European Journal of Preventive Cardiology

ESC European Society of Cardiology

Full research paper

European Journal of Preventive Cardiology
2018, Vol. 25(13) 1397–1405

DOI: 10.1177/2047487318769458
journals.sagepub.com/home/ejpc

Relations between the residential fast-food environment and the individual risk of cardiovascular diseases in The Netherlands: A nationwide follow-up study

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Abstract

Background: The food environment has been hypothesized to influence cardiovascular diseases such as hypertension and coronary heart disease. This study determines the relation between fast-food outlet density (FFD) and the individual risk for cardiovascular disease, among a nationwide Dutch sample.

Methods: After linkage of three national registers, a cohort of 2,472,004 adults (≥35 years), free from cardiovascular disease at January 1st 2009 and living at the same address for ≥15 years was constructed. Participants were followed for one year to determine incidence of cardiovascular disease, including coronary heart disease, stroke and heart failure. Street network-based buffers of 500 m, 1000 m and 3000 m around residential addresses were calculated, while FFD was determined using a retail outlet database. Logistic regression analyses were conducted. Models were stratified by degree of urbanization and adjusted for age, sex, ethnicity, marital status, comorbidity, neighbourhood-level income and population density.

Results: In urban areas, fully adjusted models indicated that the incidence of cardiovascular disease and coronary heart disease was significantly higher within 500 m buffers with one or more fast-food outlets as compared with areas with no fast-food outlets. An elevated FFD within 1000 m was associated with an significantly increased incidence of cardiovascular disease and coronary heart disease. Evidence was less pronounced for 3000 m buffers, or for stroke and heart-failure incidence.

Conclusions: Elevated FFD in the urban residential environment (≤1000 m) was related to an increased incidence of cardiovascular heart disease and coronary heart disease. To better understand how FFD is associated with cardiovascular disease, future studies should account for a wider range of lifestyle and environmental confounders than was achieved in this study.

Keywords

Cardiovascular diseases, population register, environmental exposure, fast food, incidence

Received 16 November 2017; accepted 17 March 2018

Introduction

Cardiovascular diseases (CVDs) significantly contribute to the global morbidity and mortality.[1] The increased number of people suffering from CVD has paralleled changes in the food environment toward

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large scale, inexpensive and calorie-dense food production.[2,3] These changes have influenced the population's food consumption, which has been hypothesized to affect the development of CVD.[4–6] The proposed environmental influence that shapes food consumption and, in succession, health aligns with socio-ecological theories.[7,8]

Food that is known as 'fast food' generally consists of processed meat and refined carbohydrates and is high in salt, saturated fat and calories.[9] Worldwide, fast-food availability has expanded rapidly. For example, the number of McDonalds outlets increased by almost 20% globally in the past decade (2005–2016).[10] Fast-food availability has also increased considerably in European countries over the past years.[11,12] Several studies have investigated the link between fast-food availability and the prevalence of CVD or CVD risk factors. The findings are mixed, but that might be caused by different fast-food exposure assessments and CVD outcome measures.[13–19] Most studies determined fast-food outlet density (FFD) for crude predefined areas (e.g. census tract), rather than including measures representing the FFD around people's residential home. In addition, most studies determined CVD *prevalence* within the predefined area, rather than the *incidence* of CVD.[13–18] Additionally, most studies did not account for the time that individuals were exposed to fast-food outlets within the studied area.[13–19]

The aim of this study was to contribute to the literature by determining the relation between FFD and one year incidence of individual-level CVD among a nationwide sample in The Netherlands. This manuscript provides a novel contribution by addressing four key points. First, our study will provide a longitudinal perspective on the fast-food environment and CVD by including CVD incidence rather than prevalence, derived from a large population-wide cohort. Second, only individuals living at the same address for a long period of time were included in order to eliminate influences of prior residential environments for a substantial timeslot. Third, this study was unique in presenting the incidence of individual-level CVD and three subtypes (coronary heart disease (CHD), stroke and heart failure), providing more detail in the studied relations. Finally, instead of geometric buffers we applied more accurate street network buffers of different sizes around the home addresses of participants in both urban and rural areas.[20]

Methods

Study design

A one-year follow-up study among a nationwide sample in The Netherlands was conducted.

Datasets

Several Dutch national registers were linked: population register, hospital discharge register (HDR), national cause of death register (NCDR), regional income survey (RIS), and the Locatus database of business addresses. The population register contains information on all legally residing citizens in The Netherlands, including date of birth, sex, current and previous address, postal code and nationality. The HDR registers medical and administrative data for all admitted and day clinic patients visiting a Dutch hospital. The HDR contains information on patients' demographics, admission data and primary and secondary diagnoses at admission. The primary discharge diagnosis is determined at discharge and coded using the ninth version of the international classification of disease codes (ICD-9 codes).[21] The NCDR contains information on date of death and causes of death. The overall validity of these registers has been proven to be high.[22,23] The RIS is a longitudinal survey primarily based on tax information that started in 1994 with a representative sample of over two million households in The Netherlands. This accounts for roughly one-third of the Dutch population and is corrected each year for migration, deceased residents and new-borns.[24] Locatus maintains a database with independently sourced retail information via annual on-site surveys from which typical outlets selling fast food were extracted over 2009 (fast-food outlets (#59.210.171); delivery/take-away outlets (#59.210.180); grillroom/kebab-outlets (#59.210.215)).

