The Impact of Physical Activity on the Prevention of Type 2 Diabetes: Evidence and Lessons Learned From the Diabetes Prevention Program, a Long-Standing Clinical Trial Incorporating Subjective and Objective Activity Measures

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OBJECTIVE

Across the Diabetes Prevention Program (DPP) follow-up, cumulative diabetes incidence remained lower in the lifestyle compared with the placebo and metformin randomized groups and could not be explained by weight. Collection of self-reported physical activity (PA) (yearly) with cross-sectional objective PA (in follow-up) allowed for examination of PA and its long-term impact on diabetes prevention.

RESEARCH DESIGN AND METHODS

Yearly self-reported PA and diabetes assessment and oral glucose tolerance test results (fasting glucose semiannually) were collected for 3,232 participants with one accelerometry assessment 11–13 years after randomization (n = 1,793). Mixed models determined PA differences across treatment groups. The association between PA and diabetes incidence was examined using Cox proportional hazards models.

RESULTS

There was a 6% decrease (Cox proportional hazard ratio 0.94 [95% CI 0.92, 0.96]; P < 0.001) in diabetes incidence per 6 MET-h/week increase in time-dependent PA for the entire cohort over an average of 12 years (controlled for age, sex, baseline PA, and weight). The effect of PA was greater (12% decrease) among participants less active at baseline (<7.5 MET-h/week) (n = 1,338; 0.88 [0.83, 0.93]; P < 0.0001), with stronger findings for lifestyle participants. Lifestyle had higher cumulative PA compared with metformin or placebo (P < 0.0001) and higher accelerometry total minutes per day measured during follow-up (P = 0.001 and 0.047). All associations remained significant with the addition of weight in the models.

CONCLUSIONS

PA was inversely related to incident diabetes in the entire cohort across the study, with cross-sectional accelerometry results supporting these findings. This highlights the importance of PA within lifestyle intervention efforts designed to prevent diabetes and urges health care providers to consider both PA and weight when counseling high-risk patients.
The fact that lifestyle intervention can prevent or delay the development of type 2 diabetes in high-risk adults was successfully demonstrated in a very rigorous randomized clinical trial. The strength of this trial, the Diabetes Prevention Program (DPP), was the diversity of its participants with regard to age, ethnicity, race, and geographic location (1). Because of its success, a group-delivered version of the lifestyle intervention that had been provided to the lifestyle participants was offered to participants from the other two randomized arms (metformin and placebo) at DPP end. All participants were then invited to continue as part of the DPP follow-up or outcomes study (DPPOS). During an average 10 years of follow-up since DPP randomization, the between-group differences in cumulative diabetes incidence persisted, although to a lesser degree than at DPP end (2).

The contributions of the two lifestyle goals of weight loss and physical activity (PA) were examined in lifestyle participants at the end of the DPP trial period (3.2 years average follow-up). Participants not meeting the year 1 weight goal but meeting the self-reported PA goal had a 46% lower diabetes incidence at DPP end compared with those meeting neither goal (3). However, change in self-reported leisure PA from baseline was not associated with diabetes incidence when adjusted for weight. The impact of change in PA levels beyond the end of the DPP trial period has not yet been reported in long-term follow-up.

The yearly collection of PA data through a validated questionnaire coupled with a one-time objective assessment of activity in DPPOS using accelerometry provided the opportunity to examine the overall impact of PA in the DPP and its follow-up phase. Specifically, this current investigation describes self-reported PA levels in the DPP over an average of 12 years in this diverse sample of adults and includes a highly accepted objective activity measure at follow-up in the DPPOS. Whether higher activity levels were maintained in lifestyle participants into follow-up and could explain, in part, their lower cumulative diabetes incidence (compared with the other randomized groups) was examined. Whether PA levels, examined as a continuous variable over the entire study period in the whole DPP cohort, were related to the development of diabetes after controlling for weight will also be investigated and is the primary focus of this investigation.

**RESEARCH DESIGN AND METHODS**

The DPP (1996–2001) was a multicenter (27 centers), randomized controlled clinical trial designed to determine whether treatment with lifestyle intervention or metformin could prevent or delay type 2 diabetes in high-risk adults. The incidence of diabetes was reduced in the lifestyle intervention and metformin arms compared with placebo by 58% and 31%, respectively, after a mean of 2.8 years of follow-up during the masked treatment phase. DPP results and methods have been described elsewhere (1,2,4).

