Personal care plans and glycaemic control: the role of body mass index and physical activity
ABSTRACT

Background
Although BMI (body mass index) and physical activity are implicated in diabetes complications, it is unclear how these factors influence personalised care planning linked to glycaemic control. This study assessed the mediating effects of BMI and physical activity on relations between personalised care plans (PCPs) and glycated haemoglobin (HbA1c) levels, using population-based data.

Method
Bootstrapping was used to analyse PCP, HbA1c, BMI, and physical activity data from 3894 respondents to the 2014 Health Survey for England, for whom HbA1c data was available, regardless of diabetes status. This group comprised 1812 (46.5%) males, 17 and 2082 (53.5%) females, aged 16 to 90 (Mean = 51.68 years, SD = 17.25).

Results
Patients with a PCP had higher HbA1c levels compared to those without a care plan. BMI influenced this relationship amongst patients aged 40 to 60; those with a PCP and higher HbA1c also tended to have higher BMI values. Physical activity did not affect the relationship between PCPs and glycaemic control.

Conclusions
BMI, but not physical activity, partly explained higher HbA1c levels in patients with a PCP. Given recent population-based evidence implicating exercise in diabetes complications, some
debate is needed on the role of physical activity in personalised care planning and glycaemic control.

**KEY POINTS**

- Patients with a PCP (personal care plan) have higher HbA$_1c$ values.
- BMI partly explains higher HbA$_1c$ levels in patients with a PCP.
- Physical activity is not implicated in the relationship between PCPs and HbA$_1c$ levels.
- Given that population-based prospective evidence implicates physical activity in diabetes-related complications, there is need for some debate on the role of physical activity in personalised care planning and glycaemic control.

**Key words**

Personal care plan; glycaemic control; BMI; physical activity

**Acknowledgements**

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INTRODUCTION

Blood glucose control is a critical aspect of diabetes care (Jia, 2016). People with diabetes, or individuals experiencing hyperglycaemia (Godoy et al., 2012, Farrokhi et al., 2011) may be offered personal care plans (PCPs) (Coulter et al., 2013) to help them manage their blood glucose (Diabetes UK, 2009). A PCP usually consists of a written document outlining specific goals, and activities designed to achieve these objectives (Diabetes UK, 2009, Coulter et al., 2013, The Health Developer Network, 2016). PCPs reflect a partnership between the doctor/nurse and their patients (Diabetes UK, 2017), are essential for effective self-management (Jansen et al., 2015, Tarkin et al., 2008), and have been implicated in improved patient outcomes (Hird et al., 2015, Russell et al., 2008).

PCPs are presumed to play an important role in HbA1c (glycated haemoglobin) levels (Diabetes UK, 2009). Setting clear goals for glycaemic control (e.g., achievable HbA1c targets), and designating specific actions to attain these objectives (e.g., weekly participation in a local sports programme), can help patients initiate and sustain key lifestyle changes essential for reducing HbA1c (Coulter et al., 2013). A recent Cochrane review of the effects of personalised care planning in adults with long-term conditions found HbA1c levels to be 0.24% lower in patients with a PCP, compared to those receiving usual care (Coulter et al., 2015). Thus, HbA1c level is an important criterion that GPs consider in deciding which patients to offer a PCP (Diabetes UK, 2017).

In 2015 Diabetes UK launched an ‘Information Prescription’ scheme to ensure diabetes patients who fail to meet HbA1c, blood pressure and cholesterol targets receive a one-page PCP containing specific action plans for improving metabolic control (e.g., reducing dietary fat, performing 150 minutes of moderate aerobic activity per week, and strength exercises ≥ 2 days per week) (Diabetes UK, 2017, Diabetes UK, 2015a). Patients with high blood pressure, high total cholesterol-to-HDL (high density lipoprotein) ratios, and
high HbA1c have a greater risk of developing complications, and hence are likely to benefit from personalised care (Diabetes UK, 2017). Information prescriptions are integrated into primary care IT systems, such as EMIS Web, so that GPs receive an automated alert if a specific patient is failing to meet their metabolic targets (Diabetes UK, 2015b). Diabetes UK literature suggests over 1000 diabetes patients a month use information prescriptions to manage their condition (Diabetes UK, 2015a). Information prescriptions can be considered a specific IT-based PCP designed to improve glycaemic control in diabetes patients with a high risk of complications (Diabetes UK, 2015a).

