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Residential greenness and risk of prostate cancer: A case-control study in Montreal, Canada



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ABSTRACT

Background: Recent studies suggest that exposure to greenness favors several health outcomes. We assessed whether living in the proximity of greener areas was related to prostate cancer incidence in a population-based case-control study in Montreal, Canada.

Materials and methods: Interviews eliciting lifetime addresses were conducted with 1933 prostate cancer cases diagnosed in 2005–2009 and 1994 population controls. Odds ratios (OR) and 95% confidence intervals (CI) estimated the association between residential greenness, both at recruitment (2005–2009) and about ten years prior (1996), defined by the normalized difference vegetation index (NDVI) around the home, and prostate cancer risk. Three models were developed adjusting for age, individual characteristics, and individual and ecological characteristics, estimating relative risk in relation to an interquartile range (IQR) increase of the NDVI.

Results: We observed inverse associations between greenness measured within home buffers of 150 m, 300 m, 500 m and 1000 m, at both time points, and risk of prostate cancer, independently of individual and ecological characteristics. For instance, using a buffer of 300 m, the OR for an IQR increase of 0.11 in NDVI at the time of recruitment was 0.82 (95%CI 0.74–0.92). The corresponding OR for an IQR increase of 0.15 in NDVI in 1996 was 0.86 (95%CI 0.74–1.00). There were little differences in risks according to buffer size, the time point of exposure, when considering prostate cancer aggressiveness, or when restricting controls to men recently screened for prostate cancer to reduce the likelihood of undiagnosed cancer among them.

Conclusion: Men living in greener areas, either recently or about a decade earlier, had lower risks of prostate cancer, independently of socio-demographic and lifestyle factors. These observations are novel and require confirmation.

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1. Introduction

The causes of prostate cancer (PCa) remain elusive. The currently recognized risk factors are age, family history and Sub-Saharan ancestry. Other factors showing suggestive evidence include obesity, alcohol intake, physical activity, air pollution and exposure to pesticides (Koutros et al., 2013; Kruk and Czerniak, 2013; McGregor et al., 2013; WCRF and AICR, 2014). Genetic factors identified to date explain only a modest proportion of familial risk (Eeles et al., 2014).

Abbreviations: PCa, prostate cancer; PSA, prostate screening antigen; DRE, digital rectal examination; NDVI, normalized difference vegetation index; CI, confidence interval.

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It has long been suspected that some PCa have environmental origins (Haas and Sakr, 1997; Hsing and Chokkalingam, 2006), with supporting evidence from migrant studies (Haenszel and Kurihara, 1968). Striking geographic variations in PCa occurrence and progression are observed at international, national, and local levels (Klassen and Platz, 2006). The use of spatially referenced data thus represents a promising approach to better understand the factors associated with this cancer.

Evidence is accruing for a beneficial effect of a natural outdoor environment in health (Bowler et al., 2010). For instance, use of spatial data has recently suggested an association between exposure to greenness and a lower risk of all-cause (non-accidental) and cancer mortality in women (James et al., 2016).

Neighborhood greenness has been associated with increased physical activity, lower odds of being overweight or obese, and lower exposure to air pollution (Hartig et al., 2014; James et al., 2015), each of which may in turn influence PCa risk (Parent et al., 2013; WCRF and AICR, 2014; Wekesa et al., 2015).

To our knowledge, the association between living in greener areas, recently and in the past, and PCa incidence has never been documented. Using data collected as part of a large case-control study conducted in Montreal, we assessed whether residential greenness was associated with PCa incidence at the time of diagnosis or recruitment, as well as about a decade earlier to take into account a potential latency period (Salinas et al., 2014). A wide array of potential personal and contextual factors, including PCa screening practices, was considered.

2. Materials and methods

2.1. Study population

We used data from The Prostate Cancer & Environment Study (PROtEuS), a large population-based case-control study conducted in Montreal, Canada. This study has been described previously (Blanc-Lapierre et al., 2015).

Eligible participants were men younger than 76 years old at diagnosis or selection, who were residents of the greater Montreal area (Montreal Island, North and South Shores), and registered on Quebec's permanent electoral list (continually updated). Cases were all patients newly diagnosed with primary PCa, actively ascertained through pathology departments across French-speaking hospitals in the Montreal area between September 2005 and December 2009. This covered over 80% of all PCa cases diagnosed in Montreal during the study period. Control subjects were selected concurrently from the population-based provincial electoral French-speaking list, and frequency-matched to cases by 5-year age group. Potential controls with a history of PCa were excluded.

Participation rates among eligible subjects were 79.4% for cases and 55.5% for controls.