Surviving participants of the original DPP were invited to participate in the long-term follow-up (DPPOS). All three DPP treatment groups were offered group-implemented lifestyle intervention. Metformin was continued in the metformin group, and the lifestyle intervention group was offered additional lifestyle support. Participants were unmasked to assignment (2). The accelerometry ancillary study involved a single, week-long collection of accelerometry data in all participants around the time of their DPPOS clinic visit (Supplementary Fig. 1).

**Participants**

Eligibility, recruitment, and random assignment of the DPP participants have been described elsewhere (5). The diverse DPP cohort comprised 3,234 adults (68% women, 45% from ethnic/racial minority groups, age ≥25 years) (Table 1) at high risk because of an elevated fasting plasma glucose (5.3–6.9 mmol/L), impaired glucose tolerance (2-h postload glucose 7.8–11.0 mmol/L), and BMI ≥24 kg/m². Eligible adults were randomly assigned to the lifestyle, metformin, or placebo group.

A total of 2,776 of the 3,149 (88%) surviving participants of the original DPP joined the DPPOS follow-up study (Supplementary Fig. 2). All active DPPOS participants who attended their annual or semiannual clinic visit between 2010 and 2012 were invited to enroll in the accelerometry ancillary study; three sites were not able to participate. The resulting accelerometry ancillary study cohort included 1,793 individuals from the 1,932 possible active DPPOS participants (93%).

**Intervention Protocols**

**DPP (1996–2001)**

Intervention protocols have been previously described (1). The lifestyle intervention group received a 16-session, individually administered core curriculum focused on reducing dietary fat and total calories and increasing moderate-intensity PA levels followed by individual and group classes with the goals of achieving and maintaining a weight loss of ≥7% initial body weight and a moderate-intensity activity level of ≥150 min/week. The metformin group received metformin 850 mg twice per day as tolerated, and the placebo group received matching placebo.

**Bridge Period (2001–2002)**

Following announcement of the primary study results, participants were unmasked to treatment assignment. Because of the success of the lifestyle intervention, all participants were offered a group-administered version of the lifestyle intervention during the 1-year bridge period, occurring between DPP end and DPPOS start.

**DPPOS Follow-Up (2002–Present)**

Maintenance group lifestyle sessions during DPPOS were offered to all participants every 3 months to reinforce the weight and activity goals, with additional classes offered to the lifestyle participants to reinvigorate their self-management behaviors. Attendance varied across the study and decreased during the DPPOS follow-up period, with attendance at DPPOS classes averaging 20% per session for all groups. Attendance and weight outcomes during the DPP/DPPOS bridge period have been published (6). With regard to the metformin arm, participants continued their medication, but the placebo was discontinued following unmasking of treatment assignments.

**Outcome Measures**

The primary DPP outcome was diabetes development diagnosed by an annual oral glucose tolerance test or a semiannual fasting plasma glucose test. Diabetes was diagnosed as follows: plasma glucose ≥126 mg/dL (7.0 mmol/L) in the fasting state or ≥200 mg/dL (11.1 mmol/L) 2 h after a 75-g oral glucose load. The diagnosis required confirmation by a second test, usually within 6 weeks, according to the same criteria.
Demographic information was collected before randomization. Weight was recorded at all clinic visits.

PA

Self-reported PA data were collected annually throughout the entire study (DPP and DPPOS) via the Modifiable Activity Questionnaire (MAQ) by trained interviewers. The MAQ assesses past year, moderate/vigorous-intensity leisure PAs common to the population in question (7,8). Although light-intensity activity comprises the largest percentage of the average adults’ day, the MAQ was not designed to measure activities that comprise that level of intensity because of concerns regarding the accuracy of recall of light-intensity activity. Only objective measures, such as the accelerometer, can validly measure activities of light intensity (9,10). Results from the MAQ are coded as MET-h/week.

On the other hand, accelerometers do capture total time spent in PAs of all intensities, including lower-intensity and unstructured activities. Each willing DPPOS participant was given an ActiGraph GT3X triaxial accelerometer at either an annual or a midyear clinic visit to wear at their waist during waking hours for 7 consecutive days. Data were collected in 1-s epochs and converted to counts (ActiLife v6 software). Nonwear (monitor removal) was identified as ≥60 consecutive min of zero counts with the exception of no more than 2 min of nonzero counts. Four or more valid days with ≥10 h of wear time each day were required for inclusion in the analyses (11). Light-intensity and moderate/vigorous-intensity activity were defined as 150–2,690 and ≥,2,691 counts/min, respectively (12,13). Data were also examined as total activity (light + moderate/vigorous activity min/day) and total monitor counts/day (intensity-weighted sum of all movement).

