified and the participant remained in the follow-up.

Body weight was measured in light indoor clothing to the nearest 100 g. Height was measured at baseline, without shoes, to the nearest 1 mm. BMI was calculated by dividing

the weight (kg) by the height (m) squared. Percentage body weight change was calculated using the baseline body weight as the denominator and the change in body weight from baseline as the numerator. The last-observation-carried-forward method was applied to all measurements for those participants who developed diabetes or who were lost to follow-up.

Lifestyle measurements Physical activity was assessed at each annual/biennial study visit using the Kuopio Ischemic Heart Disease Risk Factor Study questionnaire [14]. The participants recalled the frequency, typical duration and typical intensity of 15 common forms of physical activity during each month of the past year. Based on the reported intensity, the form of activity was categorised into low-intensity activity or moderate-to-vigorous activity. Low-intensity activity included gardening, picking berries, casual walking and bicycling at recreational intensity. Moderate-to-vigorous activity included brisk walking, jogging, skiing, swimming, rowing, forest work, gymnastics, resistance training, ball games, snow shovelling and heavy housework. The duration of total and moderate-to-vigorous activities was calculated in hours/week and is presented for baseline, the intervention period (mean over years 1–6), the early post-intervention follow-up (mean over years 1–3 after the end of the intervention) and the late post-intervention follow-up (mean over years 4–9 after the end of the intervention).

Dietary intake was assessed by 3-day food records at baseline and at each annual visit using a picture booklet of portion sizes of typical foods to help in estimating portion sizes. The food records were used to facilitate the dietary counselling of the intervention group participants during the active intervention period, e.g. to identify sources of saturated fat and fibre or estimate the amount of vegetables in the daily diet. For evaluation purposes, the mean daily nutrient intakes at baseline and years 1, 2 and 3 during the intervention period as well as at years 1 and 4 after the intervention were calculated with a dietary analysis program using the Finnish Food Composition Database (Fineli) developed at the National Public Health Institute, Helsinki, Finland [15]. Dietary data entry and analyses were completed only for these selected visits due to high resource demand of food record data entry. The habitual nutrient intakes are presented for baseline, for the intervention period (mean over years 1–3), for the early post-intervention follow-up (year 1 after the end of the intervention) and for the late post-intervention follow-up (year 4 after the end of the intervention).

Statistical analyses Kaplan–Meier survival curves were calculated to estimate the probability of remaining free of diabetes in the two groups. Participants who were lost to follow-up were treated as censored observations. The difference between the survival curves was tested using the logrank test. The Cox proportional hazards model was used to estimate the HR for the development of diabetes. The proportionality assumption of the model was assessed using graphical methods (i.e. the log-log plot).

To explore the effect of earlier lifestyle intervention on diabetes risk, similar analyses were completed for the post-intervention follow-up period, including only those participants who were free of diabetes at the beginning of the post-intervention follow-up. As the intervention and control groups were no longer balanced at the initiation of the follow-up due to a higher incidence of diabetes in the control group during the intervention period, these analyses were adjusted for sex, age, BMI and baseline 2 h plasma glucose.

Mean levels of nutrient intakes and physical activity (log-transformed values) were compared between the groups using repeated measures analysis of variance, adjusting for age and sex and the baseline level of the variable of interest. Further, analysis of covariance was used to examine these variables at the late post-intervention follow-up, adjusted for age, sex and the baseline level of the variable of interest.

To further explore the possible associations between lifestyle and incident diabetes during the post-intervention follow-up period we combined the intervention and control groups and compared the diet and physical activity (mean values during the post-intervention follow-up) of the participants who were diagnosed with diabetes with those of the participants who remained free of diabetes using the analysis of variance.

As the duration of the lifestyle counselling varied among the intervention group participants, an additional analysis was completed to study whether the duration of intervention had an effect on diabetes incidence. For these analyses the intervention group participants were divided into two groups according to the duration of the intervention: 0–4 years (n=124) and 5–6 years (n=97).

The number of predefined intervention goals achieved (success score ranging from 0 to 5) was calculated and the Cox model was used to analyse the relationship between the success score and the incidence of diabetes. The success score variable was included in the model as a categorical variable, using those who did not achieve any of the lifestyle goals as the reference category. In this analysis the intervention and control groups were pooled together.

All comparisons between groups were based on the intention-to-treat principle. The last-observation-carried-forward method was applied for those who were lost to follow-up, who did not take part in the specific annual visit or who had been diagnosed with diabetes before the visit. The number of carried values vs measured values increased

by the year (see electronic supplementary material [ESM] Table 1). We present data for body weight and glucose changes up to 10 years of follow-up where observed data were available in 83 (31%) and 65 (25%) persons in the intervention and control groups, respectively. In addition, sensitivity analyses based on multiple imputation of missing data were also performed. Also, the baseline characteristics of participants who dropped out during the study were compared with the completers.

The analyses were performed using the statistics package Stata (version 12.1; StataCorp, College Station, TX, USA) and SAS software (version 8.2; SAS Institute, Cary, NC, USA).

Results

Diabetes incidence The median total follow-up time (time from the initial randomisation visit to diabetes diagnosis, dropout or the end of 2009) was 9 years (range 0–16 years). Among the 522 participants, 246 incident cases of diabetes were diagnosed during the total follow-up period, 106 in the intensive intervention group and 140 in the control group. Kaplan–Meier survival curves to estimate the probability of remaining free of diabetes in the two groups are given in Fig. 2a. Incidence rates per 100 person-years were 4.5 (95% CI 3.8, 5.5) in the intervention group and 7.2 (95% CI 6.1, 8.5) in the control group; HR was 0.614 (95% CI 0.478, 0.789; $p<0.001$) and the absolute risk reduction was 19.4%. The number needed to treat to prevent one case of diabetes was thus 5.2. Altogether, 86 participants were lost to follow-up without a diabetes diagnosis: 49 in the intervention group and 37 in the control group. The baseline characteristics of the dropouts were similar between the groups (ESM Table 2).

During the post-intervention follow-up (median 7 years), 62 (of 200) and 68 (of 166) new diabetes cases were diagnosed among the former intervention and control group participants, respectively. The incidence rates were 4.9 (95% CI 3.8, 6.3) and 7.0 (95% CI 5.5, 8.9), respectively, with an adjusted HR of 0.672 (95% CI 0.477, 0.947; $p=0.023$), indicating that there was a 32% relative risk reduction and a 15% absolute risk reduction during the post-intervention follow-up period in favour of the former intervention group participants (Fig. 2b). Among those who developed diabetes, the median time to the onset of diabetes was 10 years in the control group (95% CI 8, 12 years) and 15 years in the intervention group (95% CI 13, 17 years).

Lifestyle Physical activity and dietary intakes in the two groups are presented in Table 1. The intervention group participants engaged more in moderate-to-vigorous physical activity in general ($p=0.0039$ for time × group interaction); however, at the end of the follow-up the difference between the groups was not statistically significant. Total hours of physical activity (low, moderate and vigorous) did not differ between the groups.

Intervention group participants made more dietary changes during the total follow-up. They reduced their total energy (p for time × group interaction <0.0001), energy proportion of fat ($p<0.0001$), saturated fat ($p<0.0001$), monounsaturated fat ($p<0.0001$), *trans*-fatty acids ($p=0.008$) and alcohol ($p=0.0333$), and increased their energy proportion of carbohydrates ($p=0.0012$) and protein ($p=0.0123$), and fibre density ($p<0.0001$). Intakes of total, saturated, monounsaturated and *trans*-fatty acids were lower and intake of protein and fibre density of diet were higher in the intervention group compared with the control group 4 years after discontinuation of the intervention.

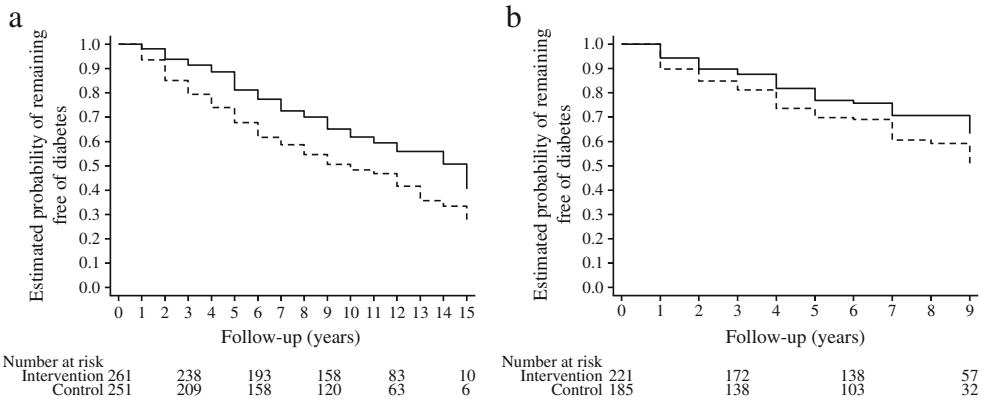

Fig. 2 Diabetes incidence in the DPS intervention (solid line) and control (dashed line) groups. (**a**) Total follow-up, with altogether 106 and 140 diabetes cases diagnosed in the intervention and control groups, respectively. Logrank test $p<0.001$, HR 0.61 (95% CI 0.48, 0.79; $p<0.001$). (**b**) Post-intervention follow-up, with 62 and 68 diabetes cases diagnosed in the intervention and control groups, respectively. Logrank test $p=0.031$, adjusted HR (adjusted for sex, age, 2 h plasma glucose and BMI at baseline) 0.67 (95% CI 0.48, 0.95; $p=0.023$)

Table 1 Physical activity and dietary intake of the DPS participants by intervention allocation

Variable	Study group	Baseline (year 0)	Intervention phase (years 1–6)[a]	Early follow-up (years 1–3 after intervention)[b]	Late follow-up (years 4–9 after intervention)[c]	p (time × group)	p[d]
Physical activity[e]							
Total activity (h/week)	Intervention	5.7 (3.2–9.1)	6.6 (4.4–9.6)	6.3 (3.8–9.9)	6.2 (3.5–9.5)	0.40[f]	0.54[f]
	Control	5.5 (3.0–9.7)	6.1 (3.3–9.8)	5.9 (3.1–9.4)	5.7 (3.3–9.3)		
Moderate to vigorous activity (h/week)	Intervention	1.8 (0.6–3.8)	3.0 (1.6–4.7)	3.5 (1.5–5.5)	3.1 (1.5–5.1)	0.0039[f]	0.15[f]
	Control	1.6 (0.4–4.2)	2.3 (1.0–4.1)	2.8 (1.3–4.8)	2.8 (1.4–5.4)		
Dietary intake[g]							
Energy (kJ)	Intervention	7,415±2,177	6,506±1,620	6,624±1,704	6,778±1,746	<0.0001	0.06
	Control	7,302±2,206	6,942±1,863	6,875±1,788	6,975±1,821		
Fat (E%)	Intervention	36.0±6.7	32.1±5.2	31.9±5.7	32.7±6.3	<0.0001	0.0009
	Control	37.1±6.5	34.6±4.9	33.9±6.1	34.7±5.9		
Saturated fat (E%)	Intervention	16.2±4.0	13.3±3.1	11.8±3.5	12.2±3.7	<0.0001	<0.0001
	Control	17.0±4.3	15.5±3.2	13.7±3.7	14.0±3.5		
Saturated fatty acids (g)	Intervention	27±12	20±8	19±8	20±9	<0.0001	<0.0001
	Control	29±12	25±10	23±10	24±10		
Monounsaturated fatty acids (g)	Intervention	22±9	18±6	18±7	19±7	<0.0001	0.0128
	Control	22±9	20±7	20±8	20±8		
trans-Fatty acids (g)	Intervention	1.1±0.8	0.9±0.5	0.7±0.4	0.8±0.4	0.008	0.0036
	Control	1.3±1.0	1.1±0.7	0.9±0.6	1.0±0.6		
Polyunsaturated fatty acids (g)	Intervention	10±4	8±3	9±3	9±4	0.22	0.71
	Control	10±5	9±3	9±4	9±4		
n-3 Polyunsaturated fatty acids (g)	Intervention	1.6±0.8	1.5±0.6	2.0±1.0	2.0±1.2	0.29	0.84
	Control	1.5±0.8	1.5±0.7	1.9±1.0	2.0±1.3		
Carbohydrates (E%)	Intervention	43.6±7.5	47.0±6.4	47.6±6.9	46.9±7.3	0.0012	0.08
	Control	43.2±6.7	45.1±6.0	46.2±6.8	45.7±6.9		
Carbohydrates (g)	Intervention	190±57	181±46	185±49	186±51	0.14	0.37
	Control	185±54	185±50	185±45	187±51		
Protein (E%)	Intervention	17.6±3.4	19.1±2.8	18.7±3.1	18.8±3.2	0.0123	0.0019
	Control	17.6±3.4	18.4±2.9	18.3±3.1	17.9±3.1		
Alcohol (E%)	Intervention	2.8±5.1	1.7±3.4	1.8±3.7	1.6±3.5	0.0333	0.34
	Control	2.1±4.1	1.9±3.2	1.5±3.0	1.7±3.3		
Total fibre (g)	Intervention	20±7	21±6	21±7	21±8	0.11	0.10
	Control	20±8	20±6	20±6	20±7		
Fibre, unsoluble (g)	Intervention	14±5	15±4	15±5	15±5	0.11	0.14
	Control	14±6	14±4	14±5	14±5		
Total fibre (g/MJ)	Intervention	2.8±1.0	3.4±0.9	3.2±1.0	3.2±0.9	<0.0001	0.0028
	Control	2.8±0.9	3.0±0.9	3.0±0.9	3.0±1.0		

Last-observation-carried-forward method was applied

[a] Mean of available data in years 1–6 (physical activity) or years 1–3 (dietary intake)

[b] Mean of available data in years 1–3 (physical activity) or year 1 (dietary intake) after the intervention

[c] Mean of available data in years 4–9 (physical activity) or year 4 (dietary intake) after the intervention

[d] p value for the difference between the intervention and control groups at years 4–9 after the intervention, adjusted for baseline and sex

[e] Median (interquartile range)

[f] Statistical tests calculated using log-transformed values

[g] Mean±SD

E%, proportion of energy

Compared with participants without diabetes, those who were diagnosed with diabetes during the post-intervention follow-up consumed, after adjusting for sex, age and body weight, a diet with lower carbohydrate (energy proportion 46.3% vs 47.7%, p=0.0477; 186 vs 197 g/day, p=0.0349) and higher total fat content (energy proportion 33.9% vs 32.4%, p=0.019). No differences were observed in other dietary variables (total energy, protein, alcohol, fibre, fatty acid categories) or physical activity (data not shown).

Body weight After the first year of the intervention, mean weight reduction was 5% in the intervention group and 1% in the control group (Fig. 3). Body weight increased gradually in the course of the follow-up in both groups; however, a statistically significant difference between the study groups prevailed (p=0.006 at year 10). In sensitivity analyses based on multiple imputation of missing data, the corresponding p value for the difference between groups was 0.021.

Glucose values After the initial decrease in fasting and 2 h plasma glucose in the intervention group during the first year, the rising trends in both fasting and 2 h plasma glucose values were similar in the two groups (Fig. 4). Similar results were observed when analyses were based on multiple imputation of missing data. Among the intervention group participants, fasting plasma glucose passed the baseline level between years 3 and 4, and 2 h plasma glucose between years 4 and 5.

Intervention and diabetes incidence The analyses of the association between the success score (the number of intervention goals achieved at year 3) and the risk of diabetes during the total follow-up are presented in Fig. 5. Taking those who did not achieve any of the intervention goals (success score=0) as the reference group, the adjusted HR

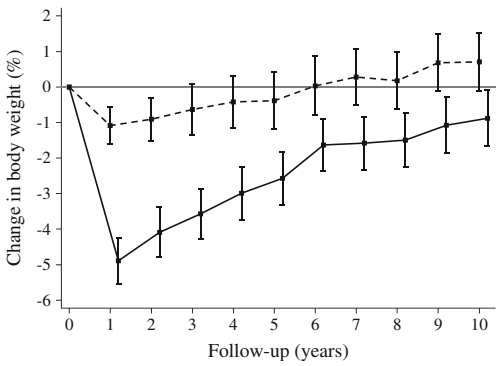

Fig. 3 Changes in body weight in the DPS intervention (solid line) and control (dashed line) groups during the total follow-up period. The last-observation-carried-forward method was applied to those who dropped out or who were diagnosed with diabetes before the end of follow-up

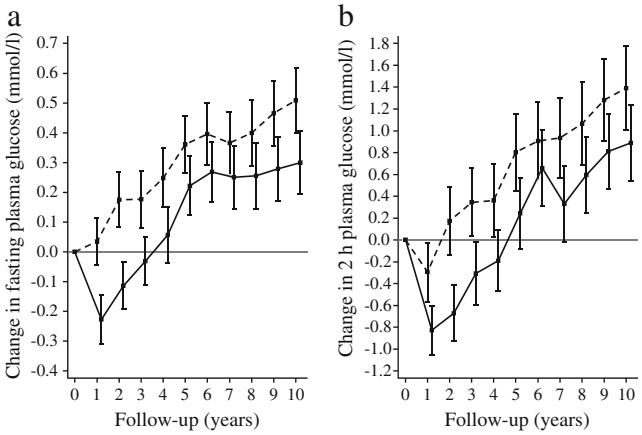

Fig. 4 Changes in fasting plasma glucose (**a**) and 2 h plasma glucose (**b**) concentrations in the DPS intervention (solid line) and control (dashed line) groups during the total follow-up period. The last-observation-carried-forward method was applied for those who dropped out or who were diagnosed with diabetes before the end of follow-up

(adjusted for age, sex, baseline BMI and 2 h plasma glucose) were as follows: 0.86 (95% CI 0.60, 1.23; p=0.407) for success score 1; 0.67 (95% CI 0.45, 1.00; p=0.051) for success score 2; 0.61 (95% CI 0.38, 0.98; p=0.040) for success score 3; 0.34 (95% CI 0.18, 0.66; p=0.001) for success score 4; and 0.20 (95% CI 0.07, 0.56; p=0.002) for success score 5.

The duration of the intervention did not influence the magnitude of the risk reduction. Compared with shorter intervention (0–4 years), the HR for diabetes in participants with longer intervention (5–6 years) was 1.17 (95% CI 0.62, 2.22; p=0.63) during the post-intervention follow-up.

Discussion

Our findings from the DPS follow-up study suggest that lifestyle intervention lasting for a median of 4 years can result in long-term protection against type 2 diabetes. We

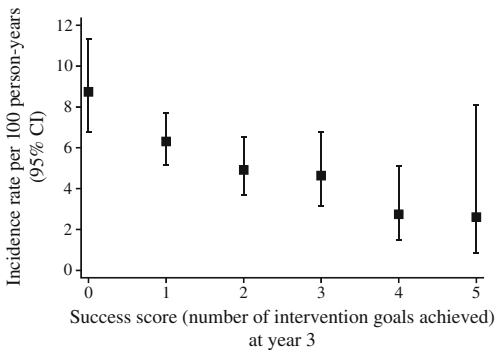

Fig. 5 Diabetes incidence rate by success score (number of intervention goals achieved at year 3 of the intervention phase) during the total follow-up period. Intervention and control groups were combined

have previously reported the results after 7 years of follow-up, until the end of 2004, which showed a 43% reduction in the relative risk [7]. By 2009, the relative risk reduction was 38% during a median follow-up of 9 years and a median time span of 13 years from baseline. These findings are consistent with the China Da Qing Diabetes Prevention Study [8], which had a 43% lower diabetes incidence over 20 years, and with the US Diabetes Prevention Program Outcomes Study [9], which had a 34% lower diabetes incidence over 10 years in the active intervention group.

Importantly, when our analysis was repeated for the follow-up period only, diabetes incidence was still 32% lower among the participants of the former intervention group, compared with those of the control group. An interesting question is whether this carry-over risk reduction can be attributed to the legacy effect of earlier improvement in glycaemia or to sustained lifestyle change. The studies from China and the USA cannot address this question because comprehensive lifestyle data were not collected. Furthermore, in the US Diabetes Prevention Program Outcomes Study lifestyle counselling was eventually offered to all participants regardless of their former treatment group (lifestyle, metformin or placebo), which may be considered ethically justified but detrimental to the original study design.

Based on our results, both past improvement in glycaemia and sustained improvement in lifestyle might play a role. Fasting and 2 h plasma glucose values decreased during the first year in the intervention group, followed by a gradual and more or less parallel increase in both groups during the rest of the follow-up.

On the other hand, the participants in the former intervention group were, 4 years after the end of active intervention, still consuming a diet with lower total fat, saturated fat and *trans*-fat, and higher fibre density, in accordance with the intervention goals. Their diet was also marginally higher in protein and lower in monounsaturated fat, although this was not the aim of the counselling. Mean body weight in the former intervention group remained slightly lower compared with the control group and was 1% below baseline weight 10 years later. These modest changes combined may contribute to the reduced diabetes risk, as shown by the success score analysis demonstrating decreasing diabetes rate by increasing number of goals achieved. We have shown before that these changes were associated with reduced diabetes risk during the active intervention period [16, 17]. The potency of small behavioural changes is a positive message to the public, as small changes are easier to accept, achieve and sustain.

Discussion continues about the optimal lifestyle intervention, especially the role of the macronutrient composition of a diet to prevent diabetes [18]. Our study was not designed to compare different diets but rather to test the feasibility and efficacy of a diet that was considered the healthy choice—even the control group participants were advised on the same type of lifestyle, but without individualised counselling or continuity. Nevertheless, the results of the success score analysis, showing that better adherence to the intervention goals was associated with a larger risk reduction, support the validity of the chosen approach and the recommendation to reduce the intake of foods with a high saturated fat content and to increase fibre-rich carbohydrate sources such as wholegrain cereal, vegetables and fruit to reduce the risk of type 2 diabetes. The observation that participants who developed diabetes consumed a diet with lower carbohydrate and higher total fat content further supports this conclusion.

The participants in the DPS had IGT based on two OGTTs at baseline; thus they had a very high risk of progressing to diabetes. The cumulative incidence of diabetes was 64% in the control group and 44% in the intervention group over 13 years. Based on the parallel glucose trends shown among both groups it is evident that the observed risk reduction implies postponing the disease rather than preventing it altogether. Nevertheless, lifestyle intervention postponed the deterioration of glycaemia from IGT to overt diabetes by 5 years. This could have an important public health impact, as population ageing is one of the most important drivers for the increasing number of people with diabetes [19], and this could be counteracted by postponing the disease to later in life. Blood glucose tends to increase with age and keeping it longer under the threshold of hyperglycaemic complications might be beneficial. This raises the question whether preventive interventions should actually be started earlier, before blood glucose has started to rise.

In the DPS, the intervention included an intensive first year, followed by a maintenance period, with a total duration of intervention of up to 6 years, depending on the time of inclusion. Longer intervention (lasting for 5–6 years) did not seem to be more effective than shorter intervention (1–4 years). This is good news, as in the real-life setting, active intervention that lasts for many years is not feasible. Unfortunately, our data do not allow us to make any conclusions about whether a 1 year intervention or even less would show similar long-term results.

The value of drugs to prevent or postpone diabetes among high-risk individuals has also been widely investigated [20]. Besides possible drug-related side effects, the disadvantage of drugs compared with lifestyle change is that several different types of drugs are needed to treat the different components of the metabolic syndrome commonly associated with high diabetes risk. These components (dyslipidaemia, hyperglycaemia, elevated blood pressure), however, can be treated simultaneously with lifestyle intervention, as has been shown also among the DPS participants [21].

Our study has both strengths and a few weaknesses. The randomised trial setting, comprehensive data collection, relatively low attrition, as well as diabetes diagnosis based on repeated OGTTs and the uniform diagnostic criteria throughout the study, allow us to make solid conclusions about the endpoints. However, the changing diagnostic criteria [13] of diabetes during the study duration were a challenge to the long-term trial follow-up. As we decided to stick to the 'old' criteria [12] throughout the study we had some participants diagnosed with diabetes by their own physician but not according to our study criteria, and this probably caused some participants to withdraw from the follow-up without a verified diabetes diagnosis. Methodological issues include reliance on self-report in diet and physical activity, without objective measures. The intervention group participants might, consciously or unconsciously, adjust their answers according to the lifestyle goals. On the other hand, the validity of the lifestyle data is supported by the observed association between the success score and diabetes incidence. Finally, the post-intervention analyses were not considered in the original power calculations and should thus be interpreted with caution.

In summary, lifestyle intervention aiming at weight reduction, a healthy diet and increased physical activity in high-risk individuals has a long-lasting effect in the prevention of type 2 diabetes. With a relatively short active lifestyle intervention, time free of diabetes can be extended by approximately 5 years. This may be due to sustained lifestyle changes as well as to the legacy effect of former improvements in glycaemia. Such interventions could play an important role in preventing chronic disease during ageing. This finding emphasises the importance of early, comprehensive lifestyle change as the primary target of a type 2 diabetes prevention strategy.

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Contribution statement All authors contributed to the study design and reviewed the manuscript critically and approved the final version. MU and JT were the principal investigators of the DPS study. MU, JT, JE and JL contributed to the development of the intervention. JL, PIP and SA contributed to the intervention delivery and the acquisition of data. JE, SKK, PIP and SA led the study centres and supervised the study. Statistical analyses, interpretation of data and drafting of the manuscript were completed by MP and JL.

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