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Occupational Exposure to Engine Exhausts and Prostate Cancer Risk

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Abstract

Background Some engine exhausts (EEs) have been classified as carcinogens and/or can have hormone-modulating properties that could play a role in prostate cancer development.

Objective We investigated associations between lifetime occupational exposure to various EEs and prostate cancer risk, overall and for aggressive cancers**.**

Methods In a population-based case–control study conducted in Montreal, Canada, 1,924 incident histologically-confirmed prostate cancer cases (436 aggressive) and 1,989 population controls were recruited. Socio-demographics, lifestyle factors and a detailed occupational history were collected during in-person interviews. Industrial hygienists conducted evaluations of intensity, frequency and reliability of exposure to EEs resulting from the combustion of several fuels (any diesel, light- and heavy-duty diesel, leaded and unleaded gasoline, propane and jet fuel) in each job held ≥ 2 years. Odds ratios (ORs) and 95% confidence intervals (CI) were estimated for exposure to each EE, in association with prostate cancer risk, adjusting for age and then for potential lifestyle and occupational confounders, accounting for a 5-year latency period. As most associations were not linear, we fitted functions for changes in percentile distributions based on natural cubic splines.

Results There was no evidence of associations between exposure to the various EEs and overall prostate cancer. However, for high-grade cancers, based on the fully-adjusted model, a change from the 25th to the 75th percentile of the exposure distribution of any diesel EE yielded an OR of 1.24 (95%CI 0.96–1.61), and of 1.27 (95% CI 0.80–2.01) for a change from the 75th to the 95th percentile. These increases reflected exposure to diesel EE from light-duty vehicles, associated with similar ORs. For leaded gasoline EE, a change from the 75th to the 95th percentile resulted in an age-adjusted OR of 1.36 (95%CI 0.88–2.11), which was attenuated to 1.12 (95%CI 0.63–2.02) after full adjustment. There were no associations with EE from unleaded gasoline, diesel from heavy-duty vehicles, jet fuel and propane.

Conclusion There was suggestive evidence for a deleterious role of occupational exposure to EE resulting from the combustion of any diesel, light-duty diesel and from leaded gasoline in the development of aggressive prostate cancer. Results were independent from prostate cancer screening patterns.

Keywords Engine exhausts, Occupational exposure, Prostate cancer, Screening

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Introduction

With an annual incidence rate of 80 cases per 100,000 men, Canada is one of the Western countries where prostate cancer is the leading cause of male solid tumors [1]. Early detection and treatment are currently the only levers of control of this cancer as its confirmed risk factors (advancing age, African ancestry and a first-degree family history of the tumor) are not modifiable. A few other factors are under particular scrutiny such as obesity [2] and alcohol intake [3], but the strongest suspicion is for occupational factors i.e., certain pesticides [4], nightshift work [5] and firefighting [6].

In the context of a population-based study conducted in Montreal, we observed elevated risks of this cancer among men residing in areas characterized by higher air pollution levels, notably from ultrafine particles [7] and nitrogen dioxide [8], a marker of traffic-related air pollution. We also reported elevated risk of prostate cancer in driving occupations, gasoline station attendants and firefighters, known to entail exposure to various chemicals including engine exhausts (EEs) [9], and pointing to EEs as a potentially fruitful research avenue on prostate cancer etiology.

EEs from diesel and gasoline combustion are ubiquitous and can be found in a large range of occupations at varying levels of exposure, subjecting workers to a complex mixture of gases (i.e., carbon monoxide and nitrogen oxides), particulate matters (i.e., ashes, sulfate and metals, carbon), organic compounds and polycyclic aromatic hydrocarbons. Diesel technology is characterized as heavy-duty when diesel is used on large vehicles (industrial, agricultural, construction and manufacturing) and as light-duty when used on cars and lighter trucks, which influence the composition of the mixture.

Deleterious effects of diesel EE are well-documented for the respiratory tract, particularly the lung for which they are carcinogenic [10]. Positive associations were also reported for bladder cancer [10] that has been recognized, with acute myeloid leukaemia, as being also caused by exposure to automative gasoline (not its exhaust) in adults [11].

Gasoline EE per se has been classified as possibly carcinogenic to humans based on sufficient evidence from animal models but inadequate evidence in humans [10]. Gasoline EE contains benzene and ethylene dibromide which are, respectively, known and suspected causes of cancer [12, 13]. It also comprises higher levels of certain gases, such as carbon monoxide, than diesel EE. Organic lead, an endocrine-disruptor [14], can find itself in leaded gasoline combustion products. Use of leaded gasoline has been prohibited by the 1980s in most high-income countries but many workers were still exposed in low-income countries until 2021, which marks the official end of its use worldwide [15]. Little is known about the role of exposure to EEs and cancer at other sites and whether exposure to the combustion of fuels other than diesel and gasoline increases cancer risk.

A handful of studies have investigated whether occupational exposure to EEs is related to prostate cancer development. Findings from cohort studies were restricted to diesel EE, included few exposed cases and poorly considered potential confounders, and typically used job titles or industries as proxies of exposure [16–19]. The only cohort study relying on expert exposure assessment of diesel EE reported null findings based on 302 exposed cases, including in the highest exposure category [20]. Case–control studies [21–25] applying expert judgment or job-exposure matrices were used to assess exposure to EEs from different fuels but all were limited by very small sample sizes. To our knowledge, no previous study investigated prostate cancer aggressiveness or the possible role of cancer detection or screening in associations with EEs. These limitations are addressed in the current study, the largest and most detailed to date on this issue.