Glycaemic control is influenced by BMI (body mass index) and physical activity (Malnick and Knobler, 2006, Hu et al., 2014, Bhupathiraju and Hu, 2016, Gay et al., 2016, Cuenca-Garcia et al., 2012, Hamer et al., 2014). Lower BMI values are associated with better HbA1c outcomes (Patiakas and Charalampous, 2010, Senechal et al., 2013, Diels et al., 2014). For example, one study of type 2 diabetes patients found that a decrease in waist circumference, and increased physical fitness, was associated with an increased likelihood of significant HbA1c reductions (> 0.5%) (Senechal et al., 2013). A prospective study of data from a 1958 birth cohort revealed that early onset of overweight/obesity was implicated in a 23.9-fold increased risk of a HbA1c value ≥ 7% (Power and Thomas, 2011). An investigation of 2707 adults at risk from type 2 diabetes implicated higher amounts of moderate-to-vigorous physical activity in lower HbA1c values (Gay et al., 2016). Moderate-to-vigorous has been found to predict improved metabolic outcomes, including HbA1c levels, in healthy adults (Hamer et al., 2014).

BMI and physical activity affect the risk of complications in diabetes patients (Segula, 2014, Blomster et al., 2013). For example, obesity (i.e., BMI ≥ 30) is strongly implicated in cardiovascular disease (Wilson et al., 2002), high blood pressure (Segula, 2014), and higher levels of LDL cholesterol (Varbo et al., 2015). Physical inactivity has been linked to impaired
renal function, increasing retinopathy, and other complications, in patients with type 1 diabetes (Waden et al., 2008). Evidence from a long-term prospective study of over 11,000 patients with type 2 diabetes found that moderate-to-vigorous levels of physical activity (of at least 15 minutes per week) at baseline was associated with a reduced incidence of cardiovascular events, microvascular complications, and mortality rates, over a 5-year period (Blomster et al., 2013).

Despite evidence implicating BMI/physical activity in HbA\(_1c\) levels (Senechal et al., 2013, Quirk et al., 2014), and diabetes complications (Blomster et al., 2013, Waden et al., 2008, Segula, 2014), it is unclear the extent to which these factors influence the relationship between PCPs and glycaemic control (Diabetes UK, 2017). Although BMI and physical activity are not part of the criteria for offering information prescriptions to patients, (Diabetes UK, 2015a), they nevertheless constitute key lifestyle changes recommended for lowering HbA\(_1c\) in personalised care planning (Diabetes UK, 2009, Diabetes UK, 2015a). Thus, it follows that HbA\(_1c\) reductions associated with having a PCP will be partly attributable to changes in BMI and/or levels of physical activity. Similarly, poor weight control, and/or failure to adhere to physical activity targets, may negative the influence of PCPs on glycaemic control.

Nurses typically form part of health care teams who work in partnership with patients to arrange and monitor PCPs (Coulter et al., 2013), including information prescriptions (Diabetes UK, 2015b, Diabetes UK, 2017). Guidance published by Diabetes UK makes provision for a health professional to be named on information prescriptions, with a statement specifically inviting patients to discuss and agree achievable HbA\(_1c\) targets with a doctor or nurse (Diabetes UK, 2015a). There is particular emphasis on controlling HbA\(_1c\) levels, in order to reduce the risk of complications (Diabetes UK, 2015a). Prescriptions makes specific reference to lifestyle factors, meaning patient consultations are likely to involve
conversations about BMI/physical activity, in relation to glycaemic control (Diabetes UK, 2015a). As both BMI and physical activity contribute significantly to HbA1c, and related complications (Segula, 2014, Bhupathiraju and Hu, 2016, Blomster et al., 2013), it is essential to better understand how these factors influence the relationship between PCPs and HbA1c levels (Diabetes UK, 2015a).

