Determinants for the Effectiveness of Lifestyle Intervention in the Finnish Diabetes Prevention Study

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OBJECTIVE — Intensive lifestyle intervention significantly reduced diabetes incidence among the participants in the Finnish Diabetes Prevention Study. We investigated whether and to what extent risk factors for type 2 diabetes and other baseline characteristics of the study participants modified the effectiveness of the lifestyle intervention.

RESEARCH DESIGN AND METHODS — Overweight, middle-aged volunteers with impaired glucose tolerance were randomly assigned to intensive lifestyle intervention ($n = 265$) or to a control group ($n = 257$) for a median of 4 years. Diabetes status was ascertained annually with repeated oral glucose tolerance testing. Incidence rates of diabetes and hazard ratios (HRs) comparing the intervention group with the control group were calculated by sex and baseline tertiles of age, BMI, waist circumference, plasma glucose concentration at fasting and 2 h after a glucose load, fasting serum insulin and insulin resistance index, and categories of composite baseline Finnish Diabetes Risk Score (FINDRISC). Interactions between the intervention assignment and baseline risk factors on diabetes risk were analyzed.

RESULTS — The intervention was most effective among the oldest individuals (HRs 0.77, 0.49, and 0.36 by increasing age tertiles, respectively; $P_{interaction} = 0.0130$) and those with a high baseline FINDRISC (HRs 1.09, 0.84, 0.34, and 0.22 by increasing risk score category, respectively; $P_{interaction} = 0.0400$). The effect of the intervention on diabetes risk was not modified by other baseline characteristics or risk factors.

CONCLUSIONS — The FINDRISC may be useful in identifying high-risk groups most likely to benefit from intensive lifestyle intervention to prevent type 2 diabetes.

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Randomized controlled trials with lifestyle intervention in individuals with impaired glucose tolerance (IGT) have conclusively demonstrated that progression to manifesting type 2 diabetes can be prevented or at least postponed (1–3). Our recent results from the postintervention follow-up of the Finnish Diabetes Prevention Study (DPS) emphasized the power of lifestyle intervention: incidence of diabetes remained reduced even after the active intervention was stopped (4).

However, it is evident that the effect of lifestyle intervention varies among individuals. We have previously shown that the risk reduction in diabetes incidence depends on adherence to the lifestyle intervention goals (2,4). Whether the effectiveness of lifestyle intervention to reduce diabetes risk is modified by baseline risk factors for type 2 diabetes or other characteristics is unclear. If individuals who are most likely to benefit from lifestyle intervention could be identified in advance, intensified actions could be directed to them in the first place. This would decrease the number needed to treat (NNT) and simultaneously also reduce the costs.

Participants were included in the DPS trial on the basis of their high risk of type 2 diabetes, defined as being overweight and having IGT according to two consecutive 75-g oral glucose tolerance tests (OGTTs). Screening for high diabetes risk in the general population using an OGTT would be expensive and time-consuming; therefore, for population screening purposes, we developed an alternative method that characterizes individuals according to their future diabetes risk: the Finnish Diabetes Risk Score (FINDRISC) (5). The FINDRISC, a simple risk assessment tool, was developed and validated using two large, population-based cohorts. The FINDRISC combines the effects of eight risk characteristics and provides an estimate of a 10-year absolute risk of type 2 diabetes. The aim of the present analyses was to clarify whether and to what extent risk factors for type 2 diabetes, the composite FINDRISC, and other characteristics of the trial partici-

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*A complete list of the members of the Finnish Diabetes Prevention Study Group can be found in the APPENDIX.

Abbreviations: DPS, the Finnish Diabetes Prevention Study; FINDRISC, the Finnish Diabetes Risk Score; HOMA-IR, homeostasis model assessment of insulin resistance; IGT, impaired glucose tolerance; NNT, number needed to treat; OGTT, oral glucose tolerance test.