2.2. Data collection

Between 2005 and 2012, in-person interviews collected information on socio-demographic characteristics, family history of cancer, medical history, PCa screening history, lifetime physical activity, smoking, alcohol consumption, dietary intake, and self-reported weight and height. The degree of aggressiveness of PCa, defined by the Gleason score, was extracted from prostate biopsy pathology reports. Residential addresses at the time of diagnosis of cases were extracted from hospital records; those of controls were obtained from electoral lists at the time of recruitment. Lifetime residential addresses were elicited through follow-up telephone interviews. All addresses were geocoded with the ArcGIS geographic information system (GIS, ESRI, Redlands, CA) using the geodatabase "Adresses Québec" that covers the whole province of Quebec, then linked to greenness exposure variables.

Exposure to nitrogen dioxide (NO₂) was used as a marker of traffic-related air pollution. Using land-use regression models, concentrations of NO₂ at the subjects' home address at index date were estimated at a resolution of 5 m² for people living in the Island of Montreal (Crouse et al., 2009a; Parent et al., 2013). Annual exposure averages were based on measurements taken on three occasions (spring, summer and winter). NO₂ values were only available for a subset of our sample (1242 cases and 1239 controls) as the survey was conducted on the Montreal Island and did not cover subjects living on the North or South Shores.

2.3. Residential greenness exposure

PCa tumors in older men are thought to have a latency period of 10 years between initial growth and their clinically detectable state, with a shorter period for those younger (Salinas et al., 2014). With a potential etiological role in mind, it was thus of interest to look at past greenness exposure. We selected two time points of exposure, i.e., the time of recruitment and 1996, the latter corresponding to about

10 years prior to the year of diagnosis of earlier cases (late 2005), or slightly longer for cases diagnosed in 2009. Assessing past exposure to greenness around or prior to the onset of tumor growth allowed us to consider address changes, and thus possible differences in greenness exposure, between the two time points. We elected to not assess earlier exposure, as errors in self-reported residential history could compromise analyses.

Satellite images covering the Montreal region, captured on August 5, 1996 and June 27, 2005 from Landsat TM5, were used to evaluate the greenness around the subjects' residences in 1996 and close to the time of diagnosis or recruitment (index date), respectively. Both images were corrected for atmospheric effects using the dark object subtraction method with the following assumptions: null downwelling diffuse irradiance and atmospheric transmittance equals to 1 (Song et al., 2001).

Landsat TM5 scans one location on Earth every 16 days. The normalized difference vegetation index (NDVI) for Montreal can only be calculated during summer months (late June to mid-September) as there is no green vegetation during winter, leading to about 6 timeslots for useable satellite imagery. Adding cloud cover constraints left only 1 image for 2005 (June 27, 2005), and we selected the August image for 1996, as vegetation had reached its highest level of photosynthesis activity.

Greenness was determined using the NDVI, an indicator of the density of green vegetation or biomass (Weier and David, 2000). Green vegetation reflects more infrared radiation and absorbs more energy in red wavelengths than non-vegetated surfaces. NDVI is calculated according to the level of reflectance of near-infrared (NIR) and visible red (VR) wavelength spectra detected by satellite. Using spectral data available at a 30-m by 30-m resolution (band 3 and 4), NDVI is calculated as: $NDVI = (NIR - VR) / (NIR + VR)$. Scores range from -1 to $+1$, with higher positive values indicating denser levels of healthy vegetation.

Average NDVI values from Landsat TM5 images were computed within 150 m, 300 m, 500 m and 1000 m radii buffers around each home address, for both time points (in 1996 and at the index date). Using several buffers of varying sizes allowed assessing sensitivity of exposure effects at various scales.

2.4. Statistical analyses

Odds ratios (OR) and 95% confidence intervals (CI) for the association between exposure to residential greenness and PCa risk were estimated using unconditional logistic regression models. A first model (Model 1) adjusted for age only. A second model (Model 2) adjusted for the following potential individual confounders: age (continuous), ancestry (Sub-Saharan, Asian, European, Other), first-degree family history of PCa (Yes, No), education (Elementary, High school, College, University), reported family income, in CAN\$ (<20,000, 20,000–29,999, 30,000–49,999, 50,000–79,999, ≥80,000), marital status (married or common law, separated, divorced or widower, single, member of religious order, other), smoking (cigarette pack-years), alcohol consumption (drink-years), dietary habits (frequency of use of fruit and vegetables 2 years earlier) and a history of diabetes (Yes, No). A third model (Model 3) further adjusted for neighborhood material and social deprivation. The latter were represented by a deprivation index constructed by Pampalon et al. (2000) available at the Dissemination Area level (small census unit inhabited by 400–700 persons). The social and material deprivation indices were derived from a principal component analysis conducted on six census data variables (the proportion of persons without high-school diploma, the ratio of employment in the population, the average income, the proportion of persons who are separated, divorced or widowed, the proportion of single-parent families and the proportion of people living alone). The first resulting component reflects variations in education, employment and income, emphasizing the material aspect of deprivation. The second component reflects variations in the indicators associated with the social aspect of deprivation (the proportions of widowed, separated and divorced persons, of single-parent families and of persons living alone). For our

analyses, area-weighted average material and social deprivation scores were calculated for each residential buffer using the underlying Dissemination Area-level indexes.