Methods

Study design

We used data from the Prostate Cancer & Environment Study (PROtEuS), a population-based case–control study carried out in Montreal, Canada in 2005–2012, and specifically conceived to investigate occupational exposures. Study details have been presented elsewhere [26, 27]. Eligible individuals were men aged 75 years or less, residents of Greater Montreal and registered on the (continually updated) electoral list.

Incident prostate cancer cases were actively ascertained across seven French-language Montreal hospitals, representing 80% of all prostate cancer cases diagnosed in the area during the study period according to the provincial tumor registry. Gleason scores at diagnosis, extracted from pathology reports, were used to characterize disease aggressiveness [28]. Throughout the recruitment period, controls, residing in the same area and frequency-matched to cases on age (± 5 years), were randomly selected from the electoral list of French-speaking men. In all, 1,924 cases and 1,989 controls were enrolled into the study (participation rates of 79% and 56% for cases and controls, respectively). Refusals were the main reason (86%) for non-participation. For 3% of cases and 4% of controls, information was collected from proxy respondents.

Data collection

Participants were interviewed face-to-face about their sociodemographic characteristics, lifestyle, medical history, anthropometric factors and occupational history.

Job titles were elicited for all paid jobs of one year or more. For jobs held for at least 2 years, extensive details on chemical agents and equipment used, tasks, protective measures and workplace characteristics were collected. Specialized questionnaires ($n=32$) probing for further details were used for complex occupations such as drivers, miners, mechanics, gas station attendants or firefighters.

PROtEuS was approved by the ethics boards of all participating institutions and all participants provided written informed consent.

Exposure assessment of EEs

The Canadian Classification Dictionary of Occupations and the Standard Industrial Classification were used to code occupations and industries [29, 30]. A team of chemists-industrial hygienists used the hybrid expert-based approach [31], which combines job exposure profiles and expert-based assessment, to assign exposures. As compared to the traditional expert-based method [32], this approach provides clearer guidelines for coding, improves on efficiency, comprehensiveness and transparency of assignments, and confidence of ratings by experts [31]. Each of the 16 065 detailed job descriptions was assigned exposure to EE from leaded and unleaded gasoline, any diesel, light- and heavy-duty diesel, jet fuel, and propane, along with hundreds of other chemicals, without knowledge of the participants case/control status. Final codings were based on a consensus between experts.

For each agent, in each job, experts assigned three separate semi-quantitative parameters of exposure: the reliability (degree of confidence that the exposure occurred: possible, probable, definite), intensity level (low, referring to exposure above the environmental background level experienced by the general population, medium, high) and frequency (proportion of working time in which the exposure occurred in a typical workweek, i.e., < 5%, 5%−30%, > 30%−90% or > 90%).

For each EE, we defined ever exposure as having been substantially exposed in at least one job, that is, at medium or high intensity over ≥ 5% of working time, with a probable or definite reliability of exposure.

In order to better reflect the estimated average latency in the development of prostate cancer, we restricted exposed participants to those whose exposure occurred before the 5 years preceding the index date (diagnosis or interview).

The duration of exposure to each EE across all jobs and cumulative exposure (CE) were calculated. For the latter, intensity values of 1, 2 and 3 were converted to 1, 5 and 25, respectively, to better reflect the quantitative exposure levels predicted by hygienists [33]. There was no universal consensus to assign quantitative levels to the three intensity categories for the hundreds of agents evaluated in the study, each of which could exhibit different quantitative meanings. Expert coders agreed that the 1:5:25 ratio based on a lognormal distribution of exposure levels, rather than 1:2:3 or 1:10:100, reflected best the relative intensity levels meant by hygienists of low:medium:high for most exposure situations, including for engine emissions. It was thus retained for the computation of intensity levels [34].

Similarly, initial semi-quantitative codings for frequency were converted to 2.5%, 17.5%, 60% and 95% of workweek, based on corresponding values in an analogous study conducted by our group in Montreal [35].

CE was the result of summation of the product of the intensity, frequency and duration across all exposed jobs as follows:

$$CE = \sum_{i=1}^{k} (I_i \times F_i \times D_i)$$

where I_i is the intensity of exposure in job i, F_i the frequency of exposure in the job and D_i is the job duration.

Potential confounders

Our minimal model was adjusted for age only, modelled continuously after confirming linearity of the logit. The full model also incorporated potential confounders identified from causal Directed Acyclic Graphs (DAGs) drawn from assumptions derived from the literature (Supplemental Figure S1). There are few recognized risk factors for prostate cancer, so the full model includes some factors for which evidence is still limited. As a result, consideration of both a minimal and a full model was felt indicated. In the full model, co-variables included age, ancestry (Sub-Saharan, Asian, French, Other European, Greater Middle Eastern, Latino, other), educational level (primary school or less, high school, college, university, other), alcohol drinking [36, 37] (drink-years), cigarette smoking [38, 39] (ever/never and cigarette-years) and body mass index [40] (continuous, in kg/m^2). Analyses investigating the role of gasoline, diesel and propane EEs were additionally adjusted for occupational factors i.e., cumulative occupational exposures to benzene, pesticides, ever farming (the latter two being weakly related in this largely urban population), and ever firefighting [9, 27, 41]. Analyses on jet fuel EE were adjusted for all previous factors apart from ever farming and occupational exposure to pesticides. We did not adjust for nightshift work which was found not to be a risk factor in PROtEuS [26].