Table 1—*FINDRISC scoring*

	FINDRISC scoring
Age	
<45 years	0
45–54 years	2
55–64 years	3
>64 years	4
BMI	
≤25 kg/m^2	0
>25–30 kg/m^2	1
>30 kg/m^2	3
Waist circumference	
Men, <94 cm; women, <80 cm	0
Men, 94 to <102 cm; women, 80 to <88 cm	3
Men, ≥102 cm; women, ≥ 88 cm	4
History of antihypertensive drug treatment	
No	0
Yes	2
Previously measured high blood glucose	
No	0
Yes	5
Consumption of vegetables, fruits, or berries	
Every day	0
Less often than once a day	1
Physical activity	
≥30 min/day	0
<30 min/day	2
Family history of diabetes	
No	0
Yes, second degree: grandparent, aunt, uncle, first cousin	3
Yes, first degree: parent, brother, sister, own child	5

pants measured at baseline modified the effectiveness of lifestyle intervention in reducing the incidence of diabetes.

RESEARCH DESIGN AND METHODS

We used the data collected at baseline and during the intensive intervention period of the DPS, a multicenter study with 522 participants. The study design was described in detail previously (2,6). According to the inclusion criteria, the participants (172 men and 350 women) were middle-aged (40–64 years) and overweight (BMI >25 kg/m^2) at baseline and had IGT according to World Health Organization 1985 criteria (7). All study participants gave written informed consent, and the study protocol was approved by the ethics committee of the National Public Health Institute, Helsinki, Finland.

After the screening examinations, the study participants were randomly assigned either to receive intensive lifestyle intervention or to serve as the control group. The participants were advised to reduce weight (>5% from baseline weight), to exercise (>4 h/week), and to consume a moderate-fat (total fat <30% of total energy consumed and saturated fat <10% of total energy consumed), high-fiber (≥15 g/1,000 kcal) diet. The participants assigned to receive intensive intervention were given individualized, detailed, continuing dietary counseling by the study nutritionist (2,6). They were also encouraged to be engaged in physical activities, and free of charge, supervised resistance training sessions were offered. For the participants in the control group, the lifestyle advice was given as standard, nonpersonalized counseling at one session at baseline.

The clinical examination included the measurement of weight (in light indoor clothes to the nearest 100 g), height (without shoes to the nearest 1 mm), and waist circumference (midway between the lowest rib and iliac crest to the nearest 1 mm). BMI was calculated by dividing weight in kilograms by the square of height in meters. The annually measured biochemical markers included fasting (12-h fast) and 2-h postchallenge (75-g OGTT) plasma glucose. Plasma glucose was determined locally according to standard guidelines. Fasting insulin was measured by a radioimmunoassay method (Pharmacia, Uppsala, Sweden) in the central laboratory in Helsinki.

Questionnaires about previous health and disease, consumption of drugs, family history of diabetes, and previously measured high blood glucose values (e.g., gestational diabetes mellitus) were collected. Participants were asked whether they considered themselves sedentary (reporting that during their spare time they mostly read, watch TV, and spend time in ways that do not restrain physically) or physically active (engaged in some kind of moderately strenuous activity, e.g., walking at least 4 h/week or >30 min/day). In addition, they were asked to complete a detailed 12-month physical activity questionnaire, and the amounts of moderate and strenuous physical activities were calculated (8). Dietary intake was assessed using 3-day food records collected at baseline and annual visits. The nutrient intakes were calculated using a dietary analysis program and the database developed in the National Public Health Institute (9).

The FINDRISC (5) value for each DPS participant was computed retrospectively using clinical and questionnaire data collected at baseline (age, BMI, waist circumference, history of antihypertensive drug treatment, previously measured high blood glucose, physical activity, consumption of fruits, berries, or vegetables, and family history of diabetes). The FINDRISC scoring is represented in Table 1. Some modifications to the original risk score had to be made. First, the DPS participants had been asked only about first-degree relatives with diabetes; therefore, information on second-degree relatives had to be omitted. Second, the FINDRISC includes a question about daily fruit, berry, and vegetable consumption, and to approximate it we derived such data from the baseline 3-day food records. Participants consuming at least 80 g (equivalent to one portion) of fruits, berries, and/or vegetables on each day of the food record were categorized as daily fruit and vegetable consumers.