Statistical Analysis

In the entire DPP cohort (n = 3,234), mixed models (autoregressive covariance matrix) were used to determine longitudinal differences across treatment arms in leisure activity across the entire study from the annually collected MAQ (MET-h/week); adjusted least squares means were also tested for treatment group differences with Tukey test, for multiple comparisons (14). Models were adjusted for baseline leisure activity, age, and sex. The effect of leisure activity from the MAQ (collected yearly from baseline to the beginning of the accelerometry study) on diabetes incidence was examined using Cox proportional hazards models with time-dependent covariates. Hazard ratios (HRs) were calculated for the whole group, for lifestyle participants alone, and for the subgroups that did or did not meet the activity goal at baseline (150 min/week) as determined by the MAQ. Because of the lack of an interaction by treatment, Cox models were examined for both the entire cohort (controlled for treatment) and the lifestyle arm alone (n = 1,077). Models were adjusted for age, sex, race/ethnicity, baseline activity, and baseline and, in some models, time-dependent weight. Although diet was assessed by food frequency questionnaire intermittently throughout DPP and DPPOS, there were negligible differences across intervention groups 9 years after randomization and, therefore, was not included in the model (15).

Finally, the same Cox models were reanalyzed over the original DPP (mean of 3.2 years) in the whole cohort and then again in lifestyle participants not meeting the activity goal at baseline. Statistical analyses were conducted using SAS 9.3 software.

DPPOS Accelerometry Validation Substudy

In the DPPOS accelerometry study cohort (a subset of the DPP cohort, n = 1,793), means and medians were calculated for accelerometer-derived activity variables. Kruskal-Wallis and Wilcoxon signed rank tests were used to determine cross-sectional differences in PA across treatment arms and diabetes status groups; respectively. The mean follow-up from DPP randomization to the DPPOS accelerometry study was 12 (SD = 1.0) years. Spearman rank order correlations were used to determine the relationship between activity levels from accelerometry (objective) and MAQ (subjective).

Table 1—Demographics and randomized arm assignment for DPP participants compared with those in the ancillary study with valid accelerometer data vs. those not in the ancillary study or without valid accelerometer data

<table>
<thead>
<tr>
<th>participants, n</th>
<th>Total randomized</th>
<th>In ancillary and valid</th>
<th>Not in ancillary or not valid</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SD</td>
<td>50.6 ± 10.7</td>
<td>50.3 ± 9.7</td>
<td>50.9 ± 11.6</td>
<td>0.161</td>
</tr>
<tr>
<td>Sex (female), n (%)</td>
<td>2,191 (67.7)</td>
<td>1,126 (69.4)</td>
<td>1,065 (66.1)</td>
<td>0.041</td>
</tr>
<tr>
<td>Race/ethnicity, n (%)</td>
<td>White 1,768 (54.7)</td>
<td>860 (53.0)</td>
<td>908 (56.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>African American 645 (19.9)</td>
<td>291 (17.9)</td>
<td>354 (22.0)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hispanic 508 (15.7)</td>
<td>274 (16.9)</td>
<td>234 (14.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Asian/Pacific Islander 142 (4.4)</td>
<td>85 (5.2)</td>
<td>57 (3.5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>American Indian 171 (5.3)</td>
<td>112 (6.9)</td>
<td>59 (3.7)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²), mean ± SD</td>
<td>34.0 ± 6.7</td>
<td>33.4 ± 6.2</td>
<td>34.5 ± 7.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Weight (kg), mean ± SD</td>
<td>94.2 ± 20.3</td>
<td>92.5 ± 19.3</td>
<td>96.0 ± 21.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Leisure MET-h/week,* median (IQR)</td>
<td>9.8 (3.9–20.6)</td>
<td>10.1 (4.0–20.7)</td>
<td>9.7 (3.8–20.4)</td>
<td>0.118</td>
</tr>
<tr>
<td>Total MET-h/week,* median (IQR)</td>
<td>16.2 (5.4–58.0)</td>
<td>16.4 (5.7–56.0)</td>
<td>16.0 (5.0–59.7)</td>
<td>0.511</td>
</tr>
<tr>
<td>Treatment arm, n (%)</td>
<td>Lifestyle 1,079 (33.4)</td>
<td>536 (33.0)</td>
<td>543 (33.7)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Metformin 1,073 (33.2)</td>
<td>543 (33.5)</td>
<td>530 (32.9)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Placebo 1,082 (33.5)</td>
<td>543 (33.5)</td>
<td>539 (33.4)</td>
<td></td>
</tr>
</tbody>
</table>

*3,234 – 2 participants with missing MAQ data: 1,621 vs. 1,622 in ancillary study and 1,612 vs. 1,611 not in ancillary study.
RESULTS

PA Levels by Treatment Arm: MAQ
Annual MAQ leisure activity levels were assessed over the course of the entire study (baseline through an average of 12 years follow-up) in the full cohort ($n = 3,234$ minus only 2 participants with missing MAQ data) (Table 1). MAQ leisure activity levels were examined by treatment arm using mixed models. Models were adjusted for age, sex, and baseline activity (which did not significantly differ by randomized treatment group when adjusted for age and sex).