Statistical analyses

Unconditional logistic regression was used to model associations between exposure to EEs and prostate cancer risk. As associations were not linear, odds ratios (ORs) and 95% confidence intervals (CI) were computed from fitted functions for changes between exposure percentiles, from the 5th to the 25th, the 25th to the 50th, the 50th to the 75th and the 75th to the 95th using a method that was previously developed for natural cubic spline functions [42].

Prostate cancers included 1,488 cases of the non-aggressive form of the tumour (Gleason scores ≤ 6 or 7 [with 3 as primary score and 4 as secondary score] and 436 aggressive cancers (Gleason scores ≥ 8 or 7 [with 4 as primary score and 3 as secondary score]) [28]. As the latter have been hypothesized to have a different set of risk factors, including occupational [27], and distinct aetiologies [43] than non-aggressive cancers [3], we conducted separate analyses for overall and aggressive prostate cancers.

Several sensitivity analyses were performed: (1) excluding controls who had not been tested for prostate cancer within the prior two years to reduce the likelihood of latent undiagnosed cancers in the control series; (2) considering a more stringent definition of aggressive tumors (Gleason scores ≥ 8) [44]; (3) increasing the latency period from 5 to 10 years; and (4) investigating associations in younger and older participants separately, as environment-induced prostate cancers may occur more often in older men whereas younger cases (≤ 55 years) would be more likely to result from genetic causes [45].

All analyses were performed using R Statistical software (v4.2.3; R Core Team 2022).

Table 1 Selected characteristics of cases and controls, PROtEuS, Montreal, Canada, 2005–2012

	Prostate cancer cases ***n*** **= 1,924** **n (%)**	**Controls** ***n*** **= 1,989** **n (%)**
Age (years)		
Mean ± sd	63.6 ± 6.8	64.8 ± 6.9
Ancestry		
Sub-Saharan African	129 (6.7)	89 (4.5)
Asian	24 (1.3)	72 (3.5)
French	1439 (75.0)	1243 (62.5)
Other European	245 (12.5)	438 (22.0)
Greater Middle Eastern	45 (2.3)	99 (5.0)
Latino	29 (1.5)	31 (1.6)
Other	1 (0.1)	3(0.2)
Missing	12 (0.6)	14 (0.7)
Level of education		
Primary school or less	448 (23.3)	427 (21.4)
High school	571 (29.6)	576 (29.0)
College	312 (16.2)	374 (18.8)
University	588 (30.6)	610 (30.7)
Other	5 (0.3)	2 (0.1)
Number of years since last PSA and/or DRE		
≤ 2	1904 (99.0)	1509 (75.9)
2.01–5	1 (0.1)	152 (7.5)
> 5	0 (0.0)	81 (4.1)
Never tested	3 (0.2)	191 (9.6)
Do not know if ever tested	2 (0.1)	29 (1.5)
Was tested but do not know when	14 (0.6)	27 (1.4)

Abbreviations: *PSA* prostate specific antigen, *DRE* digital rectal examination

Results

Overall, 1,924 prostate cancer cases and 1,989 controls were included in the analyses (Table 1). Participants were mostly of French descent. Controls were slightly older than cases and tended to be more educated. Seventy-six percent of controls and 99% of cases had undergone a prostate specific antigen (PSA) test and/or a digital rectal examination (DRE) in the two years preceding the interview.

Supplemental table S2 describes EE exposure codings associated with common occupations. Truck driving was the most frequent EE-exposed occupation with 398 jobs held. Exposure to leaded gasoline EE, unleaded gasoline EE and any diesel EE occurred most often at low intensity.

Exposure to leaded gasoline EE at high intensity mostly occurred in timber cutting occupations, concomitantly with low-intensity exposure to diesel EE. Mail carriers, bus drivers and business services salesmen had the highest prevalence of exposure to unleaded gasoline EE, at low intensity. All occupational groups with ≥ 50% prevalence of exposure to diesel EE were exposed to light-duty diesel EE, more specifically.

The top three occupational groups with highest prevalence of exposure to diesel EE were excavating, grading and related occupations, bus drivers and timber cutting occupations. Heavy-duty diesel EE occurred less frequently than light-duty diesel and was most often encountered at low intensity among foremen of mechanics and repairmen, and among inspecting and testing occupations, and equipment repair. Jobs in electrical and related equipment holding occupations involving installing and repairing were the only ones exposed to heavy-duty diesel EE at high intensity. Material handling equipment operators were exposed to propane EE at medium intensity but not to other EEs. Exposure to jet fuel EE mostly occurred in aircraft

mechanics and repairmen and in air transport operating support occupations.

Cumulative levels of exposure to each EE within the study population, along with the numbers of exposed cases, are shown in Table 2, while correlations between EEs are presented in supplemental Figure S1. The strongest correlations were found between leaded gasoline EE and unleaded gasoline EE ($r=0.53$), leaded gasoline EE and light-duty diesel EE ($r=0.46$) and unleaded gasoline EE and light-duty diesel EE ($r=0.34$).