The development of type 2 diabetes was the primary end point. Diabetes was defined according to the World Health Organization 1985 criteria (7), i.e., either fasting plasma glucose ≥7.8 mmol/l or 2-h postchallenge plasma glucose ≥11.1 mmol/l in two OGTTs. Insulin resistance

Table 2—*Baseline characteristics of the DPS participants by intervention assignment*

	Intervention group	Control group
n (male/female)	265 (91/174)	257 (81/176)
Age (years)	55 ± 7	55 ± 7
BMI (kg/m^2)		
Men	30.1 ± 3.5	29.7 ± 3.6
Women	32.1 ± 4.9	31.7 ± 4.7
Waist circumference (cm)		
Men	104 ± 10	104 ± 10
Women	101 ± 11	99 ± 11
Fasting plasma glucose (mmol/l)	6.1 ± 0.8	6.2 ± 0.7
2-h plasma glucose (mmol/l)	8.9 ± 1.5	8.9 ± 1.5
Fasting serum insulin (mU/l)	15 ± 7	15 ± 8
HOMA-IR	4.1 ± 2.2	4.2 ± 2.4
FINDRISC	13.3 ± 3.8	13.4 ± 4.2

Data are means ± SD unless otherwise indicated.

at baseline was estimated with the homeostasis model assessment of insulin resistance (HOMA-IR) using baseline fasting glucose and insulin values: fasting plasma insulin (microinternational units per milliliter) × fasting plasma glucose (millimoles per liter)/22.5 (10).

Statistical analyses were performed with the statistics package Stata (release 8.0; STATA, College Station, TX). The incidence of diabetes per 100 person-years of follow-up and 95% CIs were calculated. The Cox model was used to calculate the hazard ratios (HRs) for the development of diabetes between the groups, using the control group as the reference category, by sex and baseline tertiles of age, BMI, sex-specific waist circumference, fasting and 2-h plasma glucose concentration, fasting serum insulin, HOMA-IR, and the estimated diabetes risk category based on the FINDRISC. To test whether the effect of intervention on diabetes risk was independent of baseline risk factor levels, an interaction term between the intervention assignment and baseline risk factors, respectively, was included in the Cox models. Furthermore, the interactions between the group assignment and baseline age and FINDRISC on changes in body weight, dietary intake, and physical activity were analyzed with ANCOVA, adjusting for the baseline values of these parameters. The NNT to prevent one new case of diabetes was calculated as the inverse of the absolute risk reduction among the intervention group compared with that among the control group.

RESULTS — Baseline characteristics of the participants in the intensive intervention and control groups are given in Table 2. We were able to calculate baseline FINDRISC for 509 DPS participants. The median FINDRISC was 13 (range 1–24), similar in both groups ($P = 0.68$) and slightly higher in women than in men (mean 13.8 vs. 12.5, $P < 0.001$).

After a median follow-up of 4 years, the incidence rate of diabetes was 4.1 per 100 person-years in the intervention group and 7.4 per 100 person-years in the control group. The overall HR (intervention group compared with control group) thus was 0.54 (95% CI 0.37–0.78). Table 3 shows the diabetes incidence rates and HRs by baseline characteristics. Among men the HR was 0.43 (0.22–0.81), and among women it was 0.61 (0.39–0.97), with no statistically significant interaction between sex and intervention assignment ($P = 0.33$). The diabetes incidence rate did not differ by age among the control group participants, but in the intervention group it decreased by increasing age. Accordingly, the HRs in the intervention group compared with those in the control group decreased by increasing age tertile and were 0.77 (0.44–1.38) among the youngest tertile (age <51 years), 0.49 (0.26–0.93) among the middle tertile (age 51–61 years), and 0.36 (0.17–0.80) among the oldest tertile (age >61 years at baseline). The interaction between baseline age as a continuous variable and intervention assignment was statistically significant with $P = 0.0130$.

