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THE EFFECT OF LEISURE-TIME PHYSICAL ACTIVITY ON OBESITY, DIABETES, HIGH BP AND HEART DISEASE AMONG CANADIANS: EVIDENCE FROM 2000/2001 TO 2005/2006

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ABSTRACT

Although studies have looked at the effect of physical activity on obesity and other health outcomes, the causal nature of this relationship remains unclear. We fill this gap by investigating the impact of leisure-time physical activity (LTPA) and work-related physical activity (WRPA) on obesity and chronic conditions in Canadians aged 18–75 using instrumental variable and recursive bivariate probit approaches. Average local temperatures surrounding the respondents' interview month are used as a novel instrument to help identify the causal relationship between LTPA and health outcomes. We find that an active level of LTPA (i.e. walking ≥ 1 h/day) reduces the probability of obesity by five percentage points, which increases to 11 percentage points if also combined with some WRPA. WRPA exhibits a negative effect on the probability of obesity and chronic conditions. Copyright © 2014 John Wiley & Sons, Ltd.

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1. INTRODUCTION

Research shows that leisure-time physical activity (LTPA) is associated with lower risks of obesity, preventable chronic diseases and premature mortality (US Department of Health and Human Services, 1996; Craig *et al.*, 2005; Chen and Mao, 2006; Tjepkema, 2006; Warburton *et al.*, 2006; Brown *et al.*, 2007; Ross *et al.*, 2007; Godley and McLaren, 2010). Although interesting, these associations may not be casual because the decision to participate in LTPA and its duration are correlated with unobservable factors like the enjoyment of physical activity, time preference, opportunity cost of time and risk aversion (Komlos *et al.*, 2004; Smith *et al.*, 2005; Norton and Han, 2008). The decision to participate in LTPA may also be correlated with other behaviours like healthy eating. Consequently, physical activity is potentially an endogenous variable in health-related outcome equations, similar to that found between other lifestyle variables and health (Contoyannis and Jones, 2004; Balia and Jones, 2008; Schneider and Schneider, 2012).

If individuals were randomly assigned into different physical activity levels, the limitation of unobservable confounders being correlated with LTPA and health outcomes could be overcome. Such large-scale experiments are

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difficult to conduct. Thus, one has to rely on observational data to estimate the effect of LTPA on obesity and chronic diseases. An instrumental variable (IV) method of estimation can purge endogeneity bias if exogenous instruments are correlated with LTPA but uncorrelated with the error term in the relevant health outcome equations.

To the best of our knowledge, only Humphreys *et al.* (2014) employed such a methodology in their recursive bivariate probit modelling framework. Using self-reported 'sense of belonging to the local community' as an exclusion restriction, they show that participation in LTPA reduces the probability of diabetes, high blood pressure (BP), heart disease, asthma, arthritis and self-reported poor health. However, studies have shown that the 'sense of belonging to the local community' is associated with better health outcomes in Canada (Ross, 2002; Wister and Wanless, 2007; Shields, 2008; Kitchen *et al.*, 2011; Romans *et al.*, 2011) and higher social capital (Wister and Wanless, 2007; Laporte *et al.*, 2008; Kitchen *et al.*, 2011). Thus, the exogeneity of the sense of belonging variable is called into question as it appears to be correlated with both the LTPA and health outcomes. Our study employs a hitherto unexploited exogenous instrument, the monthly average temperatures in the respondents' local neighbourhood, which is highly correlated with LTPA but uncorrelated with the error term in the outcome equations.

Most studies fail to consider work-related physical activities (WRPAs) when examining the effects of LTPA, and vice versa (Fogelholm and Kukkonen-Harjula, 2000; Wareham *et al.*, 2005; Summerbell *et al.*, 2009). For instance, Humphreys *et al.* acknowledge the importance of WRPA but ignore it in their analysis, which may cause specification bias. Our paper examines the effects of both LTPA and WRPA on obesity and chronic diseases; additionally, we look at how the effect of LTPA varies across three distinct levels of WRPA: (i) sedentary; (ii) stand/walk; and (iii) lift light or heavy loads.

Finally, our paper contributes to the existing literature by the use of consistent and high-quality information on LTPA over time. A number of studies rely on a global self-reported physical activity measure that may be a good proxy for participation but ignores the intensity, frequency and duration of physical activity (Wareham *et al.*, 2005). Our study captures these three elements.

2. THEORETICAL MOTIVATION AND EMPIRICAL METHODOLOGY

The theoretical underpinning of this study is based on Grossman's model where rational individuals make decisions about how to allocate their time to produce health in order to maximize lifetime utility, subject to constraints (Grossman, 2000). Accordingly, healthy behaviours, like regular exercise, are regarded as investments in the production of good health. The stock of health, H , is produced by individuals according to the production technology: $H = H(L, E, X, u)$, where L represents lifestyles that include regular exercise and other health-related behaviours, E represents educational status (affecting gross investments in health), X denotes variables influencing either the depreciation rate or the productivity of investments (e.g. age and sex), and u represents individual-specific unobservables affecting the stock of health. In this framework, the opportunity costs of time and preferences will influence decisions regarding exercise, including its intensity and duration (Mullahy and Robert, 2010; Brown and Roberts, 2011; Humphreys and Ruseski, 2011; Maruyama and Yin, 2012). While time spent on physical activity is a source of disutility because of opportunity costs, it may serve to enhance discounted lifetime utility by increasing the availability of healthy days in future periods. Furthermore, the opportunity cost of time will be lower for those who enjoy physical activity (Hatziaandreu *et al.*, 1988; Hagberg and Lindholm, 2010). The key point is that unobservable factors will likely affect both physical activity and health outcomes.

Our empirical specification is similar to the reduced-form model of Humphreys *et al.* (2014). We use three econometric approaches: a univariate probit model, an IV model and a recursive bivariate probit model.

2.1. Univariate probit model

We begin with a reduced-form model of obesity (or chronic disease) a la Humphreys *et al.* (2014), but in which we include WRPA

$$H_i^* = \alpha + \delta_1(LTPA)_i + \delta_2(WRPA)_i + \beta' X_i + \varepsilon_i. \quad (1)$$

For each individual i , H_i^* is the latent health stock, $LTPA_i$ and $WRPA_i$ are as previously defined, X_i is a vector of demographic and socio-economic characteristics and ε_i is the standard disturbance term. We do not observe H_i^* ; instead, we observe $H_i = 1$ if $H_i^* \geq 1$ and 0 otherwise. We consider five dichotomous H_i variables: overweight, obesity, diabetes, high BP, and heart disease. Like Humphreys *et al.*, three variables reflect participation and the intensity of LTPA: (i) daily; (ii) moderate; and (iii) active. Our WRPA measure consists of four dummy variables (including the reference case) based on responses to the question 'thinking back over the past 3 months, which of the following best describes your usual daily activities or work habits? : i) usually sit during the day and don't walk around very much (defined as sedentary) ii) stand or walk quite a lot during the day but don't have to carry or lift things very often (defined as stand/walk), iii) usually lift or carry light loads, or have to climb stairs or hills often (defined as light load), and iv) do heavy work or carry very heavy loads (defined as heavy load)'.