Figure 1 shows the marginal fully-adjusted prostate cancer response patterns associated with EEs. Responses functions for EEs from leaded and unleaded gasoline, any diesel and light-duty diesel were quite similar and showed slight increases in risk for low levels of exposure. The risk then decreased monotonically with increased exposures, which had wide confidence intervals owing to small numbers. Response patterns for EEs from heavy-duty diesel, propane and jet fuel showed decreased risks regardless of the exposure level, based on few exposed participants.

Table 3 shows associations between cumulative levels of exposure to EEs and prostate cancer risk, for the age-adjusted and full models. Results are presented for overall and high-grade cancers, as no associations emerged for low-grade tumors (data not shown).

Leaded gasoline EE

Based on the age-adjusted model, a change from the 75th to the 95th percentile in leaded gasoline EE distribution yielded an OR of 1.21 (95%CI 0.91 to 1.60) for overall

Table 2 Distributions of cumulative levels of exposure[a] to engine exhausts, overall and by disease status, PROtEuS, Montreal, Canada, 2005–2012

Engine exhaust (EE) n Ca/n Co	Min	5th percentile	25th percentile	Median	75th percentile	95th percentile	Max	Mean	Interquartile range	Standard deviation
Leaded gasoline EE (720/697)										
Total	3	18	60	158	383	2,743	19,688	548	323	1,419
Cases	3	16	48	150	360	2,791	19,688	537	312	1,413
Controls	3	18	68	174	420	2,700	17,438	560	352	1,426
Unleaded gasoline EE (759/731)										
Total	1	15	63	146	420	1,000	16,800	386	357	1,018
Cases	3	15	63	140	400	1,000	13,125	358	337	856
Controls	1	15	65	150	441	1,000	16,800	415	376	1,162
Any diesel EE (597/596)										
Total	2	15	50	150	500	1,900	15,500	452	450	1,003
Cases	3	13	50	150	475	1,570	6,188	401	425	685
Controls	2	15	45	150	520	2,100	15,500	504	475	1,241
Heavy-duty diesel EE (18/25)										
Total	20	45	90	203	680	3,000	14,500	845	590	2,245
Cases	20	20	145	290	938	1,688	1,688	520	793	529
Controls	60	60	90	206	480	3,200	1,4500	1079	390	2,913
Light-duty diesel EE (595/593)										
Total	2	15	50	150	506	1,920	15,694	456	456	1,016
Cases	3	13	50	150	475	1,571	6,265	403	425	694
Controls	2	15	45	150	526	2,126	15,694	509	481	1,258
Propane EE (98/108)										
Total	3	9	55	123	420	1,600	17,500	448	365	1,334
Cases	5	9	70	133	420	2,363	3,320	399	350	631
Controls	3	9	50	120	419	1,600	17,500	493	369	1,744
Jet fuel EE (15/50)										
Total	3	6	26	90	500	3,200	15,500	659	474	2,043
Cases	6	6	17	125	500	2,200	2,200	334	483	557
Controls	3	5	26	83	650	3,800	15,500	756	624	2,307

Abbreviations: *Ca* cases, *Co* controls, *EE* engine exhaust

[a] Cumulative exposure levels were calculated by summing the product of the intensity, frequency and duration across all exposed jobs (this index has no units)

prostate cancer risk and 1.36 (95%CI 0.88 to 2.11) for high-grade tumors. Fully adjusted models generally led to decreased estimates. ORs for changes in other exposure percentiles were around unity.

Unleaded gasoline EE

Increasing the level of exposure to unleaded gasoline EE across percentiles showed no association with overall prostate cancer risk. However, a change from the 75th to the 95th percentile of the exposure distribution was associated with a slight increase in risk of high-grade cancers in the age-adjusted model (OR = 1.16, 95%CI 0.98 to 1.37).

Any diesel EE

Increased levels of exposure to any diesel EE were not associated with overall prostate cancer risk. For high-grade cancers, changes from the 25th to the 95th percentile of the exposure distribution showed ORs > 1 both in the age-adjusted and the full models. Estimates were somewhat stronger for exposures from the 25th to the 75th percentile of the distribution, i.e., $OR_{age\text{-}adj.} = 1.31$ (95%CI 1.04 to 1.65) and $OR_{full\ model} = 1.24$ (95%CI 0.96 to 1.61).

Light- and heavy-duty diesel EE

Findings for light-duty diesel EE exhibited similar patterns of increased risk as observed for any diesel EE. Contrastingly, there was no evidence of excess risk with heavy-duty diesel EE, to which exposure was infrequent.

Propane and jet fuel EE

Exposure to propane and jet fuel EE showed no association with prostate cancer risk, based on limited numbers.

We also examined prostate cancer risk associated with exposure to the various EEs, in isolation and in combination, for those with sufficient numbers (at least 10 exposed cases), using men never exposed to any engine exhaust as the reference category (supplemental file – Table S2). For overall prostate cancer, the OR for exposure to both leaded gasoline and diesel light EEs was 1.25 (95%CI 0.87 to 1.80) and it was 1.20 (95%CI 0.81 to 1.78) for exposure to unleaded gasoline and diesel light EEs. Regarding high-grade cancers, based on limited numbers of participants, the OR for exposure to leaded and unleaded gasoline EEs was 1.24 (0.82 to 1.88), for exposure to leaded gasoline and diesel light EEs it was 1.27 (95%CI 0.72 to 2.26) and 1.32 (95%CI 0.72 to 2.42) for common exposure to unleaded gasoline and diesel light EEs. Exposure to propane EE was rarely found in combination with other EEs (data not shown).