Adiposity markers at baseline (BMI and waist circumference) were related to diabetes incidence in both control and intervention groups. The effect of lifestyle intervention appeared to be similar regardless of baseline BMI and waist circumference ($P_{interaction}$ 0.75 and 1.00, respectively).

Baseline glycemic (fasting and 2-h glucose) status was directly associated with diabetes incidence in both control and intervention groups. The effect of intervention was independent of glycemic status ($P = 0.68$ and $P = 0.69$ for interaction between intervention assignment and fasting and 2-h glucose levels, respectively). Similarly, markers of insulin sensitivity, fasting insulin and HOMA-IR, were associated with diabetes incidence (fasting insulin only in the control group), but there was no statistically significant interaction between those markers and intervention assignment on diabetes risk reduction ($P_{interaction} = 0.85$ and 0.98, respectively).

The FINDRISC was directly associated with diabetes incidence among the control group ($P < 0.001$) but not among the intervention group participants ($P = 0.941$). The HRs in the intervention group compared with those for the control group were 1.09 (95% CI 0.38–3.09), 0.84 (0.45–1.59), 0.34 (0.19–0.62), and 0.22 (0.06–0.88) by increasing FINDRISC category, respectively. The interaction between the continuous FINDRISC at baseline and intervention assignment on diabetes risk was statistically significant ($P = 0.0400$).

To further clarify the reason for intervention effect modification, we analyzed the participants' adherence to the specific lifestyle goals (weight reduction, decrease in total and saturated fat and increase in fiber intake, and increase in physical activity) by age and FINDRISC categories. Baseline age was inversely associated with weight reduction among the intervention group participants (−5.2, −5.0, and −3.3 kg according to increasing age tertile; $P_{trend} = 0.020$) and change in fiber density of the diet (+3.1 g/1,000 kcal, +2.9 g/1,000 kcal, and +1.6 g/1,000 kcal according to increasing age tertile; $P_{trend} = 0.031$). The interaction term (age at baseline × change in fiber density) was statistically significant ($P = 0.048$). There were no statistically significant interactions between baseline FINDRISC and intervention assignment on achievement of any of the intervention goals.

Finally, we used the DPS data to calculate the NNTs to prevent one case of diabetes. In the entire study population after a 4-year intervention, the NNT was 7.7. Among those with a baseline FINDRISC value <15, the NNT was 24.8, and

Table 3—*Incidence rates and HRs (95% CIs) for diabetes by baseline characteristics*