A probit regression model of Eqn (1) provides our baseline analysis. The estimated coefficient, $\hat{\delta}_1$, measures the impact of LTPA on H_i . Because LTPA is a dichotomous variable, its average partial effect is the sample average of changes in the predicted probability of being obese or having a chronic condition with discrete changes in LTPA, while evaluating all other variables, X_i , at their observed values. It is computed as

$$\frac{1}{n} \sum_{i=1}^n \left[\Phi \left(\hat{\beta}' X_i + \hat{\delta}_2(WRPA)_i + \hat{\delta}_1(LTPA)_i | LTPA = 1 \right) - \Phi \left(\hat{\beta}' X_i + \hat{\delta}_2(WRPA)_i + \hat{\delta}_1(LTPA)_i | LTPA = 0 \right) \right], \quad (2)$$

where Φ is the standard normal distribution function and $\Phi \left(\hat{\beta}' X + \hat{\delta}_1 \hat{LTPA} + \hat{\delta}_2 \hat{WRPA} \right)$ is the marginal predicted probability of participating in LTPA (or the intensity of LTPA) and is computed for each observation using the estimated coefficients from Eqn (1); n is the sample size. The average partial effects of WRPA are similarly computed.

2.2. Instrumental variable model

The main drawback of the univariate probit model is that the decision to participate in LTPA or its intensity is unlikely to be exogenous as unobservable factors like the enjoyment of physical activity, time preference and the opportunity cost of time are likely to be correlated with both LTPA and H_i .¹ Allowing LTPA to be endogenous, we respecify Eqn (1) as

$$H_i^* = \alpha + \delta_1(LTPA^*)_i + \delta_2(WRPA)_i + \beta' X_i + \varepsilon_i, \quad (3)$$

where $LTPA^*_i$ is the latent variable determining physical activity for individual i (participation or intensity). Thus,

$$LTPA_i^* = \eta + \lambda' Z_i + \gamma' X_i + u_i, \quad (4)$$

where Z_i is the vector of characteristics that influence participation in physical activity but are uncorrelated with ε_i , η is the intercept term, and u_i is the error term. We are interested in the consistent estimate of the δ s. The observed realization of the latent variable $LTPA^*_i$ takes the following form: $LTPA_i = 1$ if $LTPA^*_i \geq 1$ and 0 otherwise.

A common approach to estimating Eqns (3)–(4) is a linear IV procedure if an exogenous instrument can be found that is correlated with LTPA but uncorrelated with $\varepsilon_i | X$ (Imbens and Angrist, 1994; Angrist *et al.*, 1996). A linear IV generally yields consistent estimates for the local average treatment effect (Imbens and Angrist, 1994; Angrist *et al.*, 1996).

¹WRPA is more likely to be exogenous as it is primarily governed by educational attainment and ability. While it is possible that some people may avoid physically demanding jobs because of particular health conditions, for the most part, one expects education and ability to be the driving forces behind occupational choice.

Researchers typically rely on theoretical justifications and statistical tests for the validity of instruments because the exogeneity assumption is not directly testable. The theoretical basis for our instrument is that an individual's LTPA decisions are governed by unobservable preferences, like the enjoyment of physical activity, which is partly determined by local weather conditions. With large seasonal variations in temperatures across Canada, the average monthly temperature in a respondent's local neighbourhood is likely to be correlated with LTPA and uncorrelated with $\varepsilon_i|X_i$ as levels of LTPA will fluctuate with changes in temperature. Indeed, a systematic review finds that physical activity levels are considerably lower during colder months in countries with extreme weather (Tucker and Gilliland, 2007). Merchant *et al.* (2007) report that 64% of Canadians are inactive in the winter compared with 49% in the summer. Studies also suggest that weather accounts for over 40% of all measured physical activity (Tucker and Gilliland, 2007). Thus, variations in the local temperature provide a natural setting to study the effects of physical activity on health outcomes. Because our LTPA variable is constructed based on physical activity over a 3-month period, we use corresponding local temperature data by period for each respondent as our instruments.²

The Canadian Community Health Survey (CCHS) confidential master files provide access to six-digit residential postal codes and the date of respondent interviews. Monthly weather data for over 1200 weather stations with longitude and latitude coordinates are compiled by Environment Canada and publicly available.³ After assigning a local weather station to each respondent, we link local temperatures back 3 months beginning with the interview month.⁴ We restrict our analysis to those observations with a weather station within a 0.5 degree distance (about 55 km) from the respondent's home based on the centroid of his/her six-digit postal code.⁵ Because CCHS data were collected over 12 months, large exogenous variations in temperatures around the interview months were found and are used as the source of identification as average temperatures vary considerably in our data from +25°C to -33°C (Appendix Table A1 in Supporting Information).

The IV approach depends on the validity and relevance of the identifying instruments. The validity of the overidentifying restrictions is tested using Hansen's *J* statistic (Baum *et al.*, 2007). We also test whether the IV estimates suffer from weak or irrelevant instruments (Bound *et al.*, 1995); if the *F*-statistic in the first stage is less than 10 (for a single endogenous variable and one instrument), the instrument is weak (Staiger and Stock, 1997). Stock and Yogo (2005) developed a formal weak-identification *F*-statistic, and a robust counterpart was subsequently developed by Kleibergen and Paap (2006) to examine the bias associated with the IV estimator. These test statistics are compared with the critical values tabulated by Stock and Yogo (2005). Various tests confirm that our instruments satisfy the identification requirements, rejecting the hypothesis that our instruments are weak with a few exceptions.

2.3. Recursive bivariate probit model

Although the linear IV method is consistent for estimating the local average treatment effect, it is generally biased and may be inferior to a correctly specified bivariate probit model under certain circumstances. Altonji *et al.* (2005) found that the linear IV model produced large coefficients and standard errors compared with the bivariate probit model. Some argue that a correctly specified bivariate probit model is superior to the IV procedure, especially if the error term is non-normal (Bhattacharya *et al.*, 2006; Freedman and Sekhon, 2010). Several papers in the health economics literature have utilized recursive bivariate probit models to study the relationship between lifestyle variables and health status (Contoyannis and Jones, 2004; Balia and Jones, 2008; Schneider and Schneider, 2012) and physical activity and health outcomes (Humphreys *et al.*, 2014).

² Respondents were asked about details of their physical activities in the past 3 months. When the fourth month average temperature was included, it often failed to have explanatory power, suggesting that temperature does indeed affect LTPA.

³ http://climate.weatheroffice.gc.ca/prods_servs/cdn_climate_summary_e.html

⁴ If a respondent was interviewed on January 21, 2001, we link the average temperatures in January 2001, December 2000 and November 2000 from the nearest weather station to this person.

⁵ More than 87% of respondents were assigned to a local weather station with consistent temperature data over a 3-month period. Sensitivity analysis suggested that our results are unaffected within a 0.3 degree (about 33 km) to 1 degree (about 111 km) range.

Identification in a bivariate probit model rests on exclusion restrictions—that is, excluding at least one variable from the structural equation and including it in the reduced-form equation (Maddala, 1983). Wilde (2000) argues that it is necessary to have at least one varying exogenous regressor in order to avoid identification problems, although exclusion restrictions as such are not required to estimate a bivariate probit model. Conceptually, we think that exclusion restrictions are superior to having an irrelevant exogenous variable or to having identification by functional form. Our instrument constitutes the exclusion restriction. The recursive bivariate probit model is specified as per Eqns (3)–(4) with the additional assumption that $Cov(\varepsilon, u) = \rho$; ρ is interpreted as the conditional tetrachoric correlation between H and $LTPA$. Average

Table I. Variable definitions

Variable	Definition
Overweight	Bias-corrected BMI $\geq 25 \text{ kg/m}^2 = 1$, 0 if bias-corrected BMI is greater than or equal to 18.5 kg/m^2 and less than 25 kg/m^2
Obese	Bias-corrected BMI $\geq 30 \text{ kg/m}^2 = 1$, 0 if bias-corrected BMI is greater than or equal to 18.5 kg/m^2 and less than 25 kg/m^2
Diabetes	Reported diagnosis of diabetes = 1, otherwise = 0
High BP	Reported diagnosis of high BP = 1, otherwise = 0
Heart disease	Reported diagnosis of heart disease = 1, otherwise = 0
Participation	Daily average LTPA lasting more than 15 min = 1, otherwise = 0
Moderate	Average daily energy expenditure on LTPA per kilogram of body weight per day (LTPA_EE) $\geq 1.5 \text{ kcal/kg/day}$ (i.e. walk 30–50 min) = 1, LTPA_EE $< 1.5 \text{ kcal/kg/day}$ (i.e. walk $< 30 \text{ min}$) = 0
Active	LTPA_EE $\geq 3.0 \text{ kcal/kg/day}$ (i.e. walk $\geq 1 \text{ h}$) = 1, LTPA_EE $< 1.5 \text{ kcal/kg/day}$ (i.e. walk $< 30 \text{ min}$) = 0
Sedentary	Usually sit during the day and do not walk very much = 1, otherwise = 0
Stand/Walk	Usually stand or walk quite a lot during the day but do not have to carry or lift things very often = 1, otherwise = 0
Light loads	Usually lift or carry light or heavy loads or have to climb stairs or hills often = 1, otherwise = 0
Heavy loads	Usually do heavy work or carry very heavy loads = 1, otherwise = 0
Female	Female = 1, male = 0
Age	Age in completed years
Age squared	Age squared
Married	Married or common law relationship = 1, single = 0
WSD	Widow, separated or divorced = 1, single = 0
Immigrant ≤ 10 years	Immigrated to Canada less than or equal to 10 years = 1, Canadian born = 0
Immigrant > 10 years	Immigrated to Canada more than 10 years ago = 1, Canadian born = 0
$< \text{Secondary}$	Less than secondary school = 1, otherwise = 0
Secondary	Secondary school graduation = 1, otherwise = 0
$< \text{Post-secondary}$	Some post-secondary education = 1, otherwise = 0
Post-secondary	College or university degree = 1, otherwise = 0
Children < 6 years	Children in the household aged less than 6 years = 1, otherwise = 0
Children < 12 years	Children in the household aged 6 years or more but less than 12 years, otherwise = 0
Employed	Full-time or part-time employed = 1, otherwise = 0
Home owner	Household owned a home with or without mortgage = 1, otherwise = 0
Income: $< 20 \text{ k}$	Household income less than \$20 000 = 1, otherwise = 0
Income: 20–50 k	Household income greater than \$20 000 but less than \$50 000 = 1, otherwise = 0
Income: 50–80 k	Household income greater than \$50 000 but less than \$80 000 = 1, otherwise = 0
Income: $> 80 \text{ k}$	Household income greater than \$80 000 = 1, otherwise = 0
Income: missing	Household income missing = 1, otherwise = 0
Urban	Living in urban area = 1, rural area = 0
NFL	Province: Newfoundland = 1, otherwise = 0
PEI	Province: Prince Edward Island = 1, otherwise = 0
NS	Province: Nova Scotia = 1, otherwise = 0
NB	Province: New Brunswick = 1, otherwise = 0
QUE	Province: Quebec = 1, otherwise = 0
ON	Province: Ontario = 1, otherwise = 0
MAN	Province: Manitoba = 1, otherwise = 0
SAS	Province: Saskatchewan = 1, otherwise = 0
AL	Province: Alberta = 1, otherwise = 0
BC	Province: British Columbia = 1, otherwise = 0
Territories	Province: Yukon, Northwest Territories, or Nunavut = 1, otherwise = 0
Cycle 1	Equal to 1 if surveyed in 2000/2001, 0 otherwise
Cycle 2	Equal to 1 if surveyed in 2003/2004, 0 otherwise
Cycle 3	Equal to 1 if surveyed in 2005/2006, 0 otherwise

Table II. Average partial effects—overweight (full sample)

Variable	Probit estimates ^a	Probit estimates	Linear IV estimates	Bivariate probit estimates
Daily	−.030*** (.003)	−.028*** (.003)	−.040 (.025)	−.050* (.026)
Stand/Walk		−.028*** (.004)	−.026*** (.004)	−.025*** (.004)
Light load		−.032*** (.004)	−.032*** (.005)	−.031*** (.005)
Heavy load		−.007 (.006)	−.007 (.006)	−.006 (.006)
Observations	270 328	269 506	243 061	243 061
Estimated ρ				.04
Moderate	−.028*** (.003)	−.026*** (.003)	−.032 (.020)	−.029*** (.020)
Stand/Walk		−.028*** (.004)	−.026*** (.004)	−.027*** (.004)
Light load		−.032*** (.004)	−.032*** (.005)	−.033*** (.005)
Heavy load		−.008 (.006)	−.006 (.006)	−.008 (.006)
Observations	270 328	269 506	243 061	243 601
Estimated ρ				.04
Active	−.044*** (.003)	−.041*** (.004)	−.043** (.022)	−.012 (.021)
Stand/Walk		−.032*** (.004)	−.030*** (.005)	−.033*** (.005)
Light load		−.036*** (.005)	−.036*** (.006)	−.040*** (.006)
Heavy load		−.011* (.007)	−.011 (.008)	−.015** (.007)
Observations	201 038	200 408	180 887	180 887
Estimated ρ				−.05
Relevant IV tests		Daily	Moderate	Active
Hansen J statistic		3.93	3.86	2.87
p -value		0.14	0.14	0.24
Kleibergen–Paap rk Lagrange multiplier statistic ^a		1645.6***	2221.1***	2488.4***
Cragg–Donald F statistic		1314.7	1856.7	1983.4
Kleibergen–Paap rk Wald F statistic ^a		561.57	768.0	871.4

Note: All regressions include the full set of control variables, X_i , defined on pages 13–14.

^aExcludes WRPA.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Critical values for Cragg–Donald Wald F statistic: 5% maximal IV relative bias: 13.91; 10% maximal IV size: 22.30.

partial effects are obtained similar to that for the probit model using the estimated parameters from the recursive bivariate probit models.

3. DATA AND VARIABLES

Data for this study come from three biennial confidential master files of the CCHS, Cycles 1.1 (2000/01), 2.1 (2003/04) and 3.1 (2005/06), conducted by Statistics Canada. Each cycle is a representative survey of more than 130 000 individuals aged 12 years or older living in all provinces and territories in Canada, except those living on Crown lands, Indian reserves, Canadian Forces bases, institutions and some remote areas. The surveys were conducted by highly skilled interviewers, with response rates of over 90%. Our sample is restricted to those aged 18 to 75 years. Excluding missing socio-demographic variables other than household income resulted in 315 833 valid observations. Sample sizes for the different regression analyses vary by the type of outcome and the chosen sub-sample criteria. All descriptive and regression analyses are weighted using the sampling weights provided by Statistics Canada.

The CCHS contains information on self-reported weight and height for all respondents 18 years and older, excluding pregnant women. However, self-reported height and weight, and hence the body mass index (BMI),⁶ are likely to be biased because individuals generally tend to overreport their height and underreport their weight. Thus, the sex-specific correction factors proposed by Connor *et al.* (2008) were applied. Overweight has $BMI \geq 25$, normal has $18.5 \leq BMI < 25$, and obese is $BMI \geq 30$.

⁶BMI is weight in kilograms divided by height in metres squared.

The intensity of LTPA is measured by the average daily energy expended on all leisure activities undertaken over a 3-month period. A list of LTPA options was provided to survey participants to indicate how many times they performed each activity, and average duration, over the past 3 months. Energy expenditure for each respondent is then calculated as $LTPA_EE = \sum_i (N_i * D_i * MET) / 365$, where N_i is the number of times engaged in activity i during past 12 months, D_i is its average duration in hours, and MET is the metabolic energy cost of the activity (a multiple of the resting metabolic rate). The MET values are of three intensity levels: high, medium and low. Because the CCHS did not collect information on the intensity of activities from the respondents, Statistics Canada adopted the low-intensity value for each activity, consistent with the guidelines of the *Canadian Fitness and Lifestyle Research Institute*: www.cflri.ca. This approach seems reasonable as it is well known that individuals tend to overstate activity intensity when surveyed. $LTPA_EE$ is expressed as kilocalories expended per kilogram of body weight per day (kcal/kg/day). A person is considered physically inactive if the daily leisure-time energy expenditure is less than 1.5 kcal/kg/day, moderately active if the energy expenditure is 1.5–2.9 kcal/kg/day, and active if the energy expenditure is 3.0 kcal/kg/day or more. Binary variables capture these levels. Practically speaking, physical inactivity refers to walking less than 30 min daily; moderate activity is walking 30–59 min daily, while walking 60 min or more is an ‘active’ person.

A wide variety of demographic and socio-economic variables are included in all regression models. Table I provides the detailed definitions of all variables used in this paper, and the corresponding descriptive statistics are reported in Appendix B in Supporting Information.

4. RESULTS

Because the effects of physical activity on obesity and chronic conditions are our primary focus, we present the results of LTPA and WRPA. Given space constraints, descriptive results and analyses are presented in

Table III. Average partial effects—obese (full sample)

Variable	Probit estimates ^a	Probit estimates	Linear IV estimates	Bivariate probit estimates
Daily	−.070*** (.004)	−.066*** (.004)	−.034 (.032)	−.057* (.030)
Stand/Walk		−.051*** (.004)	−.053*** (.005)	−.050*** (.005)
Light load		−.058*** (.004)	−.065*** (.006)	−.060*** (.006)
Heavy load		−.026*** (.007)	−.032*** (.008)	−.028*** (.008)
Observations	166 791	166 284	150 016	150 016
Estimated ρ				−.02
Moderate	−.073*** (.003)	−.068*** (.003)	−.028 (.025)	−.025 (.025)
Stand/Walk		−.050*** (.004)	−.053*** (.005)	−.052*** (.005)
Light load		−.057*** (.004)	−.065*** (.006)	−.064*** (.006)
Heavy load		−.026*** (.007)	−.033*** (.008)	−.031*** (.008)
Observations	166 791	166 284	150 016	150 016
Estimated ρ				−.08*
Active	−.108*** (.004)	−.101*** (.004)	−.065** (.027)	−.053** (.027)
Stand/Walk		−.056*** (.005)	−.060*** (.006)	−.060*** (.006)
Light load		−.062*** (.006)	−.070*** (.007)	−.069*** (.007)
Heavy load		−.034*** (.008)	−.045*** (.009)	−.043*** (.009)
Observations	124 744	124 351	112 328	112 328
Estimated ρ				−.09*
Relevant IV tests		Daily	Moderate	Active
Hansen J statistic		9.42	9.3	4.83
p -value		0.01	0.01	0.09
Kleibergen–Paap rk Lagrange multiplier statistic ^a		993.0***	1377.1***	1511.4***
Cragg–Donald F statistic		784.15	1144.9	1191.9
Kleibergen–Paap rk Wald F statistic ^a		337.9	475.1	525.2

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

^aExcludes WRPA.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Critical values for Cragg–Donald Wald F statistic: 5% maximal IV relative bias: 13.91; 10% maximal IV size: 22.30.

Table IV. Average partial effects—diabetes (full sample)

Variable	Probit estimates ^a	Probit estimates	Linear IV estimates	Bivariate probit estimates
Daily	−.004*** (.001)	−.002* (.001)	.015 (.010)	.005 (.007)
Stand/Walk		−.012*** (.001)	−.017*** (.002)	−.013*** (.002)
Light load		−.018*** (.002)	−.024*** (.002)	−.019*** (.002)
Heavy load		−.019*** (.002)	−.024*** (.002)	−.020*** (.003)
Observations	299 159	298 250	268 825	268 825
Estimated ρ				−.06
Moderate	−.010*** (.001)	−.008*** (.001)	.012 (.008)	.007 (.007)
Stand/Walk		−.011*** (.001)	−.017*** (.002)	−.013*** (.002)
Light load		−.017*** (.002)	−.024*** (.002)	−.019*** (.002)
Heavy load		−.018*** (.002)	−.023*** (.002)	−.020*** (.003)
Observations	299 159	298 250	268 825	268 825
Estimated ρ				−.12***
Active	−.014*** (.001)	−.011*** (.001)	.016* (.009)	.008 (.007)
Stand/Walk		−.012*** (.001)	−.021*** (.002)	−.015*** (.002)
Light load		−.018*** (.002)	−.028*** (.002)	−.022*** (.002)
Heavy load		−.020*** (.003)	−.027*** (.002)	−.022*** (.003)
Observations	223 257	222 557	200 723	200 723
Estimated ρ				−.14***
Relevant IV tests		Daily	Moderate	Active
Hansen J statistic		3.67	3.7	5.2
p -value		0.16	0.16	0.07
Kleibergen–Paap rk Lagrange multiplier statistic ^a		1794.1***	2394.2***	2675.6***
Cragg–Donald F statistic		1437.0	2004.4	2143.9
Kleibergen–Paap rk Wald F statistic ^a		612.7	827.7	936.4

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

^aExcludes WRPA.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Critical values for Cragg–Donald Wald F statistic: 5% maximal IV relative bias: 13.91; 10% maximal IV size: 22.30.

Appendix C in Supporting Information. The estimated average partial effects on the probability of being overweight or obese or having diabetes, high BP or heart disease are presented in Tables II–VI respectively.⁷ Each table presents the results of four regression models: Column 2 presents the probit estimates omitting WRPA, column 3 presents the probit estimates including WRPA, column 4 presents the linear IV estimates, and finally, column 5 reports the results from the recursive bivariate probit model. The linear IV and recursive bivariate probit without WRPA estimates are presented in Appendix D in Supporting Information. Note that the probability of overweight refers to overweight or obesity, and the probability of obesity refers only to obesity in our analysis, where the comparison group is normal weight in both cases.

4.1. Univariate probit results

From column 2 of Tables II–VI, the estimated average partial effects of daily, moderate and active levels without controlling for WRPA are slightly higher than otherwise, suggesting that failing to control for WRPA may bias upwards the associations between LTPA and health outcomes. Previous literature that has ignored the influence of WRPA on obesity or chronic conditions may overstate the impact of LTPAs on weight and other chronic conditions.

After controlling for WRPA (column 3 of Tables II–VI), we see that both LTPA and WRPA exert a negative influence on the probability of being overweight and obese and having chronic conditions. Each of daily, moderate and active levels of LTPA participation is associated with a 2.8, 2.6 and 4.1% decrease in the probability of being overweight and 6.6, 6.8 and 10.1% decrease in the probability of obesity respectively. Compared with sedentary WRPA, being able to stand or walk as well as lift light loads is associated

⁷Detailed regression results are available from the corresponding author upon request.

Table V. Average partial effects—high BP (full sample)

Variable	Probit estimates ^a	Probit estimates	Linear IV estimates	Bivariate probit estimates
Daily	−.014*** (.002)	−.012*** (.002)	.020 (.016)	.010 (.013)
Stand/Walk		−.015*** (.002)	−.020*** (.003)	−.018*** (.003)
Light load		−.027*** (.003)	−.033*** (.003)	−.030*** (.003)
Heavy load		−.020*** (.004)	−.024*** (.004)	−.022*** (.005)
Observations	298 867	297 960	268 571	268 571
Estimated ρ				−.08*
Moderate	−.021*** (.002)	−.019*** (.002)	.016 (.013)	.014 (.012)
Stand/Walk		−.014*** (.002)	−.020*** (.003)	−.018*** (.003)
Light load		−.026*** (.003)	−.033*** (.003)	−.031*** (.003)
Heavy load		−.019*** (.004)	−.024*** (.004)	−.022*** (.005)
Observations	298 867	297 960	268 571	268 571
Estimated ρ				−0.12***
Active	−.030*** (.002)	−.028*** (.002)	.013 (.013)	.012 (.011)
Stand/Walk		−.015*** (.002)	−.021*** (.003)	−.019*** (.003)
Light load		−.025*** (.003)	−.033*** (.003)	−.031*** (.003)
Heavy load		−.019*** (.005)	−.027*** (.004)	−.025*** (.005)
Observations	223 043	222 346	200 539	200 539
Estimated ρ				−0.14***
Relevant IV tests		Daily	Moderate	Active
Hansen J statistic		0.61	0.61	0.10
p -value		0.74	0.74	0.95
Kleibergen–Paap rk Lagrange multiplier statistic ^a		1793.4***	2394.8***	2675.0***
Cragg–Donald F statistic		1436.4	2004.4	2143.6
Kleibergen–Paap rk Wald F statistic ^a		612.5	827.8	936.3

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

^aExcludes WRPA.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Critical values for Cragg–Donald Wald F statistic: 5% maximal IV relative bias: 13.91; 10% maximal IV size: 22.30.

with a decrease in the probability of being overweight of three percentage points and a reduction in the probability of obesity by five to six percentage points. However, lifting heavy loads is associated with about a three percentage point decrease in the probability of obesity, but it is not statistically significant for individuals who are overweight.

Participation in LTPA is also negatively associated with the probability of chronic conditions, with daily participation associated with a decrease in the probability of diabetes, high BP and heart disease by 0.2, 1.2 and 0.4% respectively; a moderate level of LTPA is associated with a decrease of 0.8, 1.9 and 0.6% respectively, and an active level of LTPA is associated with decreases of 1.1, 2.8 and 0.8% respectively. Being able to stand or walk and being able to lift light loads and heavy loads are associated with a decrease in the probability of diabetes, high BP and heart disease in the range of 1–2% when compared with sedentary WRPA.

The results of the probit models suggest that both LTPA and WRPA tend to reduce the probability of being overweight or obese or having diabetes, high BP and heart disease. However, the key question is as follows: Can these results be interpreted as causal effects? We now turn to the linear IV and recursive bivariate probit results that account for endogeneity bias and hence allow us to ascertain causal effects. We find that taking into consideration the unobservable factors affecting an individual's participation in or intensity of physical activity has a large impact on the resulting estimates.

4.2. Linear IV and recursive bivariate probit results

A discussion of the identification tests for our instruments is presented in Appendix E in Supporting Information. Contrary to the probit results, the linear IV results show that daily and moderate LTPA does not reduce the probability of being overweight and of obesity. These results are largely confirmed by the recursive bivariate probit models except that a moderate level reduces the probability of being overweight by 2.9%, and the effect

Table VI. Average partial effects—heart disease (full sample)

Variable	Probit estimates ^a	Probit estimates	Linear IV estimates	Bivariate probit estimates
Daily	−.005*** (.001)	−.004*** (.001)	−.004 (.009)	−.004 (.001)
Stand/Walk		−.008*** (.001)	−.013*** (.002)	−.010*** (.001)
Light load		−.015*** (.001)	−.020*** (.002)	−.014*** (.002)
Heavy load		−.017*** (.003)	−.020*** (.002)	−.017*** (.003)
Observations	299 062	298 154	268 734	268 734
Estimated ρ				0.04
Moderate	−.008*** (.001)	−.006*** (.001)	−.003 (.008)	−.007 (.007)
Stand/Walk		−.008*** (.001)	−.013*** (.002)	−.009*** (.001)
Light load		−.014*** (.001)	−.020*** (.002)	−.014*** (.002)
Heavy load		−.017*** (.002)	−.020*** (.002)	−.017*** (.003)
Observations	299 062	298 154	268 734	268 734
Estimated ρ				−.005
Active	−.010*** (.001)	−.008*** (.001)	.002 (.008)	−.007 (.007)
Stand/Walk		−.008*** (.001)	−.014*** (.002)	−.009*** (.002)
Light load		−.015*** (.002)	−.021*** (.002)	−.015*** (.002)
Heavy load		−.017*** (.003)	−.021*** (.002)	−.017*** (.003)
Observations	223 184	222 486	200 657	200 657
Estimated ρ				−.02
Relevant IV tests		Daily	Moderate	Active
Hansen J statistic		2.2	2.24	2.1
p -value		0.33	0.33	0.35
Kleibergen–Paap rk Lagrange multiplier statistic ^a		1795.0***	2394.8***	2678.3***
Cragg–Donald F statistic		1437.9	2005.8	2146.4
Kleibergen–Paap rk Wald F statistic ^a		613.0	828.2	937.4

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

^aExcludes WRPA

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Critical values for Cragg–Donald Wald F statistic: 5% maximal IV relative bias: 13.91; 10% maximal IV size: 22.30.

Table VIIa. Average partial effects—probit estimates (by WRPA status)

WRPA/LTPA	Overweight	Obese	Diabetes	High BP	Heart disease
WRPA: sedentary					
Daily	−.033*** (.006)	−.080*** (.008)	−.002 (.003)	−.015*** (.004)	−.008*** (.003)
Observations	61 201	38 953	68 390	68 315	68 370
Moderate	−.037*** (.006)	−.091*** (.007)	−.013*** (.003)	−.023*** (.004)	−.009*** (.002)
Observations	61 201	38 953	68 390	68 315	68 370
Active	−.047*** (.008)	−.123*** (.010)	−.019*** (.004)	−.037*** (.006)	−.016*** (.004)
Observations	46 690	29 984	52 710	52 652	52 689
WRPA: stand/walk					
Daily	−.033*** (.004)	−.065*** (.005)	−.001 (.002)	−.011*** (.003)	−.002 (.002)
Observations	116 350	71 834	130 444	130 348	130 390
Moderate	−.028*** (.004)	−.064*** (.005)	−.008*** (.002)	−.020*** (.003)	−.006*** (.002)
Observations	116 350	71 834	130 444	130 348	130 390
Active	−.046*** (.005)	−.101*** (.007)	−.011*** (.002)	−.028*** (.003)	−.006*** (.002)
Observations	85 208	52 886	95 695	95 618	95 659
WRPA: lift light/ heavy load					
Daily	−.017*** (.005)	−.053*** (.006)	−.002 (.002)	−.009*** (.003)	−.0006 (.001)
Observations	91 955	55 497	99 416	99 297	99 394
Moderate	−.013*** (.005)	−.050*** (.006)	−.002 (.002)	−.013*** (.003)	−.002 (.001)
Observations	91 955	55 497	99 416	99 297	99 394
Active	−.028*** (.006)	−.082*** (.007)	−.004*** (.002)	−.019*** (.003)	−.002 (.002)
Observations	68 510	41 481	74 152	74 076	74 138

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

Table VIIb. Estimated coefficients—linear IV estimates (by WRPA status)

WRPA/LTPA	Overweight	Obese	Diabetes	High BP	Heart Disease
WRPA: sedentary					
Daily	.044 (.060)	.089 (.079)	.032 (.026)	.046 (.038)	.037 (.025)
Observations	55 044	35 043	61 489	61 428	61 470
Hansen <i>J</i> statistic	3.09 [0.21]	6.83 [0.03]	3.1 [0.21]	1.4 [0.50]	1.9 [0.38]
K–P rk Lagrange multiplier (LM) statistic	316.4***	180.9***	331.5***	331.6***	331.1***
Cragg–Donald <i>F</i> statistic	254.0	143.9	267.7	267.8	267.4
K–P rk Wald <i>F</i> statistic	108.1	61.54	113.0	113.0	112.8
Moderate	.037 (.050)	.072 (.063)	.025 (.021)	.037 (.032)	.031 (.020)
Observations	55 044	35 043	61 489	61 428	61 470
Hansen <i>J</i> statistic	3.1 [0.21]	6.8 [0.03]	3.2 [0.20]	1.5 [0.48]	1.8 [0.41]
K–P rk LM statistic	389.0***	232.0***	404.8***	404.5***	404.5***
Cragg–Donald <i>F</i> statistic	324.8	193.2	337.0	336.5	336.7
K–P rk Wald <i>F</i> statistic	133.6	80.1	138.8	138.6	138.6
Active	.031 (.055)	.054 (.071)	.050* (.026)	.039 (.036)	.036 (.024)
Observations	42 145	27 091	47 543	47 498	47 522
Hansen <i>J</i> statistic	7.3 [0.03]	8.9 [0.01]	3.0 [0.22]	1.9 [0.38]	1.1 [0.58]
K–P rk LM statistic	416.2***	252.3***	429.2***	429.3***	428.8***
Cragg–Donald <i>F</i> statistic	349.5	206.7	364.1	364.5	364.0
K–P rk Wald <i>F</i> statistic	145.1	87.9	148.9	149.0	148.7
WRPA: stand/walk					
Daily	–.064* (.038)	–.068 (.048)	.008 (.015)	.004 (.025)	–.020 (.015)
Observations	104 875	64 722	117 515	117 431	117 464
Hansen <i>J</i> statistic	0.7 [0.69]	3.4 [0.18]	1.8 [0.42]	0.8 [0.67]	0.9 [0.65]
K–P rk LM statistic	686.0***	394.4***	753.3***	753.4***	753.7***
Cragg–Donald <i>F</i> statistic	559.1	319.0	616.0	616.2	616.5
K–P rk Wald <i>F</i> statistic	233.8	134.4	257.2	257.2	257.3
Moderate	–.054* (.031)	–.056 (.040)	.006 (.013)	.004 (.020)	–.016 (.012)
Observations	104 875	64 722	117 515	117 431	117 464
Hansen <i>J</i> statistic	0.6 [0.74]	3.3 [0.19]	1.8 [0.40]	0.8 [0.68]	0.9 [0.65]
K–P rk LM statistic	943.1***	562.6***	1020.1***	1021.4***	1020.3***
Cragg–Donald <i>F</i> statistic	793.4	472.8	864.7	865.8	865.0
K–P rk Wald <i>F</i> statistic	326.5	194.2	353.1	353.5	353.1
Active	–.072** (.032)	–.112*** (.040)	–.008 (.013)	–.002 (.021)	–.004 (.013)
Observations	76 793	47 672	86 173	86 105	86 140
Hansen <i>J</i> statistic	0.6 [0.75]	0.5 [0.76]	1.9 [0.38]	1.7 [0.43]	0.5 [0.79]
K–P rk LM statistic	1097.1***	631.3***	1181.4***	1181.5***	1181.4***
Cragg–Donald <i>F</i> statistic	873.7	496.8	947.1	947.4	947.4
K–P rk Wald <i>F</i> statistic+	386.3	219.4	415.5	415.6	415.6
WRPA: lift light/ heavy load					
Daily	–.062 (.040)	–.058 (.048)	.022* (.012)	.031 (.022)	–.001 (.012)
Observations	83 142	50 251	89 821	89 712	89 800
Hansen <i>J</i> statistic	1.5 [0.48]	1.3 [0.53]	2.0 [0.36]	9.0 [0.01]	3.4 [0.19]
K–P rk LM statistic	659.0***	433.8***	736.9***	735.5***	737.8***
Cragg–Donald <i>F</i> statistic	503.6	324.8	563.3	561.9	564.0
K–P rk Wald <i>F</i> statistic	225.2	147.5	252.6	252.1	252.9
Moderate	–.050 (.032)	–.046 (.038)	.018* (.010)	.023 (.018)	–.002 (.010)
Observations	83 142	50 251	89 821	89 712	89 800
Hansen <i>J</i> statistic	1.5 [0.46]	1.3 [0.52]	1.9 [0.39]	9.2 [0.01]	3.3 [0.19]
K–P rk LM statistic	920.6***	615.1***	1017.9***	1016.9***	1019.7***
Cragg–Donald <i>F</i> statistic	752.2	494.8	831.2	830.1	832.6
K–P rk Wald <i>F</i> statistic	319.9	211.9	354.7	354.3	355.3

(Continues)

Table VIIb. (Continued)

WRPA/LTPA	Overweight	Obese	Diabetes	High BP	Heart Disease
Active	-.049 (.033)	-.072* (.040)	.015 (.010)	.024 (.018)	-.003 (.010)
Observations	61 949	37 565	67 007	66 936	66 995
Hansen <i>J</i> statistic	0.5 [0.78]	0.5 [0.78]	2.3 [0.31]	7.1 [0.03]	3.5 [0.17]
K–P rk LM statistic	1005.6***	653.1***	1108.6***	1107.3***	1111.8***
Cragg–Donald <i>F</i> statistic	769.2	500.2	851.2	849.9	853.4
K–P rk Wald <i>F</i> statistic	351.1	226.8	388.7	388.2	389.9

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14. Figures in square brackets are *p*-values.

K–P, Kleibergen–Paap.

Robust standard errors in parentheses; ****p* < 0.01; ***p* < 0.05; **p* < 0.1

Critical values for Cragg–Donald Wald *F* statistic

5% maximal IV relative bias	13.91	10% maximal IV size	22.30
10% maximal IV relative bias	9.08	15% maximal IV size	12.83
20% maximal IV relative bias	6.46	20% maximal IV size	9.54
30% maximal IV relative bias	5.31	25% maximal IV size	7.80

of a daily level is significant at the 10% level. An active level of LTPA reduces the probability of being overweight and of obesity by 4.3 and 6.5% respectively based on the linear IV model; however, the bivariate probit results only support the causal link between LTPA and obesity, with the probability of being obese falling by 5.3% for physically active individuals, but does not support the causal link between LTPA and being overweight. Both linear IV and recursive bivariate probit results show that daily, moderate and active LTPA does not reduce the probability of diabetes, high BP and heart disease.⁸

Similar to the probit results, the WRPA variables are statistically significant predictors of obesity and chronic conditions in both the IV and recursive bivariate probit models. The estimated coefficients on three WRPA variables from the IV models are qualitatively similar to the probit models.

Our results show that WRPA play an important role in reducing both obesity and chronic diseases. Because these activities are primarily governed by occupation and not easily adjusted by policy levers, programmes aimed at the promotion of workplace wellness, especially in sedentary occupations, may reduce the adverse health effects attributed to leisure-time physical inactivity. Contrary to the probit results, we find that LTPA does not reduce the probability of having chronic conditions. Our results suggest that only an active level of LTPA has the potential to reduce the probability of obesity in the range of five to six percentage points. Although the IV results show that a moderate level of LTPA reduces the probability of being overweight by four percentage points, this finding cannot be confirmed with the bivariate probit model.

4.3. Interaction of LTPA and WRPA

Given the limited impact of LTPA on overweight and obesity and its lack of impact on chronic conditions, it is interesting to examine if there is any role to be played by LTPA in combination with WRPA: We wonder if LTPA and WRPA are substitutes or complements. To explore this question, we estimated the effect of LTPA on obesity and chronic conditions across three WRPA sub-groups: sedentary, stand/walk and lift light or heavy load. The estimated results from the probit, linear IV and recursive bivariate probit models are presented in Tables VIIa–VIIc. The econometric test results presented in Table VIIb are consistent with the full sample, suggesting that our instruments are relevant in the sub-group analyses.

The probit results show that each of the daily, moderate and active LTPA is associated with a decrease in the probability of being overweight and obese in all sub-groups. The overall results for sedentary and stand/walk sub-groups are similar to the full-sample results, but the magnitudes of the associations are a bit smaller in the lift

⁸The reduced sample sizes in the linear IV and recursive bivariate probit models are because of missing temperature data for one or more periods. To ensure that the simple probit regression results are not an artefact of the larger sample size, we ran the analysis based on our IV samples and did not find appreciable differences in partial effects in the probit models.

Table VIIC. Average partial effects—recursive bivariate probit estimates (by WRPA status)

WRPA/LTPA	Overweight	Obese	Diabetes	High BP	Heart Disease
WRPA: Sedentary					
Daily	.057 (.057)	.065 (.078)	.013 (.021)	.030 (.038)	.018 (.027)
Observations	55 044	35 043	61 489	61 428	61 470
Estimated ρ	−0.16	−0.25*	−0.10	−0.15	−0.19
Moderate	.050 (.050)	.091 (.070)	.004 (.017)	.025 (.033)	.008 (.020)
Observations	55 044	35 043	61 489	61 428	61 470
Estimated ρ	−0.16*	−0.34***	−0.13	−0.16	−0.13
Active	.114** (.050)	.103 (.069)	.010 (.019)	.052 (.036)	−.014 (.022)
Observations	42 145	27 091	47 543	47 498	47 522
Estimated ρ	−0.28***	−0.39***	−0.17*	−0.27***	−0.03
WRPA: stand/walk					
Daily	−.087** (.040)	−.108** (.048)	.003 (.013)	−.006 (.022)	−.023 (.022)
Observations	104 875	64 722	117 515	117 431	117 464
Estimated ρ	0.09	0.07	−0.04	−0.02	0.16
Moderate	−.056* (.031)	−.055 (.038)	.002 (.010)	.001 (.020)	−.018 (.010)
Observations	104 875	64 722	117 515	117 431	117 464
Estimated ρ	0.05	−0.02	−0.08	−0.07	0.08
Active	−.052* (.010)	−.121*** (.044)	−.0005 (.011)	−.004 (.011)	−.012 (.011)
Observations	76 793	47 672	86 173	86 105	86 140
Estimated ρ	0.01	0.03	−0.08	−0.09	0.03
WRPA: lift light/ heavy load					
Daily	−.082** (.041)	−.079* (.043)	.010 (.009)	.021 (.019)	−.002 (.010)
Observations	83 142	50 251	89 821	89 712	89 800
Estimated ρ	0.11*	0.05	−0.10	−0.13*	0.01
Moderate	−.030 (.032)	−.032 (.035)	.013* (.007)	.022 (.016)	−.004 (.009)
Observations	83 142	50 251	89 821	89 712	89 800
Estimated ρ	0.03	−0.04	−0.15**	−0.15**	0.01
Active	−.049 (.034)	−.076** (.034)	.006 (.007)	.013 (.016)	−.007 (.009)
Observations	61 949	37 565	67 007	66 936	66 995
Estimated ρ	0.04	−0.01	−0.10	−0.14**	0.04

Note: all regressions include the full set of control variables, X_i , defined on pages 13–14.

Robust standard errors in parentheses; *** $p < 0.01$; ** $p < 0.05$; * $p < 0.1$.

light or heavy load sub-group. Similar results are also found for diabetes, high BP and heart disease except for the lack of statistical significance for diabetes and heart disease in some instances, particularly in the lift light or heavy load sub-group.

For the linear IV and bivariate probit estimates, except for being overweight and obese in certain sub-groups, the statistical significance of LTPA in all other models disappeared. Clearly, LTPA does not cause a reduction in the probability of diabetes, high BP and heart disease regardless of the level of WRPA. From Tables VIIb–VIIC, LTPA has no effect on overweight and obesity among those reported having sedentary WRPA; even an active level of LTPA does not reduce the probability of being overweight and obese, meaning that LTPA and WRPA are not substitutes in this sub-group.

We find that the intensity of LTPA has the greatest effect on obesity among those who also undertake some WRPA like standing or walking. An active level of LTPA reduces the probability of being overweight by about five to seven percentage points, while it reduces the probability of obesity by 11 to 12 percentage points. We also find that daily and moderate levels tend to reduce the probability of being overweight and obese in this sub-group. An active level of LTPA also reduces the probability of being obese by about seven percentage points among those who report lifting light or heavy loads at their work. These results suggest that LTPA and WRPA seem to be complementary in nature as far as the overweight or obesity risk is concerned: Those who report some WRPA and undertake an active level of LTPA reduce the probability of being overweight and obese.

5. DISCUSSION AND CONCLUSIONS

Advances in technological innovations often lead to reductions in the energy expended in the workplace and home environments, but a lack of physical activity is regarded as an important risk factor for obesity and chronic diseases (Weinsier *et al.*, 1998; Fogelholm and Kukkonen-Harjula, 2000; Waxman and Assembly, 2004; Wareham *et al.*, 2005; Summerbell *et al.*, 2009; PHAC, 2011). While a negative association between measures of LTPA and obesity has been found in previous Canadian studies (Craig *et al.*, 2005; Chen and Mao, 2006; Tjepkema, 2006; Ross *et al.*, 2007; Godley and McLaren, 2010), the extent to which these estimates capture a causal relationship is questionable given the endogenous nature of LTPA. To address this problem, we use an IV method of estimation, and we include the potentially confounding role of WRPA using population-based, large health surveys from Canada.

Our paper makes two novel contributions. First, it includes workplace physical activity as well as LTPA in the analysis of physical activity and several health outcomes. Second, it employs a robust estimation strategy to control for the endogeneity of LTPA using respondents' neighbourhood temperatures as a novel identifying instrument. We find that controlling for WRPA results in marginally smaller average partial effects attributable to LTPA on health outcomes. However, once account is taken of the endogeneity of LTPA, we find that it has a large influence on the estimated average partial effects and statistical significance.

WRPA exerts a negative effect on being overweight or obese and having chronic diseases among Canadians aged 18 to 75 years. Consistent with the previous literature, we uncover negative associations between LTPA participation and its intensity and obesity and chronic conditions, but once the endogeneity of LTPA is taken into account, these links become more nuanced. For instance, LTPA does 'not' reduce the probability of overweight and obesity if the WRPA is sedentary. However, an active level of LTPA can cause a reduction in the probability of being overweight and of obesity by five to six percentage points and a reduction in obesity of 11 to 12 percentage points if some WRPA like standing or walking is also in the mix. We also find that intensive WRPA seems to reduce some of the effects of LTPA on obesity, suggesting a reduced marginal effect of LTPA for those engaged in occupations that involve physical strenuousness.

We find that neither LTPA participation nor intensity causes a reduction in the probability of diabetes, high BP and heart disease. By contrast, Humphreys *et al.* (2014) conclude that participation in LTPA reduces the probability of having diabetes, high BP and heart disease. Specifically, they find that daily, moderate and active LTPA lead to a reduction in the probability of having diabetes by 2.4, 8.5 and 3.4 percentage points respectively and reduction in the probability of having high BP by 3.8, 14.3 and 5.17 percentage points respectively. They also find that moderate LTPA reduces the probability of having heart disease by about 10 percentage points. Although several differences exist between Humphreys *et al.* (2014) and our study, we think that the use of different instruments is explaining differences in the results. If we exclude WRPA from the model, our results are not at all comparable with Humphreys *et al.*, reinforcing our conclusion that when appropriate instruments are employed, the causal effect of LTPA on diabetes, high BP and heart disease disappears.

Our study does not suggest that intensive physical activity has no role to play in reducing the incidence of chronic conditions. In fact, our results show that moderate WRPAs, like standing or climbing stairs, reduce the probability of having these chronic conditions. However, even an active level of LTPA cannot compensate for sedentary work patterns, underscoring the importance of regular physical activity. The integration of physical activity into daily lives is a crucial factor in reducing the incidence of preventable diseases, like obesity, diabetes, high BP and heart disease.

Although this study has several strengths, there are some limitations. The first concerns the biases introduced by self-reported data. We try to minimize this problem by employing a corrected height and weight measure for the calculation of BMI, although measurement bias will undoubtedly still exist. Self-reported chronic conditions may also be subject to bias, but it is unlikely that this bias will be large. Second, the intensity of physical activities at the individual level was not collected in the CCHS. Although Statistics Canada adopted the low-intensity measure for each activity, having information on the actual intensity at the individual level in future studies would be desirable. A third weakness is that, unlike LTPA, our WRPA variable was based

on the responses to a single-item question. Detailed data on energy expenditure associated with work-related activities could improve the study and provide a better basis for specific policy recommendations. A fourth weakness is that our exogenous instrument captures primarily the outdoor component of LTPA rather than the entire range of physical activities undertaken by the respondents. Previous literature suggests that over 40% of measured physical activity can be explained by weather, and hence, variations in temperature are the source of our identification strategy. Our results can be interpreted as the local average treatment effect, which is typical with most IV estimates found in the literature. However, separating the indoor and outdoor components of LTPA in future surveys would provide greater insights. A fifth potential weakness is that the negative relationship between WRPA and obesity and chronic conditions found in this paper could also be attributed to overall lifestyle choices insofar as those who are physically active may also be more inclined to make other healthy choices. Furthermore, the endogeneity of WRPA cannot be ruled out for some segments of the population: Plausible exogenous variation in WRPA levels may provide an avenue for future research in this area. Finally, our analysis is based on three cross-sectional surveys, thereby limiting our ability to account for time-invariant unobserved heterogeneity. Future studies using longitudinal data can overcome this limitation.

Despite these limitations, our overall results clearly suggest that policies designed to encourage active LTPAs combined with the promotion of physical activity in the workplace will reduce the risk of being overweight or obese. Moreover, the integration of physical activity into daily work lives, especially for those in sedentary occupations, can have a considerable impact on reducing the burden of preventable chronic diseases, thus improving the health of the population. Several policy avenues are possible. Fitness programmes could be facilitated and/or encouraged in the workplace by, for instance, access to free or subsidized gym memberships or by designating spaces for physical activities. Change rooms and/or shower facilities could be provided for employees who exercise during breaks or take physically active travel modes (e.g. bicycling or walking) to work. Programmes like a 'take-the-stairs' type campaign could be launched with various incentives for winning teams, and flexible hours that facilitate early morning or lunchtime activities, can all work towards informing and encouraging a more active workplace environment. The results of our paper suggest that a little effort in this regard has the potential to make a big difference to the health of individuals.

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