Sensitivity analyses

Excluding controls not recently tested for prostate cancer, increasing the latency period from 5 to 10 years and considering aggressive tumors as those with Gleason scores of 8 or more led to lowered risk estimates and wider confidence intervals for all EEs (data not shown), with a few exceptions. Using the more stringent definition of aggressive tumors, a change from the 75th to the 95th percentile distribution of leaded gasoline EE was associated with an age-adjusted OR of 1.59 (95% CI 0.92 to 2.75) and of 1.16 (95% CI 0.52 to 2.58) for the full model. For any diesel EE, the ORs associated with percentile changes from the 25th to the 75th were 1.55 (95% CI 1.14 to 2.10) and 1.39 (95% CI 0.98 to 1.98), based on the age-adjusted and full models, respectively.

We found no statistically significant interaction of age groups, using the 55-year cut point, between occupational exposure to EEs and prostate cancer (data not shown), although there were relatively few men with early-onset diagnoses. Nor were there multiplicative interactions between exposure to EEs and any of the non-occupational covariates under consideration.

Discussion

While there was no evidence in our data of elevated risks of overall prostate cancer associated with occupational exposure to EEs, there were suggestions that high levels of exposure to EEs resulting from the combustion of diesel (any), light-duty diesel and from leaded gasoline may contribute to the development of aggressive disease. Adjustment for potential confounders, considering a longer latency period and prostate cancer screening patterns, resulted in wider confidence intervals and attenuated associations.

Occupational exposure to engine exhausts has received limited attention with respect to a possible role in

(See figure on next page.)

Fig. 1 Marginal, fully-adjusted response functions for the risk of prostate cancer for each engine exhaust (EE) exposure measured using normalized cumulative exposure values. The Y axis represents the predicted probability of prostate cancer (%); the X axis represents EE normalized cumulative exposures (dimensions without unit). The solid line represents the maximum likelihood estimates using a natural cubic spline function on 3 degrees of freedom. The pointwise 95% confidence intervals are presented by the gray bands. The rug plots at the top refer to values for cases and the bottom refers to controls. Cumulative exposure values of: **A**, leaded gasoline EE. **B**, unleaded gasoline EE. **C**, any diesel EE. **D**, heavy-duty diesel EE. **E**, light-duty diesel EE. **F**, propane EE. **G**, jet fuel EE

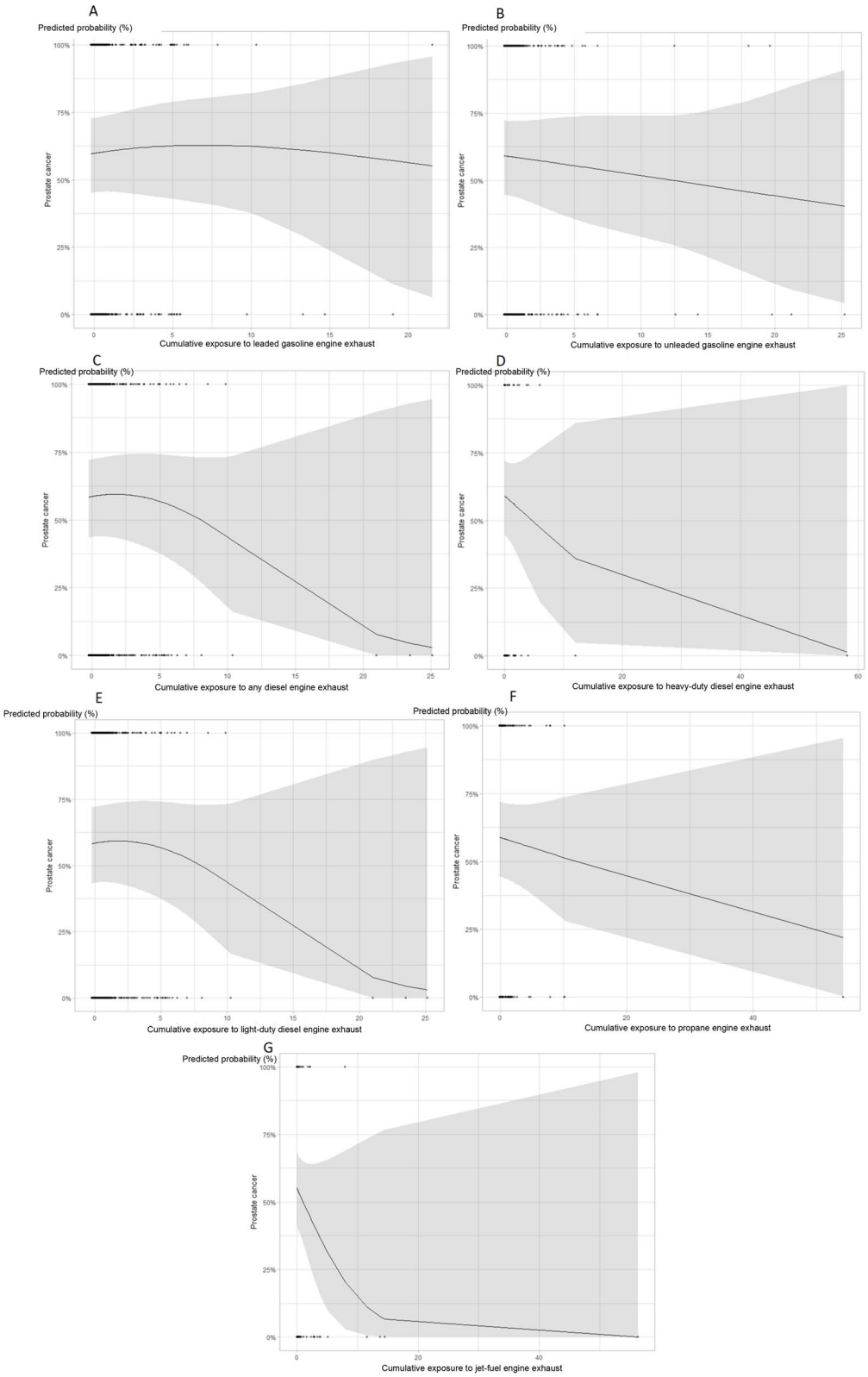

Fig. 1 (See legend on previous page.)

Table 3 Associations between changes in cumulative exposure distributions of engine exhausts (EEs) and prostate cancer risk, for overall and high-grade cancers, PROtEuS, Montreal, Canada, 2005–2012

Engine exhaust (EE)	Minimal model[a]	Full model[b]
	OR (95%CI)	OR (95%CI)
Leaded gasoline EE		
All cancers		
Change from 5 to 25th percentile (18 to 60)	1.01 (1.00 to 1.01)	1.00 (0.99 to 1.01)
Change from 25 to 75th percentile (60 to 383)	1.04 (0.98 to 1.09)	1.01 (0.95 to 1.08)
Change from 75 to 95th percentile (383 to 2,743)	1.21 (0.91 to 1.60)	1.06 (0.75 to 1.51)
High-grade cancers		
Change from 5 to 25th percentile (18 to 68)	1.00 (0.99 to 1.02)	1.00 (0.99 to 1.02)
Change from 25 to 75th percentile (68 to 428)	1.05 (0.96 to 1.15)	1.01 (0.90 to 1.14)
Change from 75 to 95th percentile (428 to 2,850)	1.36 (0.88 to 2.11)	1.12 (0.63 to 2.02)
Unleaded gasoline EE		
All cancers		
Change from 5 to 25th percentile (15 to 63)	1.00 (0.99 to 1.02)	1.00 (0.99 to 1.01)
Change from 25 to 75th percentile (63 to 420)	1.04 (0.96 to 1.12)	0.99 (0.90 to 1.08)
Change from 75 to 95th percentile (420 to 1,000)	1.05 (0.93 to 1.18)	0.98 (0.85 to 1.12)
High-grade cancers		
Change from 5 to 25th percentile (20 to 65)	1.01(1.00 to 1.03)	1.01 (0.98 to 1.02)
Change from 25 to 75th percentile (65 to 441)	1.12 (0.98 to 1.27)	1.03 (0.89 to 1.21)
Change from 75 to 95th percentile (441 to 1,000)	1.16 (0.98 to 1.37)	1.05 (0.85 to 1.29)
Any diesel EE		
All cancers		
Change from 5 to 25th percentile (15 to 50)	1.01 (1.00 to 1.02)	1.00 (0.99 to 1.01)
Change from 25 to 75th percentile (50 to 500)	1.06 (0.94 to 1.20)	1.03 (0.91 to 1.16)
Change from 75 to 95th percentile (500 to 1,900)	1.05 (0.84 to 1.32)	1.00 (0.77 to 1.30)
High-grade cancers		
Change from 5 to 25th percentile (15 to 52)	1.03 (1.00 to 1.05)	1.02 (1.00 to 1.04)
Change from 25 to 75th percentile (52 to 520)	1.31 (1.04 to 1.65)	1.24 (0.96 to 1.61)
Change from 75 to 95th percentile (520 to 2,100)	1.26 (0.84 to 1.91)	1.27 (0.80 to 2.01)
Heavy-duty diesel EE		
All cancers		
Change from 5 to 25th percentile (45 to 90)	0.99 (0.96 to 1.01)	0.98 (0.95 to 1.02)
Change from 25 to 75th percentile (90 to 680)	0.83 (0.60 to 1.14)	0.79 (0.50 to 1.26)
Change from 75 to 95th percentile (680 to 3,000)	0.48 (0.14 to 1.71)	0.41 (0.07 to 2.45)
High-grade cancers		
Change from 5 to 25th percentile (60 to 90)	0.88 (0.71 to 1.10)	0.90 (0.72 to 1.12)
Change from 25 to 75th percentile (90 to 480)	0.20 (0.01 to 3.50)	0.25 (0.01 to 4.42)
Change from 75 to 95th percentile (480 to 3,200)	-	-
Light-duty diesel		
All cancers		
Change from 5 to 25th percentile (15 to 50)	1.01 (1.00 to 1.02)	1.00 (0.99 to 1.01)
Change from 25 to 75th percentile (50 to 506)	1.06 (0.94 to 1.20)	1.03 (0.91 to 1.16)
Change from 75 to 95th percentile (506 to 1,920)	1.05 (0.83 to 1.31)	1.00 (0.76 to 1.30)
High-grade cancers		
Change from 5 to 25th percentile (15 to 50)	1.02 (1.00 to 1.05)	1.02 (1.00 to 1.04)
Change from 25 to 75th percentile (50 to 527)	1.31 (1.04 to 1.65)	1.24 (0.96 to 1.61)
Change from 75 to 95th percentile (527 to 2,126)	1.26 (0.84 to 1.91)	1.27 (0.80 to 2.00)
Propane		
All cancers		