	Incidence rate (cases/100 person-years)		HR (intervention vs. control group)	$P_{interaction}$
	Intervention group	Control group		
Sex				
Men (n = 172)	3.7 (2.2–6.2)	8.6 (5.8–12.6)	0.43 (0.22–0.81)	
Women (n = 350)	4.3 (3.0–6.2)	6.9 (5.2–9.2)	0.61 (0.39–0.97)	0.33
$P_{difference}$	0.50	0.40		
Age tertiles				
<51 years	6.0 (3.9–9.2)	7.6 (5.1–11.2)	0.77 (0.44–1.38)	
51–61 years	4.0 (2.3–6.7)	8.0 (5.5–11.5)	0.49 (0.26–0.93)	
>61 years	2.4 (1.3–4.7)	6.6 (4.2–10.3)	0.36 (0.17–0.80)	0.0130
P_{trend}	0.039	0.71		
BMI tertiles				
<28.7 kg/m^2	1.7 (0.8–3.7)	5.2 (3.3–8.1)	0.32 (0.13–0.79)	
28.7–32.3 kg/m^2	4.8 (3.0–7.7)	7.9 (5.3–11.9)	0.59 (0.32–1.10)	
>32.3 kg/m^2	5.8 (3.8–8.9)	9.6 (6.7–13.8)	0.60 (0.34–1.04)	0.75
P_{trend}	0.003	0.026		
Waist circumference tertiles				
<100.0 cm (M), <94.2 cm (W)	2.0 (0.9–4.1)	5.3 (3.4–8.3)	0.38 (0.16–0.87)	
100.0–108.0 cm (M), 94.2–104.0 cm (W)	4.1 (2.5–6.8)	7.3 (4.9–11.0)	0.55 (0.29–1.05)	
>108.0 cm (M), >104.0 cm (W)	6.3 (4.2–9.6)	10.4 (7.3–14.8)	0.60 (0.34–1.03)	1.00
P_{trend}	0.004	0.015		
Fasting glucose tertiles				
<5.8 mmol/l	2.4 (1.3–4.5)	3.8 (2.2–6.6)	0.63 (0.28–1.45)	
5.8–6.4 mmol/l	2.7 (1.4–5.1)	6.9 (4.6–10.4)	0.37 (0.18–0.79)	
>6.4 mmol/l	7.7 (5.2–11.5)	12.2 (8.8–17.0)	0.62 (0.37–1.03)	0.68
P_{trend}	0.002	<0.001		
2-h glucose tertiles				
<8.2 mmol/l	1.3 (0.5–3.0)	5.3 (3.4–8.4)	0.23 (0.09–0.61)	
8.2–9.3 mmol/l	4.2 (2.5–6.9)	5.8 (3.7–9.1)	0.70 (0.36–1.37)	
>9.3 mmol/l	7.6 (5.1–11.3)	12.0 (8.6–16.8)	0.62 (0.37–1.04)	0.69
P_{trend}	<0.001	0.003		
Fasting insulin tertiles				
<12 mU/l	3.2 (1.8–5.7)	3.9 (2.3–6.6)	0.82 (0.37–1.81)	
12–16 mU/l	4.1 (2.4–6.9)	7.2 (4.5–11.4)	0.55 (0.27–1.10)	
>16 mU/l	5.6 (3.4–9.3)	12.0 (8.5–16.9)	0.45 (0.24–0.81)	0.85
P_{trend}	0.17	<0.001		
HOMA-IR tertiles				
<2.9	2.9 (1.5–5.6)	2.6 (1.3–5.2)	1.15 (0.44–3.05)	
2.9–4.4	3.1 (1.7–5.7)	7.5 (4.9–11.5)	0.39 (0.19–0.81)	
>4.5	6.7 (4.3–10.5)	11.6 (8.2–16.3)	0.56 (0.32–0.98)	0.98
P_{trend}	0.041	<0.001		
FINDRISC				
<10 (n = 94)	4.0 (1.9–8.4)	3.6 (1.7–7.6)	1.09 (0.38–3.09)	
10–14 (n = 216)	4.0 (2.5–6.3)	4.7 (3.1–7.3)	0.84 (0.45–1.59)	
15–19 (n = 165)	4.2 (2.5–6.9)	11.7 (8.3–16.6)	0.34 (0.19–0.62)	
>19 (n = 34)	4.0 (1.0–16.0)	18.8 (10.6–33.0)	0.22 (0.06–0.88)	0.0400
P_{trend}	0.94	<0.001		

Data are incidence rates (95% CI) or HRs (95% CI). Number of subjects in each tertile is roughly 174. M, men; W, women.

among those with a baseline FINDRISC value ≥15, the NNT was 3.6.

CONCLUSIONS — The key finding of the present study was that the effectiveness of lifestyle intervention offered to the intervention group participants in the DPS was modified by their initial baseline diabetes risk, measured with the FINDRISC score. Among the participants with low baseline FINDRISC scores, the diabetes incidence rates remained virtually identical (3.6–4.7 cases/100 person-years) regardless of intervention. The participants with high baseline FINDRISC scores who were given intensive lifestyle counseling also had a similarly low incidence rate (4.0–4.2), whereas the participants with a high baseline FINDRISC value but only standard care counseling had a very high incidence rate (11.7 and 18.8 among those with FINDRISC scores of 15–19 and >19, respectively); thus, the originally increased risk in individuals with a high FINDRISC score was completely abolished with lifestyle intervention. This interaction was not explained

by better adherence to the intervention goals. These results demonstrate that even though all participants in the DPS were confirmed to have IGT at baseline, not all of them were similar in terms of their future risk of diabetes. The earlier results from the DPS control group showed that ~50% of individuals with IGT can be expected to develop diabetes within 10 years, and thus 50% will continue to have IGT or revert to normal glycemia (4). Findings from other prospective studies among Europid populations are similar (11). Albeit the FINDRISC was developed in the general population, it apparently also enables a reliable ranking of individuals with IGT regarding their future risk of developing type 2 diabetes.