In a second set of analyses, we restricted controls to men screened (by PSA and/or digital rectal examination (DRE)) within the 2 years preceding the index date, to take into account potential confounding by screening history, and to reduce the possibility of undiagnosed PCa among controls.

All analyses were repeated taking into account the degree of aggressiveness of PCa, defined by the Gleason score. Risks of low-grade (Gleason scores < 7 or 3 + 4) and high-grade (Gleason scores > 7 or 4 + 3) PCa were estimated in unconditional polytomous logistic regression models.

A sensitivity analysis excluded proxy respondents (<4% of subjects). In order to explore the possibility that associations might differ according to deprivation, we also stratified analyses according to tertiles of the distributions of social and material deprivation indices among controls.

Finally, we evaluated the impact of including body mass index (BMI), physical activity and exposure to NO₂ in our models. These might act as mediators in the association between greenness and PCa risk. Maximum lifetime BMI and NO₂ were modeled as continuous, while physical activity level during adulthood linked to recreational, occupational and residential activities was introduced as a categorical variable (Very, Moderately, Not very active).

The average NDVI was analyzed as a continuous linear variable after verifying that conditions for linearity were met. Linearity was assessed by scatterplots (for quartiles of NDVI) and by the likelihood ratio test (for the continuous variable and quartiles). ORs were expressed in relation to an interquartile range increase of NDVI.

3. Results

The study population comprised 1933 cases (1395 low-grade PCa and 535 high-grade PCa) and 1994 controls. Fig. 1 provides details on addresses collection and geocoding. The home address, geographic coordinates at the index date and greenness exposure were available for all 3927 subjects. Information on home address in 1996 was collected at re-contact for 2891 (74%) subjects. Some 2646 (92%) of these could be geocoded using “Adresses Québec”. Greenness exposure determined by satellite data for the year 1996 was available for 2616 subjects (982 low-grade PCa and 373 high-grade PCa, and 1258 controls). About one third of subjects had different addresses in 1996 and at the index date.

The characteristics of study participants are presented in Table 1. Average age was 63.6 and 64.8 for cases and controls, respectively. As expected, the case series included a greater proportion of subjects of Sub-Saharan ancestry and a lower proportion of Asian subjects. Cases reported more frequently a first-degree family history of PCa than controls, while a lower proportion of cases than controls had been diagnosed with diabetes. Cases had a slightly greater level of exposure, on average, to NO₂. There were little differences in terms of education, income, marital status, smoking and alcohol history, physical activity, BMI and frequency of use of fruit and vegetables according to case/control status. Nearly all cases (a few were diagnosed during surgery at a contiguous site) and 76% of controls had been exposed to PCa early detection efforts in the form of PSA and/or DRE testing within the 2 years preceding the interview.

Statistics of the NDVI for the study population are presented in Table 2. Distributions and interquartile ranges were similar in buffers of 150 m, 300 m, 500 m, 1000 m. NDVI averages were slightly lower for

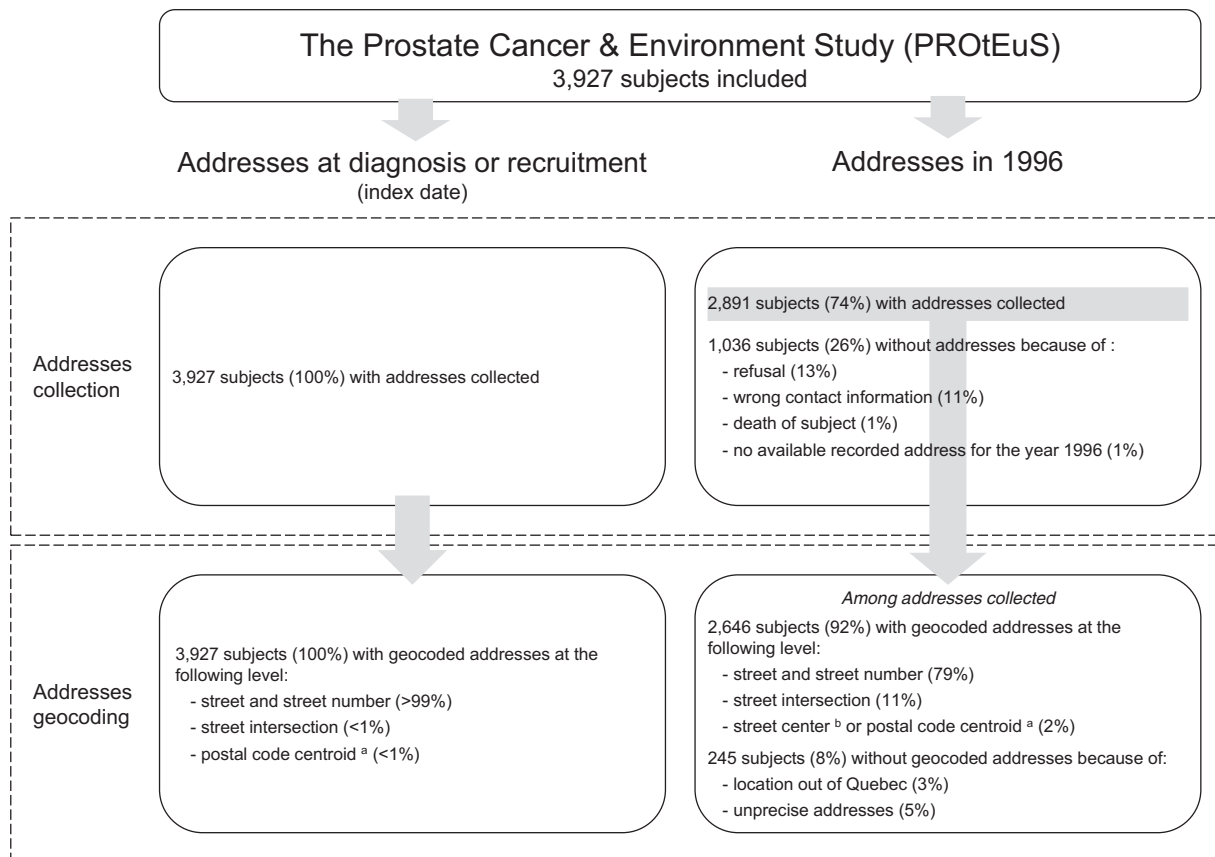


Fig. 1. Collection and geocoding of residential addresses in the Prostate Cancer & Environment Study (PROtEuS). ^aIn Montreal, the postal code area usually refers to a block face or to a large apartment complex. ^bAddresses were geocoded to the center of the street when this street was <500 m long.

Table 1
Characteristics of the study population, PROtEuS, Montreal, Canada, 2005–2012.

	Cases (n = 1933)	Controls (n = 1994)
Age in years, mean ± std	64 ± 7	65 ± 7
Ancestry, n (%)		
Sub-Saharan	129 (7)	90 (5)
Asian	24 (1)	72 (4)
European	1693 (88)	1686 (85)
Other	75 (4)	132 (7)
Don't know	12 (1)	14 (1)
First-degree family history of prostate cancer, n (%)		
No	1412 (73)	1739 (87)
Yes	450 (23)	196 (10)
Don't know	71 (4)	59 (3)
Education, n (%)		
Elementary	449 (23)	429 (22)
High school	576 (30)	578 (29)
College	313 (16)	375 (19)
University	590 (31)	610 (31)
Other	5 (0)	2 (0)
Family income in CAN\$, n (%)		
<20,000	225 (12)	245 (12)
20,000–29,999	264 (14)	252 (13)
30,000–49,999	449 (23)	462 (23)
50,000–79,999	423 (22)	410 (21)
≥80,000	425 (22)	428 (21)
Don't know	147 (8)	197 (10)
Marital status, n (%)		
Married, common law	1503 (75)	1428 (74)
Separated, divorced, widower	332 (17)	329 (17)
Single	151 (8)	164 (8)
Member of religious order, other	7 (0)	12 (1)
Don't know	1 (0)	0 (0)
Maximum body mass index in kg/m ² , mean ± std	28 ± 4	29 ± 5
Physical activity, n (%)		
Not very active	475 (25)	547 (27)
Moderately active	472 (24)	491 (25)
Very active	980 (51)	953 (48)
Don't know	6 (0)	3 (0)
Smoking in pack-years, mean ± std	23 ± 28	24 ± 28
Alcohol consumption ^a in drink-years, mean ± std	85 ± 130	85 ± 148
Daily frequency of use of fruit and vegetables, n (%)		
≤6	476 (25)	498 (25)
[7–9]	506 (26)	497 (25)
[10–12]	430 (22)	497 (25)
>12	510 (26)	498 (25)
Don't know	11 (1)	4 (0)
History of diabetes, n (%)		
No	1640 (85)	1596 (80)
Yes	290 (15)	395 (20)
Don't know	3 (0)	3 (0)
Timing of the last prostate cancer screening by PSA ^b or DRE ^c		
Within last 2 years	1913 (99)	1511 (76)
2–5 years earlier	1 (0)	154 (8)
>5 years earlier	0 (0)	81 (4)
Never screened	3 (0)	191 (10)
Don't know	16 (1)	57 (3)
Concentrations of NO ₂ in parts per billion, mean ± std	12.3 ± 2.8	11.9 ± 2.7

^a Among subjects who reported ever consuming alcohol once a month for one year or more.

^b PSA: prostate specific antigen.

^c DRE: digital rectal examination.

the index date than for 1996 residential locations. Pearson correlation coefficients between the four buffer measures of greenness varied between 0.64 and 0.93 for addresses at the index date, and between 0.71 and 0.95 for addresses in 1996.

Table 3 presents results for the association between the NDVI estimated for addresses at the index date, and the risk of PCa. Odds ratios

are shown for three different adjustment models in relation to an inter-quartile range increase of the NDVI. In Model 1, adjusting for age only, we observed a significant inverse association between the risk of PCa and greenness in all buffers. Similar ORs were observed in Model 2, adjusting for individual characteristics, and Model 3 further adjusting for neighborhood material and social deprivation (OR 0.82, 95%CI 0.74–0.92, using a buffer of 300 m), although the inverse association was slightly attenuated in the latter. Table 3 also presents results after excluding the 483 (24%) controls who had not been screened within the two years preceding the interview and who were thus more likely to have undiagnosed PCa. With this sample, inverse associations were slightly stronger in Models 1 and 2, compared to those in the main analyses.

Table 4 shows results for the association between the NDVI estimated for subjects' home addresses in 1996 and the risk of PCa. In Models 1 and 2, significant inverse associations were found in main analyses for the risk of PCa as well as in analyses restricted to recently screened controls. In Model 3, significant inverse associations were only observed for the 500 m buffer.

In analyses conducted by degree of cancer aggressiveness, both low-grade and high-grade PCa showed negative associations with greenness, nearly identical to those in the full sample (Appendix Tables A.1 and A.2). Analyses excluding proxy respondents (<4%) did not alter findings. Associations were similar across strata of area-level deprivation (data not shown).

The inclusion of physical activity and BMI in the regression models had a marginal impact on results for both time points (Appendix Tables A.3 and A.4). When testing the inclusion of NO₂ in Model 3, using only the sub-sample of subjects living on the Montreal Island and for whom survey data were available (about 60% of our total sample), the OR between exposure to greenness at the index date and PCa was 0.83 (95%CI 0.74–0.92), and 0.89 (95%CI 0.73–1.08) for exposure in 1996, using a buffer of 300 m.

4. Discussion

Research assessing natural environmental factors' relationship to health is growing. Most publications on the subject appeared in the last five years and were recently reviewed (Gascon et al., 2015; Gascon et al., 2016; Gomez et al., 2015; Hartig et al., 2014; James et al., 2015). As a whole, the emerging evidence supports a beneficial effect of the natural outdoor environment on health. Exposure to greenness has been suggested to reduce all-cause mortality (Mitchell and Popham, 2008; Villeneuve et al., 2012), cardiovascular disease incidence and mortality (Pereira et al., 2012; Richardson and Mitchell, 2010), respiratory disease mortality (Richardson and Mitchell, 2010), stroke mortality (Hu et al., 2008; Wilker et al., 2014), adverse birth outcomes (Ebisu et al., 2016), as well as obesity (Ellaway et al., 2005; Lachowycz and Jones, 2011). Positive health effects of greenness have also been observed on well-being and mental health (Alcock et al., 2014; Cohen-Cline et al., 2015; White et al., 2013), and on self-perceived health (Maas et al., 2006).

With respect to cancer risk, studies assessing a potential role of exposure to greenness have been conducted on skin cancer incidence (Astell-Burt et al., 2014), lung cancer (Bixby et al., 2015; Mitchell and Popham, 2008; Richardson et al., 2010; Richardson and Mitchell, 2010), and esophageal cancer mortality (Wu et al., 2008). Recently, an inverse association between greenness exposure and cancer mortality was observed in a prospective cohort of women in the US (James et al., 2016).

PCa incidence has been previously associated with a number of spatial factors related to the ecologic environment. These include exposure to sunlight and ultraviolet radiation (Bodiwala et al., 2003; Gilbert et al., 2009; Lin et al., 2012), exposure to NO₂ as a marker of traffic and urban air pollution (Al-Ahmadi and Al-Zahrani, 2013; Parent et al., 2013; Shekarrizfard et al., 2015), proximity of residence to industrial sites

Table 2

Mean greenness exposure (NDVI) in buffers of 150 m, 300 m, 500 m and 1000 m for addresses at diagnosis/recruitment and in 1996, and correlation between buffers.

	Addresses at diagnosis/recruitment (n = 3927)				Addresses in 1996 (n = 2616)			
	150 m	300 m	500 m	1000 m	150 m	300 m	500 m	1000 m
Buffers size								
Mean	0.33	0.33	0.33	0.32	0.42	0.42	0.42	0.41
Std	0.09	0.08	0.08	0.08	0.12	0.11	0.11	0.11
Min	0.05	0.05	0.08	0.06	−0.07	0.05	0.06	0.04
Max	0.65	0.65	0.65	0.60	0.80	0.80	0.80	0.80
Interquartile range	0.12	0.11	0.11	0.10	0.16	0.15	0.15	0.15
Pearson correlation								
	150 m	1	0.91	0.79	1	0.92	0.83	0.71
	300 m		1	0.93		1	0.95	0.81
	500 m			1			1	0.90
	1000 m							1

(Ramis et al., 2011), albeit the evidence for these is not entirely consistent (Raaschou-Nielsen et al., 2011; Yu et al., 2014). To our knowledge, ours is the first study to examine the association between exposure to residential greenness and PCa at two time points, i.e., recently and about ten years earlier using past exposure data, and PCa incidence. Findings are in line with an inverse relationship between residential greenness levels and PCa risk. This holds true at both time points, for different buffer sizes, for both low- and high-grade cancers, after adjustment for a wide range of personal socio-demographic, lifestyle and neighborhood characteristics, and taking into account the subjects' screening history.

4.1. Exposure assessment

Most previous studies analyzing how exposure to greenness relates to health outcomes have used aggregated data. Results of ecological studies based on aggregate exposure and health indicators at the group level must be interpreted with caution, especially if potential confounders are only available at the group level. In this study, greenness exposure was estimated for each subject in buffers around the subject's place of residence.

We used the NDVI as an exposure metric. Without strong a priori hypotheses about greenness as a risk factor, this index which measures overall greenness appeared to be objective and valid. NDVI was found to be highly correlated with environmental psychologists' evaluations

of greenness and judged as a valid measure for quantifying levels of greenness in residential areas (Rhew et al., 2011). We were not able to consider the impact of greenness type, which could influence physical activity behavior, nor were we able to consider greenness quality. Land-use datasets may provide more information about specific types of greenness, potentially giving an indication of their quality or usability (James et al., 2015). However, specific designations may mischaracterize green land uses, for example parks that do not contain vegetation. Additionally, land-use files are often too coarse to capture small-scale vegetation, such as gardens and street trees. Moreover if the reduced risk of PCa is explained by reductions in air pollution and noise, or reduction of stress due to nature viewing, then small amounts of green or greening of streets may be sufficient. These determinants, such as the quality of greenness and how these areas are perceived, might also be relevant, as well as other aspects of the built environment (e.g. degree of urbanization or ease of accessibility) that have been poorly explored (Nieuwenhuijsen et al., 2014). These issues need to be further studied (Gascon et al., 2016).

There were no differences across buffer sizes with respect to PCa risk. Buffers of 150 m and 300 m radii represent very local greenness while 500 and 1000 m buffers are indexes for surrounding greenness. Very local greenness could lead to improved air quality for the local neighborhood and reduced noise. Neighborhood greenness could be an indicator for a walkable environment that increases opportunity for physical activity, or be related to local urban heat islands. Presence of mature trees in 100 m and 500 m radius has

Table 3

Odds ratio (95% confidence interval) for the association between an interquartile range increase of the NDVI^a at the time of diagnosis/recruitment and prostate cancer risk, according to buffer size.

	Model 1 Age ^b	Model 2 Individual ^c	Model 3 Individual ^c + ecological ^d
All subjects (1933 prostate cancer cases and 1994 controls)			
Buffer size			
150 m	0.81 (0.74,0.89)	0.80 (0.73,0.89)	0.84 (0.75,0.94)
300 m	0.81 (0.74,0.88)	0.79 (0.72,0.87)	0.82 (0.74,0.92)
500 m	0.81 (0.74,0.88)	0.79 (0.72,0.87)	0.84 (0.75,0.94)
1000 m	0.79 (0.72,0.86)	0.78 (0.71,0.85)	0.84 (0.75,0.94)
Restricting controls to the 1511 men screened ^e within the last 2 years			
Buffer size			
150 m	0.74 (0.67,0.82)	0.77 (0.69,0.86)	0.83 (0.74,0.94)
300 m	0.75 (0.68,0.82)	0.77 (0.69,0.85)	0.82 (0.73,0.93)
500 m	0.75 (0.68,0.82)	0.77 (0.69,0.85)	0.85 (0.75,0.96)
1000 m	0.73 (0.67,0.80)	0.75 (0.68,0.83)	0.83 (0.74,0.94)

^a NDVI interquartile ranges were 0.12, 0.11, 0.11 and 0.10 for buffers of 150 m, 300 m, 500 m and 1000 m, respectively.

^b Adjusted for age.

^c Adjusted for age, ancestry, family history of prostate cancer, education, income, marital status, smoking, alcohol consumption, fruit and vegetables consumption, diabetes.

^d Adjusted for neighborhood material and social deprivation.

^e Prostate cancer screening by prostate specific antigen or digital rectal examination.

Table 4

Odds ratio (95% confidence interval) for the association between an interquartile range increase of the NDVI^a in 1996 and prostate cancer risk, according to buffer size.

	Model 1 Age ^b	Model 2 Individual ^c	Model 3 Individual ^c + ecological ^d
All subjects (1358 prostate cancer cases and 1258 controls)			
Buffer size			
150 m	0.83 (0.75,0.93)	0.82 (0.73,0.93)	0.93 (0.80,1.08)
300 m	0.80 (0.72,0.89)	0.78 (0.70,0.88)	0.86 (0.74,1.00)
500 m	0.78 (0.70,0.87)	0.76 (0.67,0.85)	0.84 (0.72,0.98)
1000 m	0.78 (0.70,0.87)	0.75 (0.67,0.85)	0.86 (0.74,1.01)
Restricting controls to the 985 men screened ^e within the last 2 years			
Buffer size			
150 m	0.77 (0.69,0.87)	0.79 (0.70,0.91)	0.92 (0.79,1.09)
300 m	0.75 (0.67,0.84)	0.76 (0.67,0.86)	0.86 (0.74,1.01)
500 m	0.73 (0.65,0.82)	0.73 (0.65,0.83)	0.83 (0.71,0.99)
1000 m	0.73 (0.65,0.82)	0.73 (0.64,0.83)	0.86 (0.73,1.01)

^a NDVI interquartile ranges were 0.16, 0.15, 0.15 and 0.15 for buffers of 150 m, 300 m, 500 m and 1000 m, respectively.

^b Adjusted for age.

^c Adjusted for age, ancestry, family history of prostate cancer, education, income, marital status, smoking, alcohol consumption, fruit and vegetables consumption, diabetes.

^d Adjusted for neighborhood material and social deprivation.

^e Prostate cancer screening by prostate specific antigen or digital rectal examination.

been associated with increased house value, whereas presence of low tree density and woodlands decreased house value in 500 m buffers (Kestens et al., 2004). Moreover, NDVI standard deviation within 1000 m (i.e. heterogeneity in land use/greenness) was positively associated with house value. Because these marginal values of house attributes are an expression of people's preferences, it may be that valuing tree presence/view improves quality of life and possibly health.

We considered exposure separately at two time points, currently and some 10–15 years earlier. Cumulative exposure to greenness would be expected to yield very similar results given that only one third of subjects had different addresses in 1996 and at the index date, and NDVI in 1996 and 2005 are highly correlated. Nevertheless, considering past exposures is important when studying the etiological role of exposures on diseases with a latency period.

Measurement error inevitably occurred in the study. Error was likely non-differential, leading to conservative estimates of the associations studied. Our exposure metrics focused on residential addresses, with no information on where participants spent time. Participants were assumed to have interacted with neighborhood greenness, which may not have been the case, leading to exposure misclassification. Also, possible exposure to greenness around workplaces was not considered.

4.2. Covariates

We conducted analyses considering three adjustment models. Adding individual factors (Model 2) had only a modest impact on risk estimates derived from the age-adjusted model (Model 1). In Model 2, while we did not have strong a priori hypotheses about potential confounding factors, we chose to adjust for a large range of individual confounders that could potentially be associated with both the exposure and PCa, leaving out factors that could be in the mediation pathway. We tested the inclusion of both individual factors and ecological characteristics in Model 3. Contextual attributes, when available, are often adjusted for in area-level studies when individual data are not available (Klassen and Platz, 2006). Contextual data may also be used to represent unmeasured confounders, known or unknown, or they can have their own meaning, such as representing social capital, for example.

Neighborhood sociodemographic characteristics have been shown to correlate with greenness (Maas et al., 2006; Mitchell and Popham, 2008). When neighborhood deprivation was incorporated in our third model, the inverse associations between greenness and PCa persisted, although they were attenuated, with some of the risk estimates no longer reaching statistical significance. The ecological deprivation index used in Model 3 combines indicators for education, employment, income, marital and familial status in the neighborhood. Since Model 3 also included variables for education, income and marital status measured at the individual level, we cannot exclude the possibility of over-adjustment. On the other hand, residual confounding by sociodemographic characteristics remains possible, as shown by Steinmetz-Wood et al. for the built environment (Steinmetz-Wood and Kestens, 2015). Geographical and historical factors described in Crouse et al. also demonstrate that Montreal is a complex setting in which to examine relationships between neighborhood-scale social and physical environmental characteristics (Crouse et al., 2009b). Despite our adjustments for multiple individual- and area-level variables, confounding by attributes that were either not measured, or imperfectly so, cannot be excluded. There was no evidence in our data that associations with residential greenness varied according to area-level material or social deprivation.

Few previous studies of PCa have been able to consider screening practices. This may have compromised the ability to identify risk factors for this disease (García-Closas and de Berrington, 2015). In sub-analyses restricting subjects to men screened recently for PCa, we were able to reduce the potential confounding by screening, as it may relate to

socio-demographic and cultural factors, as well as to greenness exposure. Moreover, this decreased the likelihood for undiagnosed PCa among controls.

4.3. Selection issues

Participation rates were high (79%) among cases and lower (56%) among controls. To evaluate the potential for selection bias in the study, we conducted analyses comparing study participants to non-participants in terms of four ecological variables derived from census tract data in 2006 for the address at recruitment. For instance, the percentage of subjects living in areas with a greater proportion of recent immigrants within the previous 5 years were 4.0% and 4.6%, for participants and non-participants, respectively. Corresponding values for participants and non-participants were 6.0% and 6.6% for higher unemployment rate, 19.5% and 20.1% of adults without a high school diploma, and 21.7% and 24.4% for the lowest quintile of household income. Moreover, mean NDVI values for a buffer of 300 m were 0.33 among participants and 0.32 among non-participants. The absence of disparities in socio-demographic indicators and NDVI values between participants and non-participants held true when considering cases and controls separately. These observations indicate that selection bias is not of major concern in the study.

4.4. Potential mechanisms

Several mechanisms have been proposed to explain the association between greenness and global health (Hartig et al., 2014; James et al., 2015). Greenness might help reduce exposure levels to environmental factors suspected to have negative health effects, and acting as mediators, such as air pollution (Schwartz, 1994), noise (Passchier-Vermeer and Passchier, 2000) and high temperatures (Basu and Samet, 2002). Stress and social cohesion have also been proposed as mediators between greenness and health (de Vries et al., 2013; Hartig et al., 2003; Maas et al., 2009). We can only speculate at this point regarding potential mediators for PCa specifically and our study was not designed to rigorously investigate this. Factors with reasonable plausibility include physical activity, obesity, and perhaps NO₂.

Both positive and negative associations between physical activity and PCa risk have been previously reported in observational epidemiological studies (WCRF and AICR, 2014; Wekesa et al., 2015). Higher physical activity levels among individuals with access to natural outdoor environments have been observed repeatedly (Chaix et al., 2014; Gong et al., 2014; Li et al., 2008; Richardson et al., 2013). In a Canadian survey, positive associations were observed between greenness and physical activity independent of income level (McMorris et al., 2015). While there is some evidence and expert consensus to suggest that living in greener areas can facilitate physical activity (Lee and Maheswaran, 2011), the evidence of a direct effect remains weak (Maas et al., 2008). However, the type of greenness is an important factor in determining whether and how it may influence health (Bixby et al., 2015). This could explain why some studies found that access to general greenness had no impact on individuals' levels of physical activity, whereas studies that restricted the measure of greenness to specific types of environments - such as parks or sports grounds - reported improved physical activity behavior with increased accessibility (Wendel-Vos et al., 2004). Adjusting for different types of self-reported physical activity levels, including recreational, had little influence on our findings but the variables used were relatively crude.

Higher levels and greater variation of neighborhood greenness have been associated with lower odds of obesity among adults of all ages (Pereira et al., 2013). Obesity has been linked to advanced PCa (WCRF and AICR, 2014). In our data, associations with greenness were similar for low- and high-grade cancer. Moreover, associations were unaltered when controlling for obesity.

Greenness is potentially correlated with other spatial and contextual factors. Reduced exposure to air pollution has been observed in greener areas (Dadvand et al., 2012), as vegetation is known to lower air pollution levels. We previously reported a positive association between NO₂ and PCa risk in this population (Parent et al., 2013). Nevertheless, the inverse association between recent greenness exposure and risk of PCa persisted after adjustment for NO₂ exposure in the sub-sample for which survey data was available.

In our study, findings didn't seem to point out to physical activity, obesity or air pollution as mediators. Imprecision in the assessment of these factors might mask a true mediating effect. Alternatively, it could be that factors unmeasured in our study such as temperature, noise, or neighborhood walkability (James et al., 2015) are implicated.

5. Conclusions

In this large population-based study, we observed decreased risks of PCa among men living in areas with higher levels of greenness. Research in this area is in its early stages and further work is needed to establish how risk of cancer interacts with local greenness. In the cancer domain, there is much to be learned from the experiences of neighborhood research in other disease areas. Incorporating social, built and natural environmental factors into research on cancer etiology and outcomes can lead to a better understanding of cancer processes, identify vulnerable populations, and generate results with translational impact relevant to interventionists and policy makers.

Ethics statement

The study was approved by the IRB of the Institut national de la recherche scientifique, along with the ethics committees from the following hospitals: Hôpital Notre-Dame, Hôpital St-Luc, Hôtel-Dieu de Montréal, Hôpital Maisonneuve-Rosemont, Hôpital Jean-Talon, Hôpital Charles-Lemoyne, Hôpital de Fleury, Hôpital du Sacré-Coeur de Montréal, Hôpital Santa Cabrini.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at doi:10.1016/j.envint.2016.10.024.

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