Table 3 (continued)

Engine exhaust (EE)	Minimal model[a]	Full model[b]
Change from 5 to 25th percentile (9 to 55)	0.99 (0.98 to 1.01)	0.99 (0.98 to 1.00)
Change from 25 to 75th percentile (55 to 420)	0.96 (0.88 to 1.04)	0.94 (0.86 to 1.03)
Change from 75 to 95th percentile (420 to 1,600)	0.87 (0.66 to 1.14)	0.83 (0.62 to 1.12)
High-grade cancers		
Change from 5 to 25th percentile (9 to 50)	0.99 (0.97 to 1.02)	0.99 (0.95 to 1.02)
Change from 25 to 75th percentile (50 to 400)	0.93 (0.75 to 1.16)	0.88 (0.66 to 1.18)
Change from 75 to 95th percentile (400 to 1,600)	0.78 (0.37 to 1.68)	0.65 (0.24 to 1.76)
Jet fuel [c]		
All cancers		
Change from 5 to 25th percentile (6 to 26)	0.98 (0.96 to 1.00)	0.98 (0.97 to 1.00)
Change from 25 to 75th percentile (26 to 500)	0.59 (0.35 to 1.00)	0.69 (0.44 to 1.08)
Change from 75 to 95th percentile (500 to 3,200)	0.05 (0.01 to 1.01)	0.12 (0.09 to 1.58)
High-grade cancers		
Change from 5 to 25th percentile (5 to 26)	0.95 (0.88 to 1.03)	0.96 (0.89 to 1.04)
Change from 25 to 75th percentile (26 to 600)	0.26 (0.03 to 2.50)	0.35 (0.04 to 3.07)
Change from 75 to 95th percentile (600 to 3,800)	-	-

Abbreviations: *OR* odds ratio, *CI* confidence intervals

a Adjusted for age

b Adjusted for age, ancestry, educational level, alcohol drinking, cigarette smoking, body mass index and (for analyses on gasoline, diesel and propane EE) cumulative occupational exposures to benzene and pesticides, ever farming and ever firefighting

c Models not adjusted for ever farming and occupational exposure to pesticides

prostate cancer development. A few studies have looked into diesel EE, in light of its high carcinogenic potential [10] and its presence in occupations repeatedly associated with elevated prostate cancer risks, such as farmers [46–48] and firefighters [6]. Cohort studies, most often using job titles as surrogate for exposure to diesel EE [16–19], observed increased incidence or mortality by prostate cancer in drivers [17], locomotive drivers [16], truck drivers [18], firefighters [6] and farmers [49, 50]. Contrastingly, no excess of risk or death by prostate cancer were reported in some other occupations including heavy equipment operators [18] and miners [19]. Potential confounding by known or suspected risk factors was rarely addressed in these studies.

Only a handful of studies have investigated the role of occupational exposure to diesel EEs per se in prostate cancer. A prospective cohort study found no evidence of excess incidence rates ratios of prostate cancer associated with the cumulative probability of exposure to diesel exhaust based on information on company name and type, product type and employment period [20]. Exposure to diesel EE, assessed using a job-exposure matrix, was found unrelated to prostate cancer mortality in an occupational cohort representing the Canadian workforce [51]. Case–control studies, focusing on overall prostate cancer, generally used expert-based exposure assessment protocols [21, 22, 24, 25] or a job-exposure matrix [23] to infer exposure to diesel EE, but these were hampered by small samples (5 to 213 exposed cases). No associations were found with substantial exposure in some studies [21, 22, 25], while others observed positive associations with ever [21, 24], long duration [22] and high cumulative [23] exposure.

Our work distinguished EE exposures from light- and heavy-duty diesel, which are characterized by different mixtures. Similar associations were found for any diesel EE and light-duty diesel EE, reflecting the preponderance of the latter among diesel EE-exposed participants. We found some evidence of elevated risks of aggressive prostate cancer associated with exposure to diesel (any) and light-duty diesel EE. One other study investigated tumor invasiveness reported no association with diesel EE [20], so did a mortality study [51].

Gasoline EE contains a mix of chemicals, and frequent co-exposure with diesel EE makes it difficult to isolate its own carcinogenic effect. Two small case–control studies of prostate cancer reported null associations with ever exposure to gasoline EE [21, 24]. Our study is the first to distinguish unleaded from leaded gasoline EE. We found a slight increase in risk of aggressive cancer among men highly exposed to the lead-containing formulation. Lead is as a powerful endocrine disruptor [52], notably due to its ability to mimic sexual hormones such as androgens, known to be involved in the normal and the pathological growth of the prostatic gland.

Propane is a gas considered to be safe, unexpensive and environmentally friendly, and is frequently used for power generation, forklifts, vehicles, cooking and

heating. Mutagenic or endocrine disrupting properties have not been reported [53]. There was no evidence in our data that exposure to propane EE might increase prostate cancer risk. Some elevated risk estimates with ever exposure were reported previously, but these were based on very few exposed cases [21, 22].

Jet fuels are kerosene-type fuels that contain different additives to meet the needs of their end use, such as civil or military aviation. These fuels contain varying amounts of paraffins, napthalenes, aromatics, olefins, antioxidants, metal deactivators or corrosion inhibitors [54]. Direct occupational exposure to jet fuel EE occurs in the aircraft industry, particularly when manually filling airplanes, during maintenance, inspection and cleaning of jet fuel storage tanks or production and installation of aircraft systems [54]. Flight attendants and pilots have also been reported to be occasionally exposed to EE from these fuels [55].

An increased cancer risk has been reported among aircraft maintenance workers [56], while an inverse association was reported for prostate cancer [9]. Investigations on flight attendants showed an increased risk of this cancer [57, 58]**.** However, air workers are exposed to a complex scenario of organizational (i.e., nightshift work), physical (i.e., ionizing radiation of cosmic origin) and chemical (i.e., hexavalent chromium) exposures, making it difficult to isolate individual health effects.

The role of jet fuel EE per se in prostate cancer risk has been examined in a single study that reported an OR of 0.7 in men ever occupationally exposed [21]. The current study is the first to investigate more informative metrics for this exposure.

Our study provides novel evidence suggesting that some EEs may be implicated in the development of high-grade prostate cancers more specifically. Recent findings suggest that grade may be established early in tumour pathogenesis and that Gleason grade progression is uncommon [59]. Low-grade prostate cancer has been shown to diverge early from high-grade cancer, and there appears to be no direct progression from low grade to metastatic disease [43]. These observations suggest that low-grade and high-grade prostate cancers may share different risk factor sets.

To our knowledge, our study is the first to document the role of occupational exposure to defined combinations of EE in prostate cancer risk, expanding beyond the one single exposure – one disease paradigm.

Some methodological considerations should be taken into account in the interpretation of our results. Some analyses suffered from limited statistical power, decreasing our ability to detect moderate or weaker associations. This was particularly the case for exposure to certain combinations of EE, for which the number of exposed subjects, especially with high-grade cancers, was limited, leading to imprecise risk estimates.

Jobs lasting less than 2 years did not contribute to exposure. These represented only 4% of work-years, and showed small differences with longer-jobs in terms of chemical exposures [60]. Finally, although the overall participation rates were relatively good in our study, the lower rates among controls could have introduced selection bias. However, only marginal differences were found for census tract indicators based on the residence of participants and non-participants, including the percentage of recent immigrants, unemployment, education and income levels.

This study benefits from several strengths. The number of exposed cases largely exceeds most previous investigations on this subject. The expert-based exposure assessment, thought to be the gold-standard for retrospective community-based studies [61, 62], enabled us to study various EEs. Moreover, covering a wide range of occupational circumstances diminishes the likelihood of strong confounding by factors commonly encountered in a specific occupation. We previously observed that prostate cancer screening behaviours varied between occupations [63]. The high overall screening uptake in our population at the time of study, along with individually-based screening information, enabled us to rule out with reasonable certainty that our findings reflected detection patterns.

This study contributes novel findings, notably by investigating the distinctive role of different formulations of gasoline and diesel EEs, whose compositions can contain varying levels of chemicals with carcinogenic or hormone disrupting properties. Although residual confounding cannot be ruled out, we had access to a wide range of lifestyle and occupational co-factors. The ability to investigate cancer aggressiveness and to examine the potential impact of undiagnosed prostate cancers in our control series are other advantages of this study.

Conclusion

This study provides evidence that occupational exposure to engine exhausts resulting from the combustion of any and light-duty diesel, and of leaded gasoline, may increase the risk of developing aggressive prostate cancer.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12940-025-01205-3.

Supplementary Material 1: Supplemental file 1. Directed acyclic graph for the association between engine exhaust (EE) and prostate cancer risk, PROtEuS, Montreal, Canada, 2005–2012. Supplemental file 2. Most frequent* occupations classified as having probable or definite exposure, PROtEuS, Montreal, 2005–2012. Supplemental file 3. Spearman correlation coefficients between cumulative exposures to engine exhaust (EE) among controls, PROtEuS, Montreal, Canada, 2005–2012. Supplemental file 4. Associations between occupational exposures to engine exhaust (EE), in isolation or combined, and prostate cancer risk.

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Clinical trial number
Not applicable.

Authors' contributions
C.B conducted the data analysis and prepared the manuscript. M-E.P conceived and led the PROtEuS study. C.B, M-C.R and M-E.P contributed to the interpretation of the data and critically revised the manuscript. All authors read and approved the final version of the manuscript.

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Data availability
As per ethics requirements, this dataset cannot be shared.

Declarations

Ethics approval and consent to participate
This study was approved by the Ethics Committees of the following institutions: Institut national de la recherche scientifique, Centre de Recherche du Centre Hospitalier de l'Université de Montréal, Hôpital Maisonneuve-Rosemont, Hôpital Jean-Talon, Hôpital Fleury, and Hôpital Charles-LeMoyne. All participants provided written informed consent.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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