Adjusted mean values for activity peaked in the lifestyle arm after 1 year of intervention (Fig. 1) but remained relatively unchanged from baseline in the metformin and placebo groups at that time. Over the course of the entire investigation, the differences in leisure activity across treatment groups were significant ($P$ value for difference [P-dif] $< 0.0001$), with higher levels of reported activity in the lifestyle group (Fig. 1). When examined separately for DPP and DPPOS, the differences in leisure activity across treatment groups were significant during the DPP (P-dif $< 0.0001$), with activity slightly higher in the lifestyle group during DPPOS follow-up (P-dif $< 0.0001$).

PA Levels and Diabetes Incidence: MAQ
To address the primary focus of this article, the longitudinal association between leisure activity and diabetes incidence was examined using Cox proportional hazard models in the full cohort and then again in the lifestyle group alone, adjusted for age, sex, baseline PA, and baseline weight. Across all treatment arms combined, there was a 6% reduction (HR 0.94 [95% CI 0.92, 0.96]; $P < 0.001$) in diabetes incidence for each 6 MET-h/week (~17 min of brisk walking per day) of reported leisure activity over the 12-year follow-up period (Fig. 2). This relationship was stronger (12% reduction) in participants not meeting the PA goal of 150 min/week of moderate-intensity activity (equivalent to 7.5 MET-h/week) at baseline ($n = 1,338$, 0.88 [0.83, 0.93]; $P < 0.0001$) and weakened but remained significant when adding time-dependent weight to the models. Treatment arm or race/ethnicity in the model did not affect the results.

In analyses restricted to lifestyle participants only ($n = 1,077$) (Fig. 2), the relationship between leisure PA and diabetes development appeared slightly stronger (HR 0.90 [95% CI 0.87, 0.94]; $P < 0.0001$). Similar to the analyses in all treatment arms combined, the relationship was strongest (20% reduction) in those lifestyle participants who reported meeting the leisure activity goal at baseline ($n = 451$, 0.80 [0.72, 0.89]; $P < 0.0001$). The relationship remained significant but weakened after adding time-dependent weight to the models.

In a previous post hoc analysis of the substudy (3), when this association between self-reported PA and diabetes incidence at DPP end (3.2 years average follow-up) was reexamined, limiting the analyses to those who reported being below the PA goal at baseline, activity was significantly related to diabetes incidence, even when adjusted for weight loss in the model (data not shown).

DPPOS Accelerometer Validation Substudy
PA was measured cross-sectionally through accelerometry after a mean follow-up of 12 years. All of the DPP sites were invited to join the accelerometry ancillary study (National Institute of Diabetes and Digestive and Kidney Disease funded, A.M.K. principal investigator), with all but three clinical sites able to participate. From the 1,932 active DPPOS adults who enrolled at these participating sites, 93% agreed to take part in the accelerometry study (9 were ineligible, and 130 did not attend clinic during substudy enrollment).

A total of 1,622 ancillary study participants (90.4% of 1,793 participants) had at least 4 valid days of accelerometer recording (on the basis of triaxial data). Individuals with complete accelerometer data (vs. those without) were more likely to have confirmed diabetes (54.4% vs. 40.1%, $P < 0.001$). There were no other significant differences in key covariates across compliance groups (data not shown). At the time of ancillary study data collection, ancillary participants with valid data ($n = 1,622$) had a mean age of 63.7 (SD = 9.8) years, 70% were female, and 47.1% had self-reported race/ethnicity other than white, non-Hispanic.

The lifestyle participants had slightly higher values for both light- and moderate/vigorous-intensity leisure activity compared with metformin and placebo participants (Table 2). When time in both intensities was combined as total activity, the median (interquartile range [IQR]) for the lifestyle group was 389.4 (319.7–468.9) min/day, which was significantly greater than each of the other two randomized groups (metformin 376.1 [308.4–450.7] min/day, P-dif = 0.001; placebo 377.4 [307.4–460.6] min/day, P-dif = 0.047). The difference across all three groups was also significant ($P$-dif = 0.047) (Table 2).
PA levels were higher in those who had not developed diabetes before the accelerometry ancillary study than in those who had. This was true for all intensities of activity and across the three intervention groups (Supplementary Fig. 3).

**Correlation of Accelerometer and MAQ Activity Levels**
As stated earlier, the MAQ and accelerometer capture different intensities of PA, with the accelerometer measuring total PA (light, moderate, and vigorous) and the questionnaire mainly assessing activities of moderate and vigorous intensity. In the DPPOS, the recording time frame for the two activity measures was different (past year MAQ and past week accelerometer). Despite these differences, the moderate-intensity activity results between these two measures collected around the same clinic visit were weakly, but significantly, correlated ($r = 0.35, P < 0.05$). In a previous study in which the two instruments were examined over similar time frames, moderate/vigorous-intensity leisure activity from a past week version of the MAQ was more strongly correlated with bouts of moderate/vigorous-intensity PA recorded by accelerometers collected over the same week ($p = 0.69–0.76, P < 0.0001$) (16).

**CONCLUSIONS**
In this cohort, PA was inversely related to the development of diabetes over the long term and remain significant even when adjusted for weight change. This relationship was particularly strong in participants with lower baseline PA, supporting national guidelines suggesting the potential for greater benefits as a result of increasing activity levels in those relatively less active.

The protective effect of increased PA in this effort was in line with findings from previous prospective studies (17–19) and other large clinical trials (20,21). Among the unique strengths of this current investigation are the yearly collected subjective activity measures over a long follow-up period and the support for persistent treatment group differences in activity provided by cross-sectional results obtained from accelerometry at follow-up by this widely accepted objective measure. Both the diversity of the DPP population and its activity intervention goal, similar to national public health activity recommendations, add to the generalizability of the findings of this investigation to adults, with characteristics similar to those of DPP participants at high risk for type 2 diabetes.

When measured objectively with accelerometry in the extended follow-up period, the lifestyle arm had higher total PA levels. Subjective activity across the entire study was also higher in the lifestyle arm. Perhaps in addition to weight loss, higher total PA levels may help to partially explain the lower cumulative incidence of diabetes in the lifestyle compared with the other two randomized arms across the entire study activity (2) that, if true, is supportive of approaches to encourage greater participation in all intensities of PA.

Finally, the current investigation also provided an important lesson in evaluating the impact of PA for investigations...
such as the DPP in which baseline weight, but not activity level, is an eligibility criterion. Change in activity from baseline to DPP end (3.2 years) previously shown not to be significantly related to diabetes development (3) is significant if the analyses are limited to those less active at baseline.

Included in the limitations of this effort is the fact that the DPP lifestyle intervention, not unlike other published lifestyle investigations in the literature, comprises weight and activity goals and was not designed to test the individual contribution of either goal alone. Another limitation involves the subjective nature of the longitudinal measure of activity, although the MAQ has been widely used and validated against several objective measures, including accelerometry (16,22,23).

Given the success of lifestyle intervention in the prevention of type 2 diabetes, such as that shown in the DPP, intervention programs that are based on these landmark clinical trials are being offered in a variety of diverse community settings/clinics for the high-risk adult. Although these community translation studies have adapted the same DPP lifestyle goals of weight loss and increased activity, a review of the translation literature documented that only about one-half of these published studies include PA change in their results (24), making it difficult to assess the impact of activity on health outcomes in these translation efforts. On the basis of our current investigation, which highlights the role of PA on the development of diabetes at various phases of the lifestyle intervention, we would urge public health researchers to examine activity data in their community-based studies and urge health care professionals to look beyond their high-risk patient’s weight and consider his or her habitual PA levels when discussing lifestyle strategies to prevent progression to type 2 diabetes.

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The sponsor of this study was represented on the steering committee and played a part in study design, how the study was done, and publication. The funding agency was not represented in the writing group, although all members of the steering committee had input on the report’s contents. All authors in the writing group had access to all data. The opinions expressed are those of the investigators and do not necessarily reflect the views of the funding agencies. A complete list of centers, investigators, and staff can be found in the Supplementary Data.

Author Contributions. A.M.K. and B.R.-W. conducted the literature search, designed the study, collected data, interpreted the data, and wrote the manuscript. S.L.E. designed the study, collected data, performed the data analysis, interpreted the data, and edited and reviewed the manuscript. G.A.B., L.M.D., M.A.H., E.S.H., E.M.V., and W.C.K. assisted with the study design and data collection, contributed to discussion and data interpretation, and edited and reviewed the manuscript. A.M.K. and S.L.E. are the guarantors of this work and, as such, had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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