Cohort identification

To construct a cohort we selected everyone in the population register at January 1st 2009 aged 35 years and older, and living at the same address for at least 15 years. Next, we linked these individuals with the HDR using a personal identifier based on linkage variables 'sex', 'date of birth', '4-digits of postal code'. Approximately 85% of the entire Dutch population has a unique combination of date of birth, sex and postal code (i.e. occur only once in the registry and thus identify one person) and these were included.[22] All persons with a hospital admission for CVD since January 1st 1995 were excluded.

Outcome measures

Through linkage with the HDR and NCDR individuals were able to be tracked over time for incident CVD events (ICD-9 codes for CVD admissions and ICD-10 codes for CVD deaths in and outside the hospital provided in Supplementary Material Table 1 online). One-year incident was defined as the number of first individual-level CVD hospital admissions or out of

hospital deaths for CVD or CVD subtypes within one year after January 1st 2009.

Determinants

FFD – street-network buffer sizes. FFD was defined as the number of fast-food outlets within network buffers around an individual address. Three different street network buffer sizes around individuals' addresses were calculated using ArcGIS 10.1 (Esri, Redlands, CA, US) and included; 500 meter (m), 1000 m (both easily to walk[25]) and 3000 m buffer sizes.

Neighbourhood-level income. Though the RIS, disposable household income was adjusted for the number of household members in the year before baseline.[24] Standardized disposable household income was divided into tertiles based on the average income per individual in the RIS (the first tertile representing the lowest income group and the third tertile the highest income group).

Comorbidity. Comorbidity was based on discharge diagnoses of previous hospital admissions up to five years prior to the index date of admission or day clinic visit with dementia. Comorbidity was defined using a modified Charlson comorbidity index (CCI), a valid and reliable method to measure comorbidity in clinical research.[26] The updated version of the CCI is based on 12 weighted discharge diagnoses (i.e. chronic pulmonary disease, diabetes mellitus). The CCI ranges from 0 to 24 points, zero points representing no comorbidity. Total scores per individual were subdivided into three groups: 0, 1–2 and ≥ 3.

Population density and degree of urbanization. By means of geospatial data available from Statistics Netherlands (www.cbs.nl), population density of individuals' residential environment was determined by five categories expressing the number of addresses per km^2, ranging from ≤ 500 addresses to > 2500 addresses. Urban areas were classified as ≥ 1000 addresses per km^2 whereas rural areas were classified as < 1000 addresses per km^2.[27]

Ethics and privacy issues

All data linkages and analysis were performed in a secure environment of Statistics Netherlands and in agreement with the privacy legislation in The Netherlands.[28] Only anonymized records and data sets were involved. According to the regulations of the research complying with the Dutch law on Medical Research in Humans, approval by an ethics committee was not required for the present study.

Statistical analyses

Logistic regression analyses using IBM SPSS version 23.0 were conducted. The dependent variable was individual incidence of CVD (1 = yes, 0 = no) as well as incident CHD, stroke and heart failure (1 = yes, 0 = no), respectively. The independent variable was FFD within the three buffers. FFD was entered as a categorical variable with four levels for 500 m, 1000 m and 3000 m buffers. The cut-off values for FFD within each level was set in such way that a sufficient number of individuals remain in each level. The category with the lowest number of fast-food outlets served as reference group in all analyses. Analyses were stratified by the degree of urbanization. In addition to the base (unadjusted) model, subsequent models were adjusted for potential confounders. In the first model, outcomes were adjusted for age, sex, ethnicity, marital status, comorbidity and neighbourhood-level income. In the final complete model, additional adjustments were made for population density.[27] The threshold for significance was set at $p < 0.05$.

Results

Participant characteristics

Baseline characteristics are presented in Table 1. In total, 2,472,004 individuals free of CVD as of January 1st 2009 and living at the same address for ≥ 15 years were included in the cohort. In total, 2.5% had an incident CVD event in 2009. About 87% of the population was native Dutch, 70% of the included population was married, approximately half of the participants were men (46%) and the majority of the population lived in urban areas (61%).

Fast-food density and CVD incidence

The relations between FFD and the incidence of overall CVD, CHD, stroke and heart failure are presented in Table 2 for urban areas and in Table 3 for rural areas. In all analyses there was a large difference between the base model, and especially model 1, in which we adjusted for age, sex, ethnicity, marital status, comorbidity and neighbourhood-level income. Further adjustment for population density (model 2) had limited effect on the outcomes.

Urban areas

500 m residential buffer. Fully adjusted models indicated that the incidence of CVD and CHD was higher in areas with one or more fast-food outlets compared with areas with no fast-food outlets. The incidence

Table 1. Baseline characteristics of study population.

	Total $N = 2{,}472{,}004$	Rural areas (<1000 addresses/km^2) $n = 964{,}485$	Urban areas (≥1000 addresses/km^2) $n = 1{,}507{,}519$
Age, mean ± SD	59.3 ± 11.8	58.5 ± 11.7	59.8 ± 11.9
Men, *n* (%)	1,130,308 (46)	457,484 (47)	672,824 (45)
Comorbidity, *n* (%)	129,834 (5.3)	46,722 (4.8)	83,062 (5.5)
Native Dutch, *n* (%)	2,152,848 (87)	897,436 (93)	1,257,412 (83)
Married, *n* (%)	1,729,417 (70)	733,931 (76)	995,486 (66)
Incident CVD, *n* (%)	61,681 (2.5)	21,372 (2.2)	40,309 (2.7)
Incident CHD, *n* (%)	13,987 (0.6)	5021 (0.5)	8966 (0.6)
Incident stroke, *n* (%)	9058 (0.4)	3142 (0.3)	5916 (0.4)
Incident HF, *n* (%)	5077 (0.2)	1635 (0.2)	3443 (0.2)

CVD: cardiovascular disease; CHD: coronary heart disease; HF: heart failure.

Table 2. Relations between fast food density and incidence of CVD, CHD, stroke and HF in urban areas (odds ratios).

		%	Base model OR (95% CI)	Model 1 OR (95% CI)	Complete model OR (95% CI)
500 m					
CVD	D0 (0 FFR within 500 m)	47.9	1	1	1
	D1 (1 FFR within 500 m)	20.0	**1.15 (1.12–1.18)**	**1.05 (1.03–1.08)**	**1.05 (1.02–1.08)**
	D2 (2 FFR within 500 m)	11.0	**1.21 (1.17–1.25)**	**1.08 (1.05–1.12)**	**1.07 (1.04–1.12)**
	D3 (≥3 FFR within 500 m)	21.1	**1.16 (1.13–1.19)**	**1.06 (1.03–1.09)**	**1.04 (1.01–1.07)**
CHD	D0 (0 FFR within 500 m)	47.9	1	1	1
	D1 (1 FFR within 500 m)	20.0	**1.20 (1.13–1.27)**	**1.11 (1.05–1.18)**	**1.11 (1.05–1.17)**
	D2 (2 FFR within 500 m)	11.0	**1.25 (1.17–1.33)**	**1.13 (1.06–1.21)**	**1.13 (1.05–1.21)**
	D3 (≥3 FFR within 500 m)	21.1	**1.18 (1.12–1.24)**	**1.08 (1.03–1.16)**	**1.08 (1.02–1.14)**
Stroke	D0 (0 FFR within 500 m)	47.9	1	1	1
	D1 (1 FFR within 500 m)	20.0	**1.16 (1.09–1.24)**	1.03 (0.96–1.10)	1.03 (0.96–1.10)
	D2 (2 FFR within 500 m)	11.0	**1.23 (1.13–1.34)**	1.05 (0.96–1.15)	1.06 (0.97–1.15)
	D3 (≥3 FFR within 500 m)	21.1	**1.22 (1.14–1.30)**	1.07 (1.00–1.15)	**1.09 (1.02–1.17)**
HF	D0 (0 FFR within 500 m)	47.9	1	1	1
	D1 (1 FFR within 500 m)	20.0	**1.23 (1.12–1.34)**	1.01 (0.92–1.11)	1.01 (0.92–1.10)
	D2 (2 FFR within 500 m)	11.0	**1.49 (1.34–1.65)**	**1.16 (1.04–1.29)**	**1.15 (1.03–1.27)**
	D3 (≥3 FFR within 500 m)	21.1	**1.42 (1.30–1.54)**	**1.11 (1.02–1.21)**	1.09 (0.99–1.19)
1000 m					
CVD	D0 (0 FFR within 1000 m)	15.0	1	1	1
	D1 (1 FFR within 1000 m)	16.9	1.03 (0.99–1.07)	1.01 (0.98–1.05)	1.01 (0.97–1.05)
	D2 (2–4 FFR within 1000 m)	30.2	**1.16 (1.12–1.20)**	**1.05 (1.01–1.08)**	**1.04 (1.00–1.07)**
	D3 (≥5 FFR within 1000 m)	37.8	**1.26 (1.22–1.30)**	**1.08 (1.04–1.11)**	**1.05 (1.02–1.09)**
CHD	D0 (0 FFR within 1000 m)	15.0	1	1	1
	D1 (1 FFR within 1000 m)	16.9	1.08 (0.99–1.16)	1.07 (0.98–1.15)	1.06 (0.98–1.15)
	D2 (2–4 FFR within 1000 m)	30.2	**1.20 (1.12–1.29)**	**1.10 (1.02–1.18)**	**1.09 (1.02–1.17)**
	D3 (≥5 FFR within 1000 m)	37.8	**1.34 (1.25–1.43)**	**1.18 (1.10–1.26)**	**1.17 (1.09–1.25)**
Stroke	D0 (0 FFR within 1000 m)	15.0	1	1	1
	D1 (1 FFR within 1000 m)	16.9	0.91 (0.82–1.00)	**0.89 (0.81–0.98)**	**0.88 (0.81–0.98)**

(continued)

Table 2. Continued.

		%	Base model OR (95% CI)	Model 1 OR (95% CI)	Complete model OR (95% CI)
	D2 (2–4 FFR within 1000 m)	30.2	**1.10 (1.01–1.19)**	0.95 (0.87–1.03)	0.94 (0.87–1.03)
	D3 (≥5 FFR within 1000 m)	37.8	**1.19 (1.10–1.28)**	0.95 (0.87–1.03)	0.96 (0.88–1.04)
HF	D0 (0 FFR within 1000 m)	15.0	1	1	1
	D1 (1 FFR within 1000 m)	16.9	1.03 (0.89–1.18)	1.01 (0.88–1.16)	1.00 (0.87–1.15)
	D2 (2–4 FFR within 1000 m)	30.2	**1.32 (1.18–1.49)**	1.06 (0.94–1.19)	1.05 (0.93–1.18)
	D3 (≥5 FFR within 1000 m)	37.8	**1.74 (1.56–1.95)**	**1.21 (1.08–1.36)**	**1.18 (1.05–1.33)**
3000 m					
CVD	D0 (0–3 FFR within 3000 m)	5.4	1	1	1
	D1 (4–10 FFR within 3000 m)	14.1	1.02 (1.00–1.04)	0.99 (0.97–1.02)	0.97 (0.95–1.02)
	D2 (11–27 FFR within 3000 m)	36.0	1.09 (1.07–1.11)	1.04 (1.01–1.06)	0.96 (0.93–1.01)
	D3 (≥28 FFR within 3000 m)	44.5	1.28 (1.25–1.31)	1.12 (1.10–1.15)	0.99 (0.96–1.04)
CHD	D0 (0–3 FFR within 3000 m)	5.4	1	1	1
	D1 (4–10 FFR within 3000 m)	14.1	0.96 (0.86–1.08)	0.98 (0.88–1.10)	0.99 (0.88–1.11)
	D2 (11–27 FFR within 3000 m)	36.0	**1.11 (1.01–1.23)**	1.07 (0.97–1.19)	1.07 (0.97–1.19)
	D3 (≥28 FFR within 3000 m)	44.5	**1.30 (1.17–1.43)**	**1.17 (1.06–1.29)**	**1.17 (1.05–1.30)**
Stroke	D0 (0–3 FFR within 3000 m)	5.4	1	1	1
	D1 (4–10 FFR within 3000 m)	14.1	**0.86 (0.75–0.98)**	0.89 (0.78–1.02)	0.89 (0.78–1.02)
	D2 (11–27 FFR within 3000 m)	36.0	1.01 (0.90–1.14)	0.96 (0.85–1.08)	0.95 (0.85–1.08)
	D3 (≥28 FFR within 3000 m)	44.5	1.12 (1.00–1.26)	0.94 (0.83–1.06)	0.94 (0.83–1.06)
HF	D0 (0–3 FFR within 3000 m)	5.4	1	1	1
	D1 (4–10 FFR within 3000 m)	14.1	**0.80 (0.66–0.96)**	0.86 (0.71–1.03)	0.85 (0.70–1.02)
	D2 (11–27 FFR within 3000 m)	36.0	0.99 (0.84–1.17)	0.93 (0.79–1.09)	0.91 (0.77–1.07)
	D3 (≥28 FFR within 3000 m)	44.5	**1.31 (1.12–1.53)**	0.98 (0.83–1.14)	0.92 (0.78–1.08)

Bold odds ratios are statistically significantly different against the reference category ($p < 0.05$). Analyses were conducted for areas with ≥ 1000 addresses per km^2. Base model: unadjusted model; Model 1: adjusted for age, sex, ethnicity, marital status, comorbidity and neighbourhood-level income; Model 2: adjusted for covariates of Model 1 plus additional adjustments for population density.
CVD: cardiovascular disease; CHD: coronary heart disease; HF: heart failure; OR: odds ratio; CI: confidence interval; FFR: fast food restaurant (outlet).

of stroke was only higher in areas with three or more fast-food outlets, whereas the incidence of heart failure was only higher in areas with two or more fast-food outlets

1000 m residential buffer. Fully adjusted models indicated that an elevated FFD was associated with a raised incidence of CVD and CHD. The analyses showed marginally higher incidence of CVD in areas with 2–4 fast-food outlets and ≥5 fast-food outlets. Statistically significant relations were found for areas with 2–4 fast-food outlets and areas with ≥5 outlets and CHD. These figures may insinuate a dose–response between FFD within the 1000 m residential buffer and the incident of individual-level CVD and CHD. For the incidence of heart failure, a statistically significant relation was found with ≥5 fast-food outlets. In contrast, an opposed direction was found for stroke. In areas with one fast-food outlet the incidence of stroke was lower than in areas without fast-food outlets within the residential buffer.

3000 m residential buffer. The relations were less pronounced when FFD was calculated for a wider area around the home address. Only a statistically significant higher CHD incidence was found in areas with the presence of 28 or more fast-food outlets.

Rural areas

500 m residential buffer. In comparison with urban areas, fully adjusted models indicated that the incidence of CHD was higher in areas with one or two fast-food outlets. The incidence of individual-level CHD dropped in areas with three or more fast-food outlets and became insignificant. Except that the incident of individual-level heart failure was higher in areas with three or more fast-food outlets, no statistically significant relations were found.

1000 m residential buffer. Fully adjusted models indicated that the incidence of CHD was only higher in areas with 2–4 fast-food outlets

Table 3. Relations between fast-food density and incidence of CVD, CHD, stroke and HF in rural areas.

		%	Base model OR (95% CI)	Model 1 OR (95% CI)	Complete model OR (95% CI)
500 m					
CVD	D0 (0 FFR within 500 m)	68.5	1	1	1
	D1 (1 FFR within 500 m)	18.2	**1.08 (1.04–1.12)**	1.02 (0.98–1.06)	1.02 (0.98–1.05)
	D2 (2 FFR within 500 m)	7.4	**1.12 (1.10–1.22)**	1.02 (0.97–1.08)	1.02 (0.97–1.08)
	D3 (≥3 FFR within 500 m)	5.9	**1.22 (1.16–1.29)**	1.01 (0.96–1.07)	1.02 (0.96–1.07)
CHD	D0 (0 FFR within 500 m)	68.5	1	1	1
	D1 (1 FFR within 500 m)	18.2	**1.15 (1.07–1.23)**	**1.09 (1.01–1.17)**	**1.09 (1.01–1.17)**
	D2 (2 FFR within 500 m)	7.4	**1.34 (1.22–1.48)**	**1.20 (1.09–1.32)**	**1.20 (1.09–1.32)**
	D3 (≥3 FFR within 500 m)	5.9	**1.21 (1.08–1.35)**	1.03 (0.92–1.16)	1.03 (0.92–1.15)
Stroke	D0 (0 FFR within 500 m)	68.5	1	1	1
	D1 (1 FFR within 500 m)	18.2	1.10 (1.00–1.20)	1.00 (0.91–1.10)	1.00 (0.91–1.10)
	D2 (2 FFR within 500 m)	7.4	1.32 (1.17–1.49)	1.10 (0.97–1.24)	1.09 (0.97–1.24)
	D3 (≥3 FFR within 500 m)	5.9	1.48 (1.30–1.69)	1.12 (0.98–1.28)	1.12 (0.98–1.28)
HF	D0 (0 FFR within 500 m)	68.5	1	1	1
	D1 (1 FFR within 500 m)	18.2	**1.21 (1.03–1.43)**	1.06 (0.90–1.25)	1.06 (0.90–1.25)
	D2 (2 FFR within 500 m)	7.4	**1.15 (0.90–1.46)**	0.87 (0.68–1.11)	0.87 (0.68–1.11)
	D3 (≥3 FFR within 500 m)	5.9	**1.97 (1.59–2.44)**	**1.27 (1.02–1.57)**	**1.25 (1.01–1.56)**
1000 m					
CVD	D0 (0 FFR within 1000 m)	37.3	1	1	1
	D1 (1 FFR within 1000 m)	21.4	0.97 (0.94–1.01)	0.98 (0.94–1.02)	0.98 (0.94–1.02)
	D2 (2–4 FFR within 1000 m)	31.2	**1.06 (1.03–1.10)**	1.00 (0.97–1.04)	1.01 (0.97–1.04)
	D3 (≥5 FFR within 1000 m)	10.0	**1.15 (1.10–1.21)**	0.98 (0.93–1.02)	0.98 (0.93–1.03)
CHD	D0 (0 FFR within 1000 m)	15.0	1	1	1
	D1 (1 FFR within 1000 m)	16.9	1.01 (0.93–1.09)	1.02 (0.94–1.10)	1.02 (0.94–1.10)
	D2 (2–4 FFR within 1000 m)	30.2	**1.13 (1.06–1.21)**	1.07 (1.00–1.15)	**1.07 (1.00–1.15)**
	D3 (≥5 FFR within 1000 m)	37.8	**1.25 (1.14–1.37)**	1.08 (0.98–1.19)	1.07 (0.98–1.18)
Stroke	D0 (0 FFR within 1000 m)	37.3	1	1	1
	D1 (1 FFR within 1000 m)	21.4	0.99 (0.89–1.09)	0.98 (0.89–1.09)	0.98 (0.99–1.08)
	D2 (2–4 FFR within 1000 m)	31.2	**1.19 (1.10–1.30)**	1.08 (0.99–1.18)	1.08 (0.99–1.17)
	D3 (≥5 FFR within 1000 m)	10.0	**1.37 (1.22–1.54)**	1.07 (0.95–1.20)	1.06 (0.94–1.19)
HF	D0 (0 FFR within 1000 m)	37.3	1	1	1
	D1 (1 FFR within 1000 m)	21.4	**1.10 (1.02–1.19)**	0.98 (0.89–1.09)	0.98 (0.99–1.08)
	D2 (2–4 FFR within 1000 m)	31.2	**1.21 (1.07–1.35)**	1.08 (0.99–1.18)	1.08 (0.99–1.17)
	D3 (≥5 FFR within 1000 m)	10.0	1.34 (0.98–1.82)	1.07 (0.95–1.20)	1.06 (0.94–1.19)
3000 m					
CVD	D0 (0–3 FFR within 3000 m)	45.5	1	1	1
	D1 (4–10 FFR within 3000 m)	43.1	1.00 (0.97 -1.02)	0.98 (0.95–1.00)	0.97 (0.94–1.01)
	D2 (11–27 FFR within 3000 m)	10.3	0.97 (0.93–1.02)	0.93 (0.88–0.97)	0.93 (0.88–0.97)
	D3 (≥28 FFR within 3000 m)	1.1	**1.26 (1.11–1.41)**	0.98 (0.87–1.10)	0.98 (0.86–1.10)
CHD	D0 (0–3 FFR within 3000 m)	45.5	1	1	1
	D1 (4–10 FFR within 3000 m)	43.1	1.06 (0.998–1.12)	1.04 (0.98–1.10)	1.03 (0.97–1.10)
	D2 (11–27 FFR within 3000 m)	10.3	1.02 (0.93–1.13)	0.98 (0.89–1.07)	0.96 (0.87–1.07)
	D3 (≥28 FFR within 3000 m)	1.1	1.19 (0.92–1.53)	1.01 (0.78–1.31)	1.00 (0.78–1.30)
Stroke	D0 (0–3 FFR within 3000 m)	45.5	1	1	1
	D1 (4–10 FFR within 3000 m)	43.1	**1.10 (1.01–1.19)**	1.05 (0.97–1.14)	1.05 (0.97–1.15)
	D2 (11–27 FFR within 3000 m)	10.3	**1.16 (1.02–1.32)**	1.08 (0.95–1.22)	1.08 (0.94–1.24)

(continued)

Table 3. Continued.

		%	Base model OR (95% CI)	Model 1 OR (95% CI)	Complete model OR (95% CI)
	D3 (≥28 FFR within 3000 m)	1.1	1.24 (0.88–1.75)	0.89 (0.63–1.25)	0.89 (0.63–1.25)
HF	D0 (0–3 FFR within 3000 m)	45.5	1	1	1
	D1 (4–10 FFR within 3000 m)	43.1	**1.10 (1.02–1.19)**	1.06 (0.98–1.14)	1.05 (0.97–1.14)
	D2 (11–27 FFR within 3000 m)	10.3	**1.21 (1.07–1.35)**	1.12 (0.99–1.26)	1.11 (0.98–1.26)
	D3 (≥28 FFR within 3000 m)	1.1	134 (0.98–1.82)	0.91 (0.67–1.24)	0.91 (0.66–1.67)

Bold odds ratios are statistically significantly different against the reference category ($p < 0.05$). Analyses were conducted for areas with < 1000 addresses per km^2. Base model: unadjusted model; Model 1: adjusted for age, sex, ethnicity, marital status, comorbidity and neighbourhood-level income; Model 2: adjusted for covariates of Model 1 plus additional adjustments for population density.
CVD: cardiovascular disease; CHD: coronary heart disease; HF: heart failure; OR: odds ratio; CI: confidence interval; FFR: fast food restaurant (outlet).

3000 m residential buffer. Similar to urban areas, no statistically significant relations were found between the presence of fast-food outlets within the rural 3000 m residential buffers and the incidence of CVD or CVD subtypes in the fully adjusted models.

Discussion

This study showed that the incidence of CVD and CHD was higher among adults living in urban areas with elevated numbers of fast-food outlets within 1000 m of the residential address compared with individuals with no fast-food outlets in this area. In the 500 m buffer, the incidence of CVD and CHD was higher in areas with one or more fast-food outlets compared with areas with no fast-food outlets. Relations between FFD and CVD and subtypes in the 3000 m buffer were largely absent and less consistent. Relations were less pronounced in rural areas or for stroke and heart failure incidence. Effect sizes of the relationships were small to moderate in magnitude, but reflect the nation-wide incidence of a population of more than two million adults. Our findings suggest urban FFD is potentially an important piece of the puzzle when understanding risk factors of CVD.

Relations between the fast-food environment and overall CVD were pronounced in urban areas, but not in rural areas. Speculating on the source of this difference, it is well understood that urbanization is associated with shifts in dietary patterns, expressed by an increase in the consumption of ultra-processed foods, saturated fat and added sugar, and a decrease in fibre intake.[29] Likewise, food intake among urban residents is shaped by the culture of commercial influences (e.g. that of fast-food outlets) or social influences (e.g. purchasing and eating behaviors in public space).[30] However, the difference may also be due to the fact that we were unable to correct for traditional cardiovascular risk behaviors such as smoking or being physical inactive, which are more pronounced among urban than among rural residents.[31]

With respect to CVD subtypes, the outcome of our study, which indicates that FFD is related to the incidence of CHD within <1000 m buffers, is in contrast with previous findings. Although in a prior study similar directions were found of death per 10% increase in FFD (relative risks = 1.39 (95% confidence interval (CI) = 1.19–1.63)), this was not different in magnitude for non-cardiovascular mortality (relative risk = 1.36 (95% CI = 1.18–1.57)). Also the availability of fruit and vegetables was not associated with cardio-vascular mortality. Based on these findings, the authors question the role of the food environment in cardiovascular outcomes.[18] Unlike our study – where we corrected for neighbourhood-level income – the authors of another study corrected for individual-level income, which resulted in the mitigation of any statistically significant relationship between FFD and CHD.[19] Yet, although not the most apparent outcome of this study, the relation between the presence of ≥3 fast-food outlets within the 500 m buffer in urban areas and the incidence of stroke is comparable to the previous studies[15,16] where elevated fast-food availability was associated with stroke risk. We found also an opposite relationship where the incidence of stroke was marginally but significantly lower in areas (1000 m) with one fast-food outlet than in urban areas without fast-food outlets. However, these studies used an area-level approach to determine fast-food exposure and are therefore less comparable to our study.

The present study had a number of strengths, such as the inclusion of a large nation-wide study sample, the use of individual-level health outcomes, the longitudinal perspective by using CVD *incidence* rather than *prevalence* as outcome measure, including CVD subtypes, the use of individual-level fast-food exposure of different sizes of street network buffers, the stratification for urban and rural areas and the inclusion of individuals living at the same address for at least 15 years that were followed for one year. However, some limitations should be considered. The most important limitation is that we could not adjust for

all individual-level risk factors such as dietary intake, alcohol consumption or smoking behaviour. Yet, we were able to correct for neighbourhood income level, which is likely an important link between individual dietary and smoking behaviour and neighbourhood FFD. Moreover, we corrected for comorbidity including the diagnosis of diseases associated with dietary and smoking behaviour (e.g. diabetes type 2, chronic pulmonary disease, malignancy). Second, in the construction of the cohort we linked the different datasets (e.g. population register, HDR) by means of the personal identifier base including a unique combination of individual characteristics. Although this is a frequently used technique, we lack exact linkage, which may have resulted into non-differential misclassification of the exposure or outcome. Although this risk is small – approximately 85% of the Dutch population has a unique combination of these characteristics – this should be taken into account when interpreting the results. Finally, we calculated FFD only for one time stamp (2009) and we acknowledge that this exposure may have changed gradually over time. Nevertheless, we expect that FFD changes over time were relatively similar for all participants in the cohort that lived at the same address for a minimum of 15 years.

In the future, studies should explore the relation between FFD and CVD incidence, accounting for a wider range of individual confounders than was achieved in the present study (e.g. smoking behaviour). Additionally, future studies could incorporate area-level proxies for smoking behaviour if available (e.g. area-level chronic obstructive pulmonary disease) or account for individual control endpoints that are unlikely to relate to FFD (e.g. lung cancer). In addition, the relationship between FFD and the hypothesized underlying mechanism of fast-food exposure and CVD, fast-food consumption or overall dietary intake should be investigated.

Public policy makers should be aware of the likely impact of urban FFD on health, especially since the number of fast-food outlets is still increasing. The 2016 European Guidelines on cardiovascular disease of the Sixth Joint Task Force of the European Society of Cardiology recommend population-based approaches to diet in managing CVD prevention. These guidelines also include the recommendation to consider the regulation and density of fast-food outlets in community settings.[32] Already efforts banning fast-food facilities in residential or school environments have been proposed to improve public health.[33]

Conclusion

The study indicated that elevated FFD in the urban residential environment (≤1000 m) was associated with an increased incidence of CVD and CHD. To better understand how FFD is associated with CVD, future studies should account for a wider range of lifestyle and environmental confounders than was achieved in the present study.

Author contribution

MP and IV drafted the manuscript and contributed to acquisition, analysis and interpretation. MS and BB contributed to acquisition and analyses. OS contributed to the analyses. GH, AN and MB contributed to the interpretation. DK contributed to the acquisition and interpretation. MH and DG contributed to the analyses and interpretation. All authors critically revised the paper, provided final approval and agreed to be accountable for all aspects of work ensuring integrity and accuracy.

Acknowledgements

Presentations: presentation MP: ISBNPA 2017. Poster IV: ESC EuroPrevent 2017. The authors would like to thank Statistics Netherlands (www.cbs.nl/en-GB) and Locatus BV (www.locatus.com) for the use of the databases. Also they would like to acknowledge the statistical work that was accomplished by Dr Carla Koopman.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: The contribution of the first author (MP) is supported by the Innovational Research Incentives Scheme (#451-16-029), financed by the Netherlands Organization for Scientific Research (NWO). Contribution of the final author (IV) is supported by the Dutch Heart Foundation. The study was funded and supported by the Global and Geo Health Data Centre of Utrecht University (https://globalgeohealthdatacenter.com/).

References

1. World Health Organization. Cardiovascular disease, http://www.who.int/cardiovascular_diseases/en/ (2017, accessed April 6 2017).
2. Popkin BM. *The world is fat: The fads, trends, policies, and products that are fattening the human race*. New York, NY: Avery Publishing Group, 2009.
3. Nestle M. *Food politics: How the food industry influences nutrition and health*. Berkeley: University of California Press, 2007.
4. Adriouch S, Julia C, Kesse-Guyot E, et al. Prospective association between a dietary quality index based on a nutrient profiling system and cardiovascular disease risk. *Eur J Prev Cardiol* 2016; 23: 1669–1667.

5. Mente A, de Koning L, Shannon HS, et al. A systematic review of the evidence supporting a causal link between dietary factors and coronary heart disease. *Arch Intern Med* 2009; 169: 659–669.
6. Fung TT, Stampfer MJ, Manson JE, et al. Prospective study of major dietary patterns and stroke risk in women. *Stroke* 2004; 35: 2014–2019.
7. Sallis JF, Owen N and Fisher EB. Ecological models of health behavior. In: Glanz K, Rimer BK and Viswanath K (eds) *Health behavior and health education: Theory, research, and practice*. San Francisco: Jossey-Bass, 2015, pp.43–64.
8. Swinburn B, Egger G and Raza F. Dissecting obesogenic environments: The development and application of a framework for identifying and prioritizing environmental interventions for obesity. *Prev Med* 1999; 29: 563–570.
9. Jaworowska A, Blackham T, Davies IG, et al. Nutritional challenges and health implications of takeaway and fast food. *Nutr Rev* 2013; 71: 310–318.
10. Statista. Number of McDonald's restaurants worldwide from 2005 to 2016, https://www.statista.com/statistics/219454/mcdonalds-restaurants-worldwide/ (2016, accessed 6 April 2017).
11. Maguire ER, Burgoine T and Monsivais P. Area deprivation and the food environment over time: A repeated cross-sectional study on takeaway outlet density and supermarket presence in Norfolk, UK, 1990–2008. *Health Place* 2015; 33: 142–147.
12. Netherlands Statistics. Branches of companies; industry, region, http://statline.cbs.nl/Statweb/publication/?DM=SLNL&PA=81578ned&D1=0&D2=908-909&D3=0,5-16&D4=a&HDR=T,G3,G2&STB=G1&VW=T (2017, accessed 6 April 2017).
13. Mohammed Kelli HM, Ahmed H, Hammadah M, et al. The association of living in food deserts with cardiovascular risk factors and subclinical vascular disease. *J Am Coll Cardiol* 2016; 67: 1883–1883.
14. Alter DA and Eny K. The relationship between the supply of fast-food chains and cardiovascular outcomes. *Can J Public Health* 2005; 96: 173–177.
15. Morgenstern LB, Escobar JD, Sánchez BN, et al. Fast food and neighborhood stroke risk. *Ann Neurol* 2009; 66: 165–170.
16. Hamano T, Kawakami N, Li X, et al. Neighbourhood environment and stroke: A follow-up study in Sweden. *PloS One* 2013; 8: e56680.
17. Chum A and O'Campo P. Cross-sectional associations between residential environmental exposures and cardiovascular diseases. *BMC Public Health* 2015; 15: 1.
18. Daniel M, Paquet C, Auger N, et al. Association of fast-food restaurant and fruit and vegetable store densities with cardiovascular mortality in a metropolitan population. *Eur J Cardiovasc Med* 2010; 25: 711–719.
19. Kawakami N, Li X and Sundquist K. Health-promoting and health-damaging neighbourhood resources and coronary heart disease: A follow-up study of 2 165 000 people. *J Epidemiol Community Health* 2011; 65: 866–872.
20. Oliver LN, Schuurman N and Hall AW. Comparