BMJ open
accessible medical research

Increase in physical activity and cardiometabolic risk profile change during lifestyle intervention in primary healthcare: 1-year follow-up study among individuals at high risk for type 2 diabetes

Urho M Kujala,[1] Jari Jokelainen,[2,3] Heikki Oksa,[4] Timo Saaristo,[4,5] Nina Rautio,[4] Leena Moilanen,[6] Eeva Korpi-Hyövälti,[7] Juha Saltevo,[8] Mauno Vanhala,[9,10,11] Leo Niskanen,[8,12] Markku Peltonen,[13] Jaakko Tuomilehto,[14] Matti Uusitupa,[15,16] Sirkka Keinänen-Kiukaannemi[2,3,17]

To cite: Kujala UM, Jokelainen J, Oksa H, *et al*. Increase in physical activity and cardiometabolic risk profile change during lifestyle intervention in primary healthcare: 1-year follow-up study among individuals at high risk for type 2 diabetes. *BMJ Open* 2011;**1**:e000292. doi:10.1136/bmjopen-2011-000292

► Prepublication history for this paper is available online. To view these files please visit the journal online (http://bmjopen.bmj.com).

Received 9 August 2011
Accepted 31 October 2011

For numbered affiliations see end of article.

Correspondence to
Professor Urho M Kujala; urho.m.kujala@jyu.fi

ABSTRACT

Objectives: To investigate the association between increase in physical activity and changes in cardiometabolic risk factors during a lifestyle intervention programme in routine clinical settings.

Design: Prospective follow-up.

Setting: 400 primary healthcare centres and occupational healthcare outpatient clinics in Finland.

Participants: Individuals at high risk for type 2 diabetes identified in the implementation project of the national diabetes prevention programme (FIN-D2D) and participating in baseline and 1-year follow-up visits. Final study group comprised the 1871 non-diabetic participants who responded at follow-up visit to a question on stability versus increase of physical activity.

Interventions: Lifestyle intervention.

Primary outcome measures: Cardiometabolic risk factors (body composition, blood pressure and those measured from fasting venous blood samples) measured at baseline and follow-up visits.

Results: Of the participants, 310 (16.6% of all responders) reported at follow-up having clearly increased their physical activity during the past year, while 1380 (73.8%) had been unable to increase their physical activity. Those who increased their activity decreased their weight by 3.6 kg (95% CI 2.9 to 4.3, age and sex adjusted, $p<0.001$) and waist circumference by 3.6 cm (95% CI 2.9 to 4.3, $p<0.001$) more than those who did not increase their activity. Similarly, those who increased their physical activity had greater reductions in total cholesterol (group difference in reduction 0.17 mmol/l, 95% CI 0.06 to 0.28, $p=0.002$), low-density lipoprotein cholesterol (0.16 mmol/l, 95% CI 0.06 to 0.26, $p=0.001$), low-density lipoprotein/high-density lipoprotein ratio (0.17, 95% CI 0.08 to 0.25, $p<0.001$) as well as fasting glucose (0.09 mmol/l, 95% CI 0.03 to 0.15, $p=0.004$) and 2 h glucose levels (0.36 mmol/l, 95% CI 0.17 to 0.55, $p=0.023$) than those who did not increase their physical activity.

Conclusion: Increasing physical activity seems to be an important feature of cardiometabolic risk reduction among individuals at high risk for type 2 diabetes participating in preventive interventions in routine clinical settings.

ARTICLE SUMMARY

Article focus

- There is evidence from randomised controlled trials that supervised exercise interventions improve cardiometabolic risk factor levels.
- It is not known how knowledge from intensive interventions of randomised clinical trials can be applied in various real-life clinical settings with limited resources.
- In this paper, we report the results of an analysis of physical activity changes and their association to cardiometabolic risk factors among individual at high risk for type 2 diabetes and participating in preventive lifestyle intervention in routine clinical settings of primary healthcare.

INTRODUCTION

Randomised clinical trials have shown that type 2 diabetes can be prevented or at least postponed by lifestyle changes including changes in diet and physical activity.[1 2] Prospective follow-up studies,[3 4] one by clinic randomised controlled trial[5] and one co-twin control study,[6] suggest that physical activity has an independent role in the prevention of

ARTICLE SUMMARY

Key messages

- Less than one-fifth of the participants reported at '1-year' follow-up having clearly increased their physical activity during the past year.
- Those who increased their activity improved clearly their cardiometabolic risk profile including reductions of waist circumference and fasting low-density lipoprotein cholesterol and glucose levels, which result persisted after the adjustment for dietary change.
- Increasing physical activity seems to be an important feature of cardiometabolic risk reduction among individuals at high risk for type 2 diabetes participating in preventive interventions in routine clinical settings.

Strengths and limitations

- FIN-D2D is the first national effort to implement the prevention of diabetes in a primary healthcare setting.
- Follow-up data on the changes in physical activity are available from a subgroup of participants.
- The limitations of this report include that physical activity changes are documented by a questionnaire.

type 2 diabetes. Also, a post hoc analysis of the Finnish Diabetes Prevention Study participants suggested that increasing physical activity may substantially reduce the incidence of type 2 diabetes in high-risk individuals.[7] In addition, there is evidence that high leisure-time physical activity (LTPA) or high physical fitness is associated with less total and visceral fat and with a reduced prevalence of high cardiometabolic risk factor levels, coronary heart disease as well as reduced mortality.[8] There is accumulating evidence from hundreds of randomised controlled trials that physical exercise may help in improving health via different mechanisms including reduced body fat per cent and improvement in many cardiometabolic risk factor levels measured from blood.[9]

Usually lifestyle interventions for patients at high risk of diabetes, delivered by a variety of healthcare providers in routine clinical settings, are feasible but appear to be of less clinical benefit than structured intensive trials.[10] We wanted to know whether an increase in physical activity is associated with beneficial changes in other cardiometabolic risk factors in an intervention carried out in real-life clinical settings with limited resources. In this paper, we report the results of a post hoc analysis of physical activity changes and their association to cardiometabolic risk factors in the high-risk cohort of the National Program for the Prevention of Type 2 Diabetes, implemented through the FIN-D2D programme in five hospital districts in Finland from 2003 to 2008 covering a population of 1.5 million.[11–13]

METHODS

Subjects

The primary strategy of the FIN-D2D was a 'high-risk strategy' aiming at preventing diabetes and reducing cardiovascular risk factor levels among high-risk individuals in daily routines in healthcare centres and occupational healthcare outpatient clinics.[12] The aim of the 'high-risk strategy' was first to identify individuals at elevated risk of developing type 2 diabetes and to support their lifestyle changes required to reduce their future risk. Altogether, 400 primary healthcare centres or occupational healthcare clinics were involved in the programme. To identify high-risk individuals for type 2 diabetes, the modified Finnish Diabetes Risk Score (FINDRISC; scoring ≥15)[14 15] was used. The FINDRISC test forms were available in primary healthcare and occupational healthcare centres, in public places and events, and in the internet.[13 15] High-risk individuals were also identified by the history of impaired fasting glucose, impaired glucose tolerance, cardiovascular events and gestational diabetes. After identification, consenting high-risk individuals for type 2 diabetes attended health check-ups conducted in the primary healthcare units as a part of the normal routine; therefore, no informed consent was used, but individuals received written information on the FIN-D2D. The Ministry of Social Affairs and Health in Finland gave the permission to collect the data from healthcare units for evaluation purposes to the National Public Health Institute.

Of the high-risk cohort,[13] those individuals (age range 18–87 years) who were non-diabetic at baseline (no previous diabetes diagnosis or screen-detected diabetes) had '1-year' follow-up data and did not get diabetes during follow-up were included in the target group of this study (figure 1). Those individuals who got diabetes (6.7%; 157 of 2355 individuals) were excluded from this study as they are usually a focus of additional intensified lifestyle interventions. Finally, those non-diabetic individuals who responded to the structured question on the stability or increase of physical activity at follow-up (N=1871) formed the final study group of this report. Follow-up visits were defined as visits occurring after 9–18 months of baseline visits (mean 14 months). Visits between 17 January 2004 and 28 August 2007 were considered as baseline visits, and '1-year' follow-up visits were between 17 January 2005 and 12 June 2008.[13]

Questions on physical activity and diet

LTPA was assessed at baseline and at follow-up by a self-administered questionnaire, which included structured questions about general physical activity level (four alternatives) (appendix question 1), frequency of at least moderate intensity LTPA (appendix question 2), duration of everyday activities (appendix question 3) and a question on changes in physical activity during the past year (appendix question 4) as well as a question on work-related physical activity (appendix question 5). We primarily classified our participants according to responses to the structured question 'Have you increased your physical activity/exercise training during the past year?' (the number of responders at follow-up being 1871) which included five options (appendix 1). Participants at follow-up choosing one of the three first

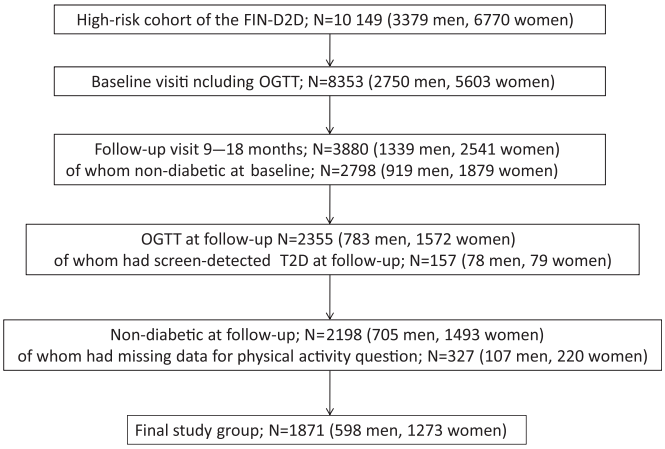

Figure 1 Flow chart of the study participants. OGTT, oral glucose tolerance test; T2D, type 2 diabetes.

options (No, and I do not intend to increase it; No, but I intend to increase it in the near future or I have tried to increase it) were classified as those who had 'No increase in physical activity' (n=1380). Those who chose option four (I have clearly increased it) were classified as those who 'Increased physical activity' (n=310). Others (n=181) were the group who chose the fifth alternative (I have already previously been highly physically active).

Changes in the use of amount and quality of fat and vegetables, fruits and berries during the past year were asked for in the questionnaire at follow-up (see appendix questions 6–8). Responses to these questions were classified into two classes. Participants who chose one of the three first options or the last option were classified as individuals who had 'no change' and those who chose option four were classified as those who 'changed diet'. If the participant had a change in one of these variables, he/she was considered as having dietary change.

Measurements at baseline and follow-up

The measurements were instructed to be carried out in the primary healthcare setting according to written working instructions. Height was measured to the nearest centimetre. Weight was measured to the nearest 0.1 kg in light clothing. Body mass index (BMI) was calculated as weight (kilograms) divided by height^2 (square metres). Waist circumference was instructed to be measured to the nearest centimetre on bare skin midway between the lowest ribs and the iliac crest during expiration. Blood pressure (BP) was instructed to be measured according to the current Finnish guidelines (two times at 1 min interval from the right arm of the sitting subject after 5 min rest using a standard mercury sphygmomanometer or electronic BP measurement device with the recommended cuff size, the mean of two measurements was used in the analysis).

For lipid and lipoprotein determinations, fasting venous blood samples were drawn. Serum levels of total cholesterol, high-density lipoprotein (HDL) cholesterol and triglycerides were determined in the local healthcare centre or occupational healthcare centre laboratories using enzymatic methods. Identical examinations were performed at baseline and at 1-year visit. Low-density lipoprotein (LDL) cholesterol was calculated according to the Friedewald's formula.[16]

The examination also included an oral glucose tolerance test (OGTT) with a glucose load of 75 g and fasting and 2 h plasma samples.[17] The subjects received written instructions on preparation for the test. The test started in the morning after overnight fasting and 20% of the tests used capillary and 80% venous plasma samples at baseline and 15% and 85%, respectively, at 1-year follow-up. Glucose tolerance was classified according to WHO 1999 criteria.[17] Individuals reporting at baseline that they had diabetes were not included in the OGTT, and those with fasting venous or capillary plasma glucose level >7.0 mmol/l or 2 h venous plasma glucose >11.1 mmol/l or 2 h capillary plasma glucose >12.2 mmol/l were classified as having diabetes.

All laboratories participated in the national External Quality Assessment Schemes organised by the Labquality (http://www.labquality.fi), and the measurements met the national primary healthcare standards.

Intervention visits

Intervention visits were either individual counselling visits or group sessions, at which the intervention visit form was filled. Counselling based on the intervention experiences in the Diabetes Prevention Study[18] and applying different stages of change in behaviour was recommended. The focus of the visits was weight, meal frequency, fat intake, quality of fat, use of salt, fibre intake, alcohol consumption, exercise or smoking taking into account the preferences of the individuals. Group sessions varied from weight maintenance groups to exercise groups and lectures on diabetes and lifestyle changes. The frequency of intervention visits varied between health centres depending on local circumstances and resources, and the total number of intervention visits was recorded. Typical physical activities recommended included brisk walking, cross-country skiing, bicycling and swimming. More comprehensive guidelines of the interventions are freely available in web.[19]

Statistical methods

Summary statistics are presented as frequencies and percentages for categorical variables and as means (SD) for continuous variables. Pearson's χ^2 test was used to compare categorical variables between analysis groups and Student t test was used for continuous variables. Appropriate 95% CIs were calculated for parameter estimates. A paired Student t test or Bhapkar's test was used to examine the changes in self-reported physical activity at the baseline and 1-year follow-up visits.

Mixed models of repeated analyses were used to analyse changes during follow-up in risk factor levels according to self-reported changes in physical activity during follow-up. We first adjusted the results for sex,

age at baseline and dietary change during follow-up. As physical activity is known to reduce intra-abdominal fat, we also analysed the results adjusted for sex and age at baseline as well as change in weight or waist circumference. Additionally, we adjusted the results for the number of intervention visits. Statistical analyses and data management were performed using SAS (V.9.2) for Windows. All significance tests were two tailed, and values of $p<0.05$ were considered statistically significant.

RESULTS

Baseline characteristics of the participants show that the mean BMI was over 30 and mean waist circumference was over 100 cm among both those who increased and did not increase physical activity during follow-up. Mean levels of some of the serum cardiometabolic risk factor levels, such as total cholesterol and LDL cholesterol, were near the upper limit of the recommended range (for details, see table 1).

Of the participants, 1871 responded at follow-up to the question on increase in physical activity (see appendix question 4) of whom 310 (16.6% of all responders and 18.3% of those who did not report having been highly physically active already previously) reported having clearly increased their physical activity during the past year (table 2), while 1380 were unable to increase their physical activity. Those who reported that they had increased physical activity during follow-up have also increased their physical activity level from baseline to follow-up according to the questions on general physical activity level ($p<0.001$) and the frequency of at least moderate intensity LTPA ($p<0.001$), which was not seen among those reporting no increase in physical activity (table 2). Of those participants who increased physical activity during follow-up, 73.2% also changed their diet and 26.8% did not change their diet. Corresponding figures for those who did not increase physical activity were 46.6% and 53.4%, respectively ($p<0.001$).

Those who increased their activity decreased their weight by 3.6 kg (95% CI 2.9 to 4.3, sex and age adjusted for group difference, $p<0.001$), BMI by 1.27 kg/m^2 (95% CI 1.04 to 1.51, $p<0.001$) and waist circumference by 3.6 cm (95% CI 2.9 to 4.3, $p<0.001$) more than those who did not increase their activity (table 3). Similarly, compared with those who did not increase their physical activity, greater reductions during follow-up were seen among those who increased their physical activity in BP values, the group difference in the reduction of systolic BP being 2.0 mm Hg (95% CI 0.3 to 3.8, $p=0.027$) and that of diastolic BP being 1.7 mm Hg (95% CI 0.6 to 2.7, $p=0.002$). Similarly, those who increased their physical activity had higher reductions in total cholesterol (group difference in the reduction 0.17 mmol/l, 95% CI 0.06 to 0.28, $p=0.002$), LDL cholesterol (0.16 mmol/l, 95% CI 0.06 to 0.26, $p=0.001$), LDL/HDL ratio (0.17, 95% CI 0.08 to 0.25, $p<0.001$) as well as fasting glucose (0.09 mmol/l, 95% CI 0.03 to 0.15, $p=0.004$) and 2 h glucose levels (0.36 mmol/l, 95% CI 0.17 to 0.55, $p=0.023$) at OGTT than those who did not increase their physical activity (table 3). HDL cholesterol increased more (by 0.05 mmol/l, 95% CI 0.01 to 0.08, $p=0.014$) among those who increased their physical activity compared with those who did not do so. As these results were similar for men and women (p for gender × group interaction >0.5 for all variables), the results are not shown separately.

Most of the differences between those who increased and did not increase physical activity persisted after adjustment for the self-reported dietary change (table 3). After adjustment for sex and age at baseline as well as change in waist circumference, those who increased their physical activity during follow-up decreased their weight ($p\leq0.001$, adjusted for sex, age and change in waist circumference), diastolic BP ($p=0.015$), total cholesterol ($p=0.010$) and LDL cholesterol ($p=0.005$) more than those who did not increase their physical activity.

Table 1 Baseline characteristics of the participants according to self-reported changes in physical activity at follow-up

	No increase in physical activity (N=1380)		Increase in physical activity (N=310)		
Variable	**n**	**Mean (SD)**	**n**	**Mean (SD)**	**p Value**
Age, years	1380	54.4 (10.6)	310	51.4 (10.3)	<0.001
Weight, kg	1380	88.4 (16.7)	310	88.4 (15.6)	0.974
BMI, kg/m^2	1374	31.5 (5.2)	310	32.1 (5.1)	0.062
Waist, cm	1346	102.2 (12.6)	301	102.2 (11.8)	0.999
Systolic BP, mm Hg	1375	138.1 (16.7)	310	139.3 (17.4)	0.241
Diastolic BP, mm Hg	1375	85.6 (9.2)	310	87.0 (9.2)	0.016
Total cholesterol, mmol/l	1313	5.2 (1.0)	293	5.3 (1.0)	0.026
HDL cholesterol, mmol/l	1309	1.4 (0.4)	292	1.4 (0.4)	0.832
LDL cholesterol, mmol/l	1298	3.0 (0.9)	290	3.2 (0.9)	0.004
LDL/HDL ratio	1297	2.3 (0.9)	290	2.4 (0.9)	0.054
Triglycerides, mmol/l	1309	1.6 (0.9)	292	1.5 (0.8)	0.718
Fasting glucose, mmol/l	1380	5.7 (0.6)	310	5.8 (0.6)	0.652
2 h glucose, mmol/l	1380	6.8 (1.8)	310	7.0 (1.8)	0.291

BMI, body mass index; BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

Table 2 Self-reported physical activity at baseline and follow-up according to self-reported changes in physical activity at follow-up

	Self-reported changes in physical activity (appendix question 4)					
	No increase in physical activity			Increase in physical activity		
	Baseline	Follow-up	p Value	Baseline	Follow-up	p Value
General physical activity level (appendix question 1), n (%)						
1	366 (29.9)	386 (29.0)	0.625*	48 (17.0)	15 (4.9)	<0.001*
2	681 (55.7)	741 (55.7)		170 (60.3)	169 (55.4)	
3	176 (14.4)	203 (15.2)		64 (22.7)	120 (39.3)	
4	0	1 (0.1)		0	1 (0.3)	
Frequency of at least moderate intensity LTPA (appendix question 2), mean (SD)	2.3 (2.4)	2.5 (4.3)	0.094†	2.7 (1.8)	4.2 (5.2)	<0.001†
Everyday activities (appendix question 3), n (%)						
1	194 (16.3)	180 (15.2)	0.718*	33 (12.0)	21 (7.6)	0.034*
2	421 (35.4)	429 (36.1)		92 (33.3)	87 (31.5)	
3	263 (22.1)	270 (22.7)		60 (21.7)	59 (21.4)	
4	109 (9.2)	97 (8.2)		33 (12.0)	31 (11.2)	
5	201 (16.9)	212 (17.8)		58 (21.0)	78 (28.3)	
Work-related physical activity (appendix question 5), n (%)						
1	645 (58.0)	664 (59.7)	0.234*	148 (56.5)	139 (53.1)	0.225*
2	217 (19.5)	208 (18.7)		40 (15.3)	51 (19.5)	
3	198 (17.8)	197 (17.7)		63 (24.1)	59 (22.5)	
4	53 (4.8)	44 (4.0)		11 (4.2)	13 (5.0)	

*Differences in frequency distributions (cross-tab marginal homogeneity) were checked for significance using Bhapkar's test for multiple categories.
†By paired t test.
LTPA, leisure-time physical activity.

The total number of intervention visits was higher among those who increased their physical activity than those who did not increase it (mean 3.7 vs 2.8, p<0.0001 by Kruskall–Wallis test). After adjustment for sex and age at baseline as well as the number of intervention visits, the group differences in the changes of risk factors persisted either statistically significant or borderline significant (p<0.1, see table 3).

DISCUSSION

Principal finding

Our study shows that increases in LTPA among individuals at high risk for type 2 diabetes and participating in lifestyle interventions in routine clinical settings of the primary healthcare system were associated with reductions in weight, waist, BP, serum lipid risk factor levels and plasma glucose levels. This finding is in line with the findings of meta-analyses from randomised controlled trials on patients with chronic diseases.[9] However, less than one-fifth of those reporting not having been highly physically active already previously reported having increased their physical activity and thus the positive responses can be generalised only to the subgroup that really increased their physical activity during the life intervention programme.

Strengths and weaknesses

FIN-D2D is the first national effort to implement the prevention of diabetes in a primary healthcare setting. The first limitation of our study is that the possibility to participate in the intervention was provided to a large number of individuals, and those who participated may only represent the highly motivated fraction of the population. Second, our study includes limitations related to the analysis of effects of real-life interventions concerning documentation time points and standardisation of different measurements. However, it is likely that these shortcomings do not cause biases which having a major effect on our results. Standardisation of laboratory analyses in particular is a known challenge in large-scale multicentre intervention studies. During our study period, there were some changes in the methodology of measuring fasting and 2 h glucose levels. We recorded the method used and did a subgroup analysis among those participants whose glucose levels had been measured with an identical method at baseline and follow-up. These results are not shown separately as the finding in the subgroup was similar to that presented in our results.

Physical activity changes have been quantified using a variety of subjective and objective methods[20] but it is not possible to draw any definite conclusions concerning the validity of self-report measurements compared with various direct methods.[21] Unfortunately, good studies on the validity of documenting changes in physical activity are lacking, but we used as the main indicator the self-reported change in physical activity, which we consider reliable. This is supported by the fact that the reported

Table 3 Changes from baseline to follow-up in cardiometabolic risk factors according to self-reported changes in physical activity during follow-up

	No increase in physical activity		Increase in physical activity							
Variable	n	Mean (SD)	n	Mean (SD)	p Value*	p Value†	p Value‡	p Value§	p Value¶	p Value**
Weight, kg	1380	−0.67 (5.20)	310	−4.27 (7.03)	<0.001	<0.001		<0.001	<0.001	<0.001
BMI, kg/m^2	1373	−0.26 (1.73)	310	−1.53 (2.56)	<0.001	<0.001	0.326	<0.001	<0.001	<0.001
Waist, cm	1343	−0.71 (5.31)	301	−4.30 (6.59)	<0.001	<0.001	<0.001		<0.001	<0.001
Systolic BP, mm Hg	1367	−1.21 (14.32)	308	−3.24 (14.17)	0.027	0.043	0.199	0.061	0.064	0.197
Diastolic BP, mm Hg	1367	−1.27 (8.58)	308	−2.93 (8.10)	0.002	0.003	0.059	0.015	0.008	0.013
Total cholesterol, mmol/l	1264	−0.15 (0.83)	283	−0.32 (0.85)	0.002	0.005	0.028	0.010	0.001	0.005
HDL cholesterol, mmol/l	1256	0.02 (0.29)	283	0.07 (0.30)	0.014	0.082	0.202	0.099	0.064	0.028
LDL cholesterol, mmol/l	1231	−0.17 (0.75)	278	−0.33 (0.76)	0.001	0.004	0.008	0.005	0.001	0.012
LDL/HDL ratio	1231	−0.17 (0.69)	278	−0.33 (0.66)	<0.001	0.002	<0.001	0.007	0.001	0.001
Triglycerides, mmol/l	1253	−0.02 (0.80)	282	−0.12 (0.69)	0.068	0.102	0.719	0.266	0.085	0.007
Fasting glucose, mmol/l	1380	-0.02 (0.49)	310	−0.11 (0.48)	0.004	0.021	0.102	0.078	0.027	0.011
2 h glucose, mmol/l	1378	−0.20 (1.53)	309	−0.56 (1.56)	0.023	0.004	0.016	0.031	0.002	0.001

*p Values for difference between those who did not increase versus increased physical activity during follow-up adjusted for sex and age at baseline (analysis of variance).
†p Values adjusted for sex and age at baseline and dietary change.
‡p Values adjusted for sex and age at baseline and change in weight during follow-up.
§p Values adjusted for sex and age at baseline and change in waist circumference during follow-up.
¶p Values adjusted for sex and age at baseline and number of intervention visits.
**p Values adjusted for sex and age at baseline and baseline values.
BMI, body mass index; BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

change associated expectedly with the changes between baseline and follow-up in the reports of LTPA and everyday activities (table 2).

It is noteworthy that those who increased their physical activity had more intervention visits than those who did not increase, which may contribute to increased physical activity. Those who increased their physical activity also reported more changes in their diet, but the main results remained either statistically significant or borderline significant after adjustment for the number of intervention visits and after the adjustment for dietary change. Our primary analysis strategy did not include adjustment for baseline values as the baseline values may have an influence on how intensively the intervention guidelines are given. However, we adjusted our results also for baseline values (table 3). After adjustment for baseline values, other group differences persisted as statistically significant but the borderline difference in systolic BP was no more statistically significant after the adjustment. However, it is to note that the group difference in triglyceride levels between those who increased and did not increase physical activity became statistically significant after the adjustment for baseline values (p=0.007). Our study did not include fitness tests, which is a limitation, as a possible increase in aerobic fitness would have provided additional proof for the benefits of physical activity. Increase in physical fitness is one of the most direct and consistent effects of increasing physical activity and is important for future functioning and health.[9]

Comparisons to other studies

Randomised controlled trials with structured, intensive lifestyle interventions promoting healthy eating and moderate physical activity and focusing on the prevention of diabetes in people at high risk[1 2 5 22] have shown that clinically significant weight loss of ≥3.5 kg can be achieved. In our subgroup increasing their physical activity level, the mean reduction was 4.3 kg. It has been shown that physical activity maintains or may increase muscle mass but exercise interventions reduce visceral fat.[23] In our study, too, waist circumference was reduced as an indirect indicator of intra-abdominal fat reduction. Furthermore, as maintaining weight loss over time is a challenge,[24] regular physical activity is an important way of maintaining both a healthy body weight and a healthy body composition.

The mean decreases in systolic (−3.2 mm Hg) and diastolic (−2.9 mm Hg) BP levels among those who increased their physical activity are in line with those observed in randomised controlled trials.[25] Our results are in line with the existing literature that exercise training is usually beneficial for the lipid risk factor

levels, although there has been some variation in the results of different randomised controlled trials.[26–28] It is noteworthy that reduction in LDL levels was statistically significant also after adjustment for change in diet, change in body weight or change in waist circumference. Physical activity improves serum lipid risk factor levels also via other mechanisms than reduction of body fat.[29] Reduced LDL levels are obviously associated with a reduced risk for cardiovascular events,[30 31] although longer follow-ups are needed to see whether this is true in our participants. Also, we found a small increase in HDL levels, which usually increases fairly consistently in response to long-term vigorous physical activity.[26] Reduced LDL levels and increased HDL levels contributed to the reduced LDL/HDL ratio (table 3).

Fasting glucose levels and 2 h glucose levels in the OGTT also decreased in the subgroup who increased physical activity, which is in accordance with the finding of increased insulin sensitivity usually seen in physically active individuals. The known independent effect of physical activity on insulin sensitivity was seen in particular in the 2 h glucose levels (table 3).

Meaning of the study and implications

Increasing physical activity seems to be an important component in cardiometabolic risk reduction among individuals at high risk for type 2 diabetes participating in preventive interventions in routine clinical settings. In this study, we did not compare individuals who received physical activity counselling with individuals who did not receive counselling. However, our finding supports current recommendations on that increasing physical activity is an important part of the preventive interventions of individuals at elevated risk for type 2 diabetes. The finding concurs with the abundant observational evidence on the importance of increasing physical activity on health.[8 32 33]

Unanswered questions and future research

The purpose of this analysis was not to investigate the factors that predict increases in LTPA. As only a small proportion of our target group increased their physical activity, there is a need to investigate how to identify those sedentary individuals able to increase their physical activity levels and also to study the most effective ways of implementing physical activity recommendations in real life, possibly taking into account individuals' self-efficacy, psychological flexibility and other factors.

Conclusion

Increasing physical activity seems to be an important feature of cardiometabolic risk reduction among individuals at high risk for type 2 diabetes participating in preventive interventions in routine clinical settings.

Author affiliations
[1]Department of Health Sciences, University of Jyväskylä, Jyväskylä, Finland
[2]Institute of Health Sciences, Faculty of Medicine, University of Oulu, Oulu, Finland
[3]Unit of General Practice, Oulu University Hospital, Oulu, Finland
[4]Pirkanmaa Hospital District, Tampere, Finland
[5]Finnish Diabetes Association, Tampere, Finland
[6]Department of Medicine, Kuopio University Hospital, Kuopio, Finland
[7]Department of Internal Medicine, Seinäjoki Central Hospital, Seinäjoki, Finland
[8]Department of Internal Medicine, Central Finland Hospital District, Jyväskylä, Finland
[9]Unit of Family Practice, Central Finland Hospital District, Jyväskylä, Finland
[10]Kuopio University Hospital, Kuopio, Finland
[11]University of Eastern Finland, Kuopio, Finland
[12]Faculty of Health Sciences, University of Eastern Finland, Kuopio, Finland
[13]National Institute for Health and Welfare, Helsinki, Finland
[14]Department of Public Health, Hjelt Institute, University of Helsinki, Helsinki, Finland
[15]Institute of Public Health and Clinical Nutrition, University of Eastern Finland, Kuopio, Finland
[16]Research Unit, Kuopio University Hospital, Finland
[17]Unit of General Practice, Oulu University Hospital District, Oulu, Finland

Funding FIN-D2D was supported by hospital districts of Pirkanmaa, Southern Ostrobothnia, Northern Ostrobothnia, Central Finland and Northern Savo, the Finnish National Public Health Institute, the Finnish Diabetes Association, the Ministry of Social Affairs and Health in Finland, Finland's Slottery Machine Association, the Academy of Finland (grant number 129293) and Commission of the European Communities, Directorate of Public Health (grant agreement number 2004310) in cooperation with the FIN-D2D Study Group and the Steering Committee: Huttunen J, Kesäniemi A, Kiuru S, Niskanen L, Oksa H, Pihlajamäki J, Puolakka J, Puska P, Saaristo T, Vanhala M and Uusitupa M. Role of study sponsors: the funding organisations (except for the hospital districts) had no role in the design and conduct of the study; the collection, management, analysis and interpretation of data; and the writing of the manuscript for publication.

Competing interests None.

Ethics approval Ministry of Social Affairs and Health in Finland gave the permission to collect the data from healthcare units specifically for scientific evaluation purposes including scientific publication of data to National Public Health Institute.

Contributors UMK designed this post hoc substudy, drafted the manuscript and is the guarantor. JJ analysed the data. All authors contributed to the study design, collection and interpretation of data and writing of the manuscript. All authors declare that they accept full responsibility for the conduct of the study, had access to the data and controlled the decision to publish.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement No additional data available.

REFERENCES

1. Tuomilehto J, Lindström J, Eriksson JG, *et al*. Prevention of Type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343–50.
2. Knowler WC, Barret-Connor E, Fowler SE, *et al*; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393–403.
3. Manson JE, Rimm EB, Stampfer MJ, *et al*. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. *Lancet* 1991;338:774–8.
4. Helmrich SP, Ragland DR, Leung RW, *et al*. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. *N Engl J Med* 1991;325:147–52.
5. Pan XR, Li GW, Hu YH, *et al*. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537–44.
6. Waller K, Kaprio J, Lehtovirta M, *et al*. Leisure-time physical activity and type 2 diabetes during a 28-year follow-up in twins. *Diabetologia* 2010;53:2531–7.
7. Laaksonen DE, Lindström J, Lakka TA, *et al*; The Finnish Diabetes Prevention Study. Physical activity in the prevention of type 2 diabetes. *Diabetes* 2005;54:158–65.
8. Physical Activity Guidelines Advisory Committee. *Physica Activity Guidelines Advisory Committee Report, 2008*. Washington, DC: U.S. Department of Health and Human Services, 2008.

9. Kujala UM. Evidence of the effects of exercise therapy in the treatment of chronic disease. *Br J Sports Med* 2009;43:550–5.
10. Cardona-Morrell M, Rychetnik L, Morrell SL, *et al*. Reduction of diabetes risk in routine clinical practice: are physical activity and nutrition interventions feasible and are the outcomes from reference trials replicable? A systematic review and meta-analysis. *BMC Public Health* 2010;10:653. http://www.biomedcentral.com/1471-2458/10/658
11. Finnish Diabetes Association. *Implementation of Type 2 Diabetes Prevention Plan. Project Plan 2003-2007, FIN-D2D Project*. Tampere, Finland. 2006. http://www.diabetes.fi/files/1107/Implementation_of_Type_2_Diabetes_Prevention._Project_Plan_2003-2007.pdf
12. Saaristo T, Peltonen M, Keinänen-Kiukaanniemi S, *et al*. National type 2 diabetes prevention programme in Finland: FIN-D2D. *Int J Circumpolar Health* 2007;66:101–2.
13. Saaristo T, Moilanen L, Korpi-Hyövälti E, *et al*. Lifestyle intervention for prevention of type 2 diabetes in primary health care: one-year follow-up of the Finnish national diabetes prevention program (FIN-D2D). *Diabetes Care* 2010;33:2146–51.
14. Lindström J, Tuomilehto J. The diabetes risk score: a practical tool to predict type 2 diabetes risk. *Diabetes Care* 2003;26:725–31.
15. Saaristo T, Moilanen L, Jokelainen J, *et al*. Cardiometabolic profile of people screened for high risk of type 2 diabetes in a national prevention programme (FIN-D2D). *Prim Care Diabetes* 2010;4:231–9.
16. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without the use of the preparative ultracentrifuge. *Clin Chem* 1972;18:499–502.
17. WHO. *Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications. Report of a WHO Consultation. Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva, Switzerland: World Health Organization, 1999. Report No 99.2.
18. Lindström J, Louheranta A, Mannelin M, *et al*. The Finnish Diabetes Prevention Study (DPS). Lifestyle intervention and 3-year results on diet and physical activity. *Diabetes Care* 2003;26:3230–6.
19. Finnish Diabetes Association. *Implementation of Type 2 Diabetes Prevention Plan: Project Plan 2003-2007, FIN-D2D Project [Article Online]*. 2006. http://www.diabetes.fi/files/1107/Implementation_of_Type_2_Diabetes_Prevention._Project_Plan_2003-2007.pdf
20. Baker PRA, Francis DP, Soares J, *et al*. Community wide interventions for increasing physical activity. *Cochrane Database Syst Rev* 2011;(4):CD008366.
21. Prince SA, Adamo KB, Hamel ME, *et al*. A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review. *Int J Behavior Nutr Phys Activity* 2008;5:56. doi:10.1186/1479-5868-5-56.
22. Ramachandran A, Snehalatha C, Mary S, *et al*. The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* 2006;49:289–97.
23. Ohkawara K, Tanaka S, Miyachi M, *et al*. A dose-response relation between aerobic exercise and visceral fat reduction: systematic review of clinical trials. *Int J Obesity* 2007;31:1786–97.
24. Svetkey LP, Stevens VJ, Brantley PJ, *et al*. Comparison of strategies for sustaining weight loss: the weight loss maintenance randomized controlled trial. *JAMA* 2008;299:1139–48.
25. Cornelissen VA, Fagard RH. Effect of endurance training on blood pressure, blood pressure-regulating mechanisms, and cardiovascular risk factors. *J Hypertens* 2005;23:251–9.
26. Leon AS, Sanchez OA. Response of blood lipids to exercise training alone or combined with dietary intervention. *Med Sci Sports Exerc* 2001;33:502–15.
27. Kelley GA, Kelley KS, Vu Tran Z. Aerobic exercise, lipids and lipoproteins in overweight and obese adults: a meta-analysis of randomized controlled trials. *Int J Obesity* 2005;29:881–93.
28. Kelley GA, Kelley KS. Effects of aerobic exercise on lipids and lipoproteins in adults with type 2 diabetes: a meta-analysis of randomized-controlled trials. *Public Health* 2007;121:643–55.
29. Leskinen T, Rinnankoski-Tuikka R, Rintala M, *et al*. Differences in muscle and adipose tissue gene expression and cardio-metabolic risk factors in the members of physical activity discordant twin pairs. *PLoS One* 2010;5:e12609. doi:10.1371/journal.pone.0012609.
30. Vartiainen E, Puska P, Pekkanen J, *et al*. Changes in risk factors explain changes in mortality from ischaemic heart disease in Finland. *BMJ* 1994;309:23–7.
31. Vartiainen E, Sarti C, Tuomilehto J, *et al*. Do changes in cardiovascular risk factors explain changes in mortality from stroke in Finland? *BMJ* 1995;310:901–4.
32. Byberg L, Melhus H, Gedeborg R, *et al*. Total mortality after changes in leisure time physical activity in 50 year old men: 35 year follow-up of population based cohort. *BMJ* 2009;338:b688. doi:10.1136/bmj.b688.
33. Weiler R, Stamatakis E, Blair S. Should health policy focus on physical activity rather than obesity? *Yes. BMJ* 2010;340:c2603. doi:10.1136/bmj.c2603.