AIM

This study had two objectives. The first was to establish the association between PCPs and HbA1c levels. Current literature suggests HbA1c can be both a precursor and outcome of PCPs. In the former scenario HbA1c level is used as a criterion for offering PCPs to patients (Diabetes UK, 2015a, Diabetes UK, 2015b). In the latter situation, PCPs can help patients lower their HbA1c level (Coulter et al., 2015). Both directions of causality are valid. For the purposes of this paper, PCP status (i.e., whether or not a patient has a PCP) was treated as the ‘predictor’ variable, and HbA1c as the ‘outcome’ measure. This is consistent with a primary objective of personalised care – to improve glycaemic control (Coulter et al., 2015) – but does not preclude the use of HbA1c as a basis for offering PCPs to patients (Diabetes UK, 2015a, Diabetes UK, 2017). The second objective was to determine the extent to which BMI and physical activity explain any relationship between PCPs and HbA1c levels.

It was expected that (a) patients with PCPs will have lower HbA1c levels compared to patients who had not agreed a care plan, and (b) BMI and physical activity will be implicated in this relationship, as mediating factors, such that the relationship between PCPs and HbA1c is partly explained by BMI and physical activity. Thus, for example, lower HbA1c values in patients with a PCP may partly reflect lower BMI scores, and/or greater physical activity levels in such patients. These hypotheses were tested both prior to and following adjustments for selected covariates, including diabetes status.
METHOD

Sample and procedure

This study analysed data on PCP status, HbA\textsubscript{1c}, BMI, and physical activity, obtained from the 2014 Health Survey for England (HSE), an annual exercise that assesses health-related parameters and lifestyle factors in children and adults (Health Survey for England, 2014).

The survey is commissioned by the Health and Social Care Information Centre, and consists of an interview (including self-administered questionnaires), followed by a visit by a nurse to collect biomedical data. The 2014 survey was completed by 8,077 adults (aged 16 and over), and over 2000 children (aged 0 to 15). The study reported here analysed data from 3894 adults for whom HbA\textsubscript{1c} data was available. This group comprised 1812 (46.5%) males, and 2082 (53.5%) females, aged 16 to 90 (Mean = 51.68 years, SD = 17.25). The sample (92%) was predominantly Caucasian.

Measures

Glycated haemoglobin (HbA\textsubscript{1c}) was based on non-fasting blood samples, and (for this study) calibrated in mmol/mol. HbA\textsubscript{1c} data provides a measure of average blood glucose levels over the previous three months (Jia, 2016).

PCP status was assessed via two questions. Firstly, respondents were asked if (a) they had ever had a PCP-related discussion with a doctor/nurse regarding a long-term condition, ‘Yes’ (1)/ ‘No or not sure’ (0); and (b) whether they had agreed a PCP with a health professional during the past 12 months, 'no PCP agreed' (0)/ 'agreed a PCP < or > 12 months ago' (1). Responses to both items were combined to form a PCP index, with a higher indicating a better PCP status (e.g., discussed and/or agreed a PCP).
Physical activity was measured using the short IPAQ (International Physical Activity Questionnaire) (Booth, 2000). The IPAQ/Short assesses three activity levels – walking, moderate-intensity, and vigorous-intensity – across several domains (leisure time, domestic/gardening, work/transport-based). Respondents receive a score for each level, reflecting a summation of duration (minutes) and frequency (days). For the purposes of this study, six separate scores were evaluated: total number of minutes usually spent per day, and in the last 7 days, doing (a) ‘vigorous-intensity’ activities, (b) ‘moderate-intensity’ activities, and (c) walking.

BMI was computed by dividing weight in kilograms by the square of height in metres squared (kg/m²) (Nuttall, 2015). Adults (aged > 16) were classified into the following groups: Less than 18.5 ‘Underweight’; 18.5 to less than 25 ‘Normal’; 25 to less than 30 ‘Overweight’; 30 or more ‘Obese’; 40 or more ‘Morbidly obese’. The present study evaluated raw BMI scores, for the purposes of hypotheses testing, and BMI groups (excluding the underweight category, and combining obese and morbidly obese groups) for descriptive statistics.

Other variables assessed included blood pressure, and diabetes status. Blood pressure was assessed using the Omron HEM 907 blood pressure monitor. Respondents were classified into three groups: BP under 130/80, BP under 140/90, but not under 130/80, and BP over 140/90. Respondents also indicated whether they had been diagnosed with high blood pressure by a doctor; ‘Yes’ (1), ‘No’ (2). Diabetes status was assessed by asking respondents if they currently have or have ever had diabetes, ‘Yes’ (1)/ ‘No’ (0).

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Insert Table 1 about here

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RESULTS

Descriptive statistics (means/SD) are presented in Table 1. PCP data was available for 42.7% (n = 1662) respondents. The vast majority of this subgroup (87.7%) had not agreed a PCP with a health professional, while the remainder (12.3%) had agreed a PCP with a doctor/nurse, ≤ or ≥ 12 months ago. These groups did not differ on measures physical activity (both groups achieved the recommended ≥ 150 minutes of moderate-intensity activity), BMI score (both groups classified as ‘Overweight’), mental wellbeing score, or gender distribution. There were also no group differences in the proportion who had been diagnosed with high blood pressure by a doctor/nurse (over 98% of respondents had received this diagnosis), and the proportion receiving earnings from employment or self-employment. However, patients who had agreed a PCP were slightly younger, more likely to have been diagnosed with diabetes, and had higher blood glucose levels (HbA1c).

Table 2 shows the bivariate associations between variables. Patients who had discussed and agreed a PCP with their doctor were younger, generated higher HbA1c values, more likely to have been diagnosed with diabetes, and performed fewer minutes of moderate-
intensity physical activity during the previous 7 days. In addition to denoting diabetes status, higher HbA$_{1c}$ values were associated with greater BMI, and fewer minutes of physical activity per day/week. Higher BMI values also depicted older age, doctor-diagnosed HBP, having ever had diabetes, and fewer minutes of physical activity. Gender differences are also noteworthy: overall, females had lower blood pressure, and lower levels of light (i.e., walking), moderate-, and vigorous-intensity physical activity per day, and during the past 7 days.

Hypothesis testing was performed using a bootstrapping SPSS dialogue (Hayes, 2013, Hayes, 2009). Bootstrapping was performed separately for each of the following age-related subgroups: aged up to 39; over 40; over 50; and over 60. In each bootstrapping model HbA$_{1c}$ (mmol/mol) was entered as the outcome (‘Variable Y’), while PCP status was treated as the predictor (‘Variable X’). BMI and the six IPAQ/physical activity levels, were treated as mediator variables (‘Variables M’). Diabetes status (whether participants had ever had diabetes), blood pressure (doctor-diagnosed), and gender, were treated as control variables (i.e., covariates). The conservative Sobel test was used to determine mediation. Results are shown in Tables 3 and 4.

0 to 39 years. PCP status directly predicted glycaemic control, such that people who had agreed a PCP had higher HbA$_{1c}$ values compared to those without a PCP. Neither BMI nor physical activity mediated this relationship.

Over 40 years. PCP status directly predicted HbA$_{1c}$; patients with a PCP tended to have poorer glycaemic control. This relationship was mediated by BMI, whereby patients who had a care plan had both higher BMI and higher HbA$_{1c}$ values, compared to those without a plan. This depicted a mediator effect because greater BMI was also associated with higher HbA$_{1c}$ levels (see Figure 1). This indirect effect was significant based on the conservative Sobel test ($z = 2.15$, $p <0.05$), and accounted for 13.1% of the total effect of
PCP status on HbA\textsubscript{1c}. Controlling for diabetes status and other covariates attenuated the indirect effect (Sobel test $p > 0.05$), but did not completely abolish it (see Table 3).

Over 50 years. Having a PCP was associated with higher HbA\textsubscript{1c} levels in this group. This association was mediated by BMI, whereby those with a care plan had higher BMI, and poorer glycaemic control, compared to people without a PCP (see Figure 2). The Sobel test for this indirect effect was significant ($z = 2.06, p < 0.05$). The mediator effect accounted for 18.2% of the total effect of PCP status on HbA\textsubscript{1c}. Adjusting for covariates weakened but did not entirely negate the indirect effect (Table 3).

Over 60 years. Although having a PCP predicted higher HbA\textsubscript{1c} values, neither BMI nor physical activity mediated this relationship.