The FINDRISC has been developed and validated in two Finnish population–based cohorts to predict future drug-treated diabetes (5). It has also been validated in Italy (12) and in Germany (13). The present results from the control group participants in the DPS show that it performs well also when diabetes diagnoses are done carefully in a strictly structured way using the OGTT. The FINDRISC has also been shown to perform satisfactorily when the aim is to identify people with prevalent diabetes or milder forms of glucose intolerance (14). Even though the FINDRISC evidently fails to identify all cases of IGT in a general population, that might not be a problem because the present analyses suggest that those individuals presumably have a relatively low risk of progressing to diabetes.

The present analyses also show that the participants' age was significantly associated with the effectiveness of lifestyle intervention. The intervention was most effective among the oldest (age >61 years) individuals, with a relative risk reduction of 64% compared with that in the control group. The diabetes incidence rate did not increase by increasing age in the DPS population, contrary to general trends (15). The fact that all DPS participants had IGT and were overweight and not a random population sample is a plausible explanation for this finding. Even though the generalizability of our results may therefore not be straightforward, at least they suggest that lifestyle intervention should be offered to all age-groups and not only to young people. The findings from the U.S. Diabetes Prevention Program are in concordance with ours (16). In that study, the participants in the oldest age-group (60–85 years at baseline) who achieved the largest risk reduction also lost more weight and were more physically active compared with the younger age-groups. In our study, changes in lifestyle were not more favorable among the oldest age-group. Therefore, the finding is difficult to explain but could be related to other nonmeasured components of lifestyle or better sensitivity to even modest lifestyle changes.

The incidence of diabetes increased with increasing BMI and waist circumference in both intervention and control groups, as expected. Interestingly, the effect of intervention was of the same magnitude in all BMI groups. Markers of glycemia and insulin resistance were also directly associated with diabetes risk, but there was no statistically significant interaction between any of them and group assignment, indicating that the effect of intervention on diabetes risk does not depend on any single clinical risk factor.

Lifestyle intervention is labor-intensive and therefore costly, which is one of the barriers against setting up programs with intensive lifestyle intervention to prevent type 2 diabetes. In a recent meta-analysis of published diabetes prevention trials, Gillies et al. (17) estimated that the NNT for lifestyle intervention is 6.4 for lifestyle and 10.8 for oral diabetes drugs (using the reported follow-up times ranging from 3 to 6 years). The present results suggest that by using the FINDRISC as a prescreening method, the cost-efficiency of lifestyle interventions could be drastically increased. The NNT among the whole IGT population in the DPS was estimated to be halved from 7.7 to 3.6, by a simple paper-and-pencil screening questionnaire that takes only a few minutes to complete and does not require trained personnel or laboratory equipment. Thus, in the DPS total population, eight participants in the intervention group had to be managed for 4 years to prevent one case of diabetes. By selecting only the high-risk individuals (FINDRISC ≥15), managing four individuals would have given the same result.

The weakness of the present analysis is that the DPS participants were by definition a carefully selected group with IGT based on two consecutive OGTTs. It remains, thus, somewhat unclear whether the findings can be generalized to other populations with differing risk profiles. Such data may become available from current ongoing projects such as the Finnish diabetes prevention implementation project, the FIN-D2D (18), and the Diabetes in Europe: Prevention using Lifestyle, Physical Activity and Nutritional Intervention (the DE-PLAN) (19), which use the FINDRISC as the principal screening instrument to identify individuals with a high risk of diabetes (http://www.diabetes.fi/english/risktest).

In summary, intensive lifestyle intervention in individuals with IGT was most effective among those with higher baseline age or a high FINDRISC. To improve cost-effectiveness, the FINDRISC could be used to identify target groups for lifestyle intervention to prevent type 2 diabetes.

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APPENDIX

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