APPENDIX 1
Question on leisure physical activity and diet

Physical activity

1. How much do you exercise and exert yourself physically in your leisure time?
If it varies greatly according to the season mark the alternative which best describes the average situation.

1 □ In my leisure time I read, watch TV, and do household tasks which does not make me move much and which does not physically tax me.
2 □ In my leisure time I walk, cycle or exercise otherwise at least 4 hours per week. This includes walking, fishing and hunting, light gardening etc. but excludes travel to work.
3 □ In my leisure time I exercise to maintain my physical condition, e.g. running, jogging, skiing, gymnastics, swimming, playing ball games or I do heavy gardening or the like for at least 3 hours per week.
4 □ In my leisure time I regularly engage in competitive sports such as running, orienteering, skiing, swimming, playing ball games or other heavy sports several times a week.

2. How many times per week do you exercise in your leisure time so that you are at least mildly out of breath and sweaty?
(if not at all, mark 0.)

|__|__| times per week

3. During your leisure time how many minutes do you spend daily walking, cycling or engage in a hobby that requires moving about (yard work or gardening, fixing or cleaning the house)?
Do not count in the activity needed at work, traveling to work or leisure time sports.

1 □ Less than 15 minutes per day
2 □15-29 minutes per day
3 □ 30-44 minutes daily
4 □ 45-59 minutes daily
5 □ Over an hour per day

4. Have you increased your physical activity/exercise training during the past year?

1 □ No, and I do not intend to increase
2 □ No, but I intend to increase in near future
3 □ I have tried to increase
4 □ I have clearly increased
5 □ I have already previously been highly physically active

5. How demanding is your work physically? The activity at work is divided into four groups.
If you do not work mark 1.

1 □ My work is mainly done sitting down and I do not walk much during my working hours
2 □ I walk quite much in my work, but I do not have to lift or carry heavy objects
3 □ I have to walk and lift much or to take the stairs or go uphill
4 □ My work is heavy manual labor in which I have to lift or carry heavy objects, dig, shovel or split wood

Diet

6. Have you decreased the amount of fat in your diet during the past year? (For example changed the light milk to skim milk or reduced fat on bread or tried to choose low fat food or products.)

1 □ No, and I do not intend to decrease
2 □ No, but I intend to decrease in near future
3 □ I have tried to decrease
4 □ I have clearly decreased
5 □ My diet has been already previously low-fat diet

7. Have you changed the quality of fat you have used to softer one during the past year? (For example changed butter-vegetable oil mixture to soft margarine, begun to use oil in cooking or increased the amount of fish meals)

1 □ No, and I do not intend to change
2 □ No, but I intend to change in near future
3 □ I have tried to change
4 □ I have clearly changed
5 □ I have already previously used mainly soft fats

8. Have you increased the use of vegetables, fruits and berries during the past year?

1 □ No, and I do not intend to increase
2 □ No, but I intend to increase in near future
3 □ I have tried to increase
4 □ I have clearly increased
5 □ I have already previously used a lot of vegetables, fruits and berries

STROBE 2007 (v4) checklist of items to be included in reports of observational studies in epidemiology*

Checklist for cohort, case-control, and cross-sectional studies (combined)

Section/Topic	Item #	Recommendation	Reported on page #
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract	1, 2
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found	2, 3
Introduction			
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	4
Objectives	3	State specific objectives, including any pre-specified hypotheses	4
Methods			
Study design	4	Present key elements of study design early in the paper	4-6
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection	4-6
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants	4-6, Fig. 1
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case	-
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers. Give diagnostic criteria, if applicable	4-6
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	*4-6*
Bias	9	Describe any efforts to address potential sources of bias	4-6, 8-9
Study size	10	Explain how the study size was arrived at	4-6, Fig. 1
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	4-6
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding	6
		(*b*) Describe any methods used to examine subgroups and interactions	6
		(*c*) Explain how missing data were addressed	-

		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy	4-6, Fig. 1
		(*e*) Describe any sensitivity analyses	-
Results			
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	4-6, Fig. 1
		(b) Give reasons for non-participation at each stage	Fig. 1
		(c) Consider use of a flow diagram	
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	Table 1.
		(b) Indicate number of participants with missing data for each variable of interest	Fig 1., Tables
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)	6
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time	7-8, Tables
		Case-control study—Report numbers in each exposure category, or summary measures of exposure	-
		Cross-sectional study—Report numbers of outcome events or summary measures	-
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included	7-8, Table 3.
		(*b*) Report category boundaries when continuous variables were categorized	-
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	-
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	3-9
Discussion			
Key results	18	Summarise key results with reference to study objectives	8
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	8-9
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	8-9
Generalisability	21	Discuss the generalisability (external validity) of the study results	9
Other information			
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	11

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.