DISCUSSION

Contrary to what was hypothesised patients with a PCP had higher HbA\textsubscript{1c} levels. However, higher BMI scores partly explained this relationship (see Figures 1 and 2). Previous research has implicated BMI in elevated HbA\textsubscript{1c} (Power and Thomas, 2011, Senechal et al., 2013, Patiakas and Charalampous, 2010). Interestingly, there was no evidence implicating physical activity in PCP – HbA\textsubscript{1c} relations, despite previous studies associating exercise with glycaemic control (Umpierre et al., 2011, Gay et al., 2016, Hamer et al., 2014).

Previous research has implicated PCPs in lower HbA\textsubscript{1c} (Coulter et al., 2015). An obvious explanation for the higher HbA\textsubscript{1c} levels observed here is the mediating effect of BMI. It is
possible PCPs may lead to elevated HbA\textsubscript{1c} values, if patients are gaining weight, perhaps due to noncompliance with PCP targets or action plans, and/or other factors, such poor doctor-patient interaction (Paternotte et al., 2015). Previous research shows a strong connection between higher BMI scores and higher HbA\textsubscript{1c}, with one study linking elevated BMI scores in childhood to a 23.9-fold increased risk of a HbA\textsubscript{1c} \geq 7\% later in life (Power and Thomas, 2011). Thus, patients with high HbA\textsubscript{1c}/BMI stand to benefit considerably from information prescriptions (Diabetes UK, 2015a) and other forms of personalised care (Coulter et al., 2013) that specifically target weight control. The fact that BMI mediated the PCP – HbA\textsubscript{1c} relationship specifically in 40 to 60 year olds suggests BMI plays a particularly important role in personalised care and glycaemic control in middle-aged patients (Owen et al., 2015).

Another possible explanation for the higher HbA\textsubscript{1c} levels in patients with PCPs is that care plans tend to be offered to patients with poorer glycaemic control (Diabetes UK, 2015a). Offering PCPs to people with higher HbA\textsubscript{1c} reflects current recommendations that information prescriptions should target individuals at high risk of complications (i.e., high HbA\textsubscript{1c}) (Diabetes UK, 2017). The mediating effect of BMI may simply reflect the fact that patients with high HbA\textsubscript{1c} also tend to have high BMI scores (Power and Thomas, 2011), and/or that GPs are simply more likely to offer PCPs to patients exhibiting both risk factors (Diabetes UK, 2009).

The fact that physical activity did not affect relations between PCPs and HbA\textsubscript{1c} is worrying given that inactivity significantly increases the risk of complications (Waden et al., 2008, Blomster et al., 2013). Evidence from a long-term prospective study associates moderate-to-vigorous levels of activity with a reduced risk of cardiovascular problems, microvascular complications, and premature mortality (Blomster et al., 2013). Although other research suggests no link between exercise and complications (Makura et al., 2013), the availability of population-based prospective data (Blomster et al., 2013) suggests physical
inactivity should be an important factor in personalised care planning and glycaemic control. This seems particularly relevant to middle-aged/older patients. This demographic may find moderate-to-vigorous intensity exercises (e.g., fast cycling, running) particularly challenging, especially if conducted on a regular basis (Sparling et al., 2015), negating the glycaemic benefits (Kennedy et al., 2013). Other factors, such as increased calorie intake, or variations in insulin dosage, may also attenuate the effect of physical activity on HbA\textsubscript{1c}, and should be carefully explored by doctors and patients when setting up PCPs (Kennedy et al., 2013).

This study has some limitations. Firstly, while BMI mediated the PCP – HbA\textsubscript{1c} association, BMI is a poor index of body fat, or morbidity and mortality risk (Nuttall, 2015). Another problem is that data analysis did not control for every covariate relevant to PCP status, BMI, and HbA\textsubscript{1c} (e.g., dietary intake, or insulin resistance). Additionally, there is uncertainty regarding the actual content of PCPs agreed with patients in this data set; due to the personalised nature of PCPs, the HSE does include individual HbA\textsubscript{1c} targets, or recommended lifestyle changes. Furthermore, the HSE data analysed here pre-dates the launch of information prescriptions by Diabetes UK (Diabetes UK, 2015b). As this new personalised care tool is IT-based and can be deployed in a matter of minutes (Diabetes UK, 2017), it’s impact on glycaemic control may be more dramatic than more generic PCP formats (Coulter et al., 2013). Finally, the cross-sectional nature of the design precludes inferences about the possible direction of causality.

This is the first study to examine how BMI and physical activity influence relations between personalised care planning and glycaemic control (Diabetes UK, 2017). The study suggests BMI partly explains higher HbA\textsubscript{1c} levels in patients with a PCP. The irrelevance of physical activity in this context is worrying given recent population-based prospective evidence implicating exercise intensity in diabetes complications (Blomster et al., 2013). These findings are particularly important given the current emphasis on the use of
information prescriptions to improve patient outcomes (Diabetes UK, 2015b). If physical activity level is a precursor for complications (Blomster et al., 2013), then there needs to be some debate amongst doctors/nurses, in partnership with patients, on the role of exercise in personalised care regarding glycaemic control.

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*Archives of Internal Medicine*, 162, 1867-1872.
**Figure 1** Mediating effect of BMI on the PCP – HbA\textsubscript{1c} association in patients aged ≥ 40

\( \begin{array}{c}
\text{Body mass index} \\
\text{(raw score)}
\end{array} \)

- (a-path) 0.57\textsuperscript{a}
- (b-path) 0.42\textsuperscript{c}
- (c-path) 1.57\textsuperscript{a}

PCP status

HbA\textsubscript{1c}

\( ^{a}p<0.05, ^{c}p<0.01, ^{b}p<0.001 \)

**Figure 2** Mediating effect of BMI on the PCP – HbA\textsubscript{1c} association in patients aged ≥ 50

\( \begin{array}{c}
\text{Body mass index} \\
\text{(raw score)}
\end{array} \)

- (a-path) 0.60\textsuperscript{a}
- (b-path) 0.43\textsuperscript{c}
- (c-path) 1.20\textsuperscript{a}

PCP status

HbA\textsubscript{1c}

\( ^{a}p<0.05, ^{c}p<0.01, ^{b}p<0.001 \)
Table 1 – **Descriptive statistics by PCP status.**

<table>
<thead>
<tr>
<th>Variables</th>
<th>No PCP agreed</th>
<th>Agreed PCP &lt; or &gt; 12 months ago</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample size (%)</td>
<td>1458 (87.7%)</td>
<td>204 (12.3%)</td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>57.85 (16.87)</td>
<td>55.08 (15.94)</td>
<td>$t(1660) = 2.25, p&lt;0.05$</td>
</tr>
<tr>
<td>Gender (Male/Female)</td>
<td>638 (86.8%)/820 (88.5%)</td>
<td>97 (13.2%)/107 (11.5%)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>HbA1c (mmol/mol)</td>
<td>39.67 (10.32)</td>
<td>42.07 (13.98)</td>
<td>$t(1660) = -2.36, p&lt;0.05$</td>
</tr>
<tr>
<td>BMI (Body mass index)</td>
<td>28.52 (5.52)</td>
<td>29.13 (6.05)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Diabetes status (currently have, or ever had diabetes) (Yes/No)</td>
<td>172 (11.8%)/1284 (88.2%)</td>
<td>37 (18.1%)/167 (81.9%)</td>
<td>$\chi^2(1) = 6.50, p &lt;0.01$</td>
</tr>
<tr>
<td>High blood pressure – doctor diagnosed (Yes/No)</td>
<td>585 (98%)/12 (2%)</td>
<td>82 (98.8%)/1(1.2%)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes VPA per day</td>
<td>57.62 (106.13)</td>
<td>52.20 (104.47)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes MPA per day</td>
<td>64.00 (103.14)</td>
<td>50.33 (85.31)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes Walking per day</td>
<td>78.18 (99.62)</td>
<td>85.04 (118.45)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes VPA per week</td>
<td>214.06 (542.67)</td>
<td>217.42 (578.30)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes MPA per week</td>
<td>279.64 (581.86)</td>
<td>212.51 (482.57)</td>
<td>Not Significant</td>
</tr>
<tr>
<td>Minutes Walking per week</td>
<td>424.20 (624.99)</td>
<td>456.83 (749.69)</td>
<td>Not Significant</td>
</tr>
</tbody>
</table>

Figures show the mean (+ standard deviation) or count (+ percentage). PCP = Personal care plan (status); MPA = Moderate-intensity activity; VPA = Vigorous-intensity activity; BMI = Body mass index.
Table 2 Bivariate correlations and descriptive statistics

<table>
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<th>(1)</th>
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<th>(3)</th>
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<tbody>
<tr>
<td>1) PCP index</td>
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<tr>
<td>2) Age</td>
<td>-0.057&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td>3) Gender (M/F)</td>
<td>-0.037</td>
<td>0.011</td>
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<tr>
<td>4) HbA&lt;sub&gt;1c&lt;/sub&gt; mmol/ml</td>
<td>0.088&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.297&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.016</td>
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<tr>
<td>5) HBP (doctor)</td>
<td>-0.002</td>
<td>0.381&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.034&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.255&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>6) Diabetes (Y/N)</td>
<td>0.086&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.165&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.022</td>
<td>0.599&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.203&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>7) BMI score</td>
<td>0.047</td>
<td>0.163&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.029</td>
<td>0.249&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.225&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.178&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<td>-</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>8) VPA min p/d</td>
<td>-0.025</td>
<td>-0.111&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.177&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.030</td>
<td>-0.071&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.038&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.049&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
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<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>9) MPA min p/d</td>
<td>-0.041</td>
<td>-0.074&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.135&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.053&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.063&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.061&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.026</td>
<td>0.474&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>10) WK min p/d</td>
<td>0.012</td>
<td>-0.104&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.075&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.057&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.082&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.033</td>
<td>-0.057&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.345&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.361&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>11) VPA min p/w</td>
<td>-0.029</td>
<td>-0.096&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.173&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.023</td>
<td>-0.063&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.041&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.034&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.907&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.454&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.340&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>12) MPA min p/w</td>
<td>-0.057&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.075&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.131&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.039&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.052&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-0.047&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.010</td>
<td>0.443&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.905&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.368&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.485&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>13) WK min p/w</td>
<td>0.002</td>
<td>-0.111&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.073&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.062&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.086&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.036&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.059&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.345&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.369&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.945&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.360&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.404&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>14) ≥ 30 min of MPA/VPA p/wk</td>
<td>-0.020</td>
<td>-0.225&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.106&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.171&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.155&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.116&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-0.138&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.420&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.431&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.164&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.311&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.335&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.156&lt;sup&gt;b&lt;/sup&gt;</td>
<td>-</td>
</tr>
</tbody>
</table>

Note. HBP (High Blood Pressure), BMI (Body Mass Index), VPA (Vigorous-intensity physical activity), MPA (Moderate-intensity physical activity), WK (Walking), p/d (per day), p/w (per week). HBP reflects doctor-diagnosed cases. All physical activity variables denote total number of minutes spent on the specified activity. Superscripts: <sup>a</sup>p < .05, <sup>b</sup>p < .01, <sup>c</sup>p < .001.
Table 3 – *Mediating effects of BMI and physical activity on the PCP – HbA1c association, before and after adjusting for diabetes status and other covariates.*

<table>
<thead>
<tr>
<th>Regression pathways</th>
<th>0-39</th>
<th>Over 40</th>
<th>Over 50</th>
<th>Over 60</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Effect (CI)</td>
<td>Effect (CI)</td>
<td>Effect (CI)</td>
<td>Effect (CI)</td>
</tr>
<tr>
<td><strong>Total effect of PCP on HbA1c</strong></td>
<td>1.58&lt;sup&gt;a&lt;/sup&gt; (0.34, 2.82)</td>
<td>1.84&lt;sup&gt;a&lt;/sup&gt; (0.87, 2.81)</td>
<td>1.46&lt;sup&gt;a&lt;/sup&gt; (0.39, 2.52)</td>
<td>1.60&lt;sup&gt;a&lt;/sup&gt; (0.45, 2.74)</td>
</tr>
<tr>
<td><strong>Direct effect of PCP on HbA1c</strong></td>
<td>1.53&lt;sup&gt;a&lt;/sup&gt; (0.28, 2.78)</td>
<td>1.57&lt;sup&gt;a&lt;/sup&gt; (0.62, 2.53)</td>
<td>1.20&lt;sup&gt;a&lt;/sup&gt; (0.15, 2.24)</td>
<td>1.48&lt;sup&gt;a&lt;/sup&gt; (0.37, 2.59)</td>
</tr>
<tr>
<td><strong>Indirect effect of PCP on HbA1c via:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Minutes VPA per day</td>
<td>0.03 (-0.16, 0.64)</td>
<td>-0.02 (-0.18, 0.02)</td>
<td>-0.03 (-0.22, 0.03)</td>
<td>-0.00 (-0.23, 0.05)</td>
</tr>
<tr>
<td>Minutes MPA per day</td>
<td>-0.28 (-0.84, 0.02)</td>
<td>0.06 (-0.02, 0.23)</td>
<td>0.05 (-0.01, 0.26)</td>
<td>-0.01 (-0.23, 0.04)</td>
</tr>
<tr>
<td>Minutes Walking per day</td>
<td>0.18 (-0.03, 1.16)</td>
<td>0.00 (-0.11, 0.31)</td>
<td>0.00 (-0.09, 0.18)</td>
<td>-0.01 (-0.24, 0.04)</td>
</tr>
<tr>
<td>Minutes VPA past week</td>
<td>-0.01 (-0.48, 0.22)</td>
<td>-0.01 (-0.14, 0.05)</td>
<td>-0.01 (-0.20, 0.04)</td>
<td>-0.00 (-0.15, 0.08)</td>
</tr>
<tr>
<td>Minutes MPA past week</td>
<td>0.29 (-0.02, 0.72)</td>
<td>-0.00 (-0.13, 0.13)</td>
<td>-0.01 (-0.19, 0.09)</td>
<td>0.00 (-0.05, 0.12)</td>
</tr>
<tr>
<td>Minutes Walking past week</td>
<td>-0.18 (-1.01, 0.03)</td>
<td>-0.00 (-0.26, 0.12)</td>
<td>-0.00 (-0.10, 0.07)</td>
<td>-0.01 (-0.18, 0.06)</td>
</tr>
<tr>
<td>BMI (body mass index)</td>
<td>0.02 (-0.10, 0.23)</td>
<td>0.24&lt;sup&gt;a&lt;/sup&gt; (0.03, 0.48)</td>
<td>0.26&lt;sup&gt;a&lt;/sup&gt; (0.05, 0.56)</td>
<td>0.17 (-0.08, 0.49)</td>
</tr>
</tbody>
</table>

<sup>a</sup>p<0.05 or CI range excludes ‘0’. PCP = Personal care plan (status); MPA = Moderate-intensity activity; VPA = Vigorous-intensity activity. BMI = Body mass index.

For simplicity the table does not include the effects of variable X (PCP) on variables M (physical activity, BMI), and effects of variables M on variable Y (HbA1c).
Table 4 – *Mediating effects of BMI on the PCP – HbA1c association, before and after adjusting for diabetes status and other covariates.*

<table>
<thead>
<tr>
<th>Regression pathways</th>
<th>Age groups</th>
<th>Effect</th>
<th>CI</th>
<th>Effect</th>
<th>CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total effect of PCP on HbA1c</td>
<td>Over 40</td>
<td>0.78&lt;sup&gt;a&lt;/sup&gt;</td>
<td>(0.02, 1.54)</td>
<td>0.72</td>
<td>(-0.12, 1.58)</td>
</tr>
<tr>
<td>Direct effect of PCP on HbA1c</td>
<td>Over 50</td>
<td>0.71</td>
<td>(-0.05, 1.47)</td>
<td>0.66</td>
<td>(-0.18, 1.52)</td>
</tr>
<tr>
<td>Indirect effect of PCP on HbA1c via:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI (body mass index)</td>
<td></td>
<td>0.07&lt;sup&gt;a&lt;/sup&gt;</td>
<td>(0.00, 0.18)</td>
<td>0.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>(0.01, 0.25)</td>
</tr>
</tbody>
</table>

<sup>a</sup>p<0.05 or CI range excludes ‘0’. Lower confidence interval for the BMI effect in the ‘over 40 group’ exceeded zero (0.003). For simplicity only the significant mediator variable (BMI) is included here; the table does not include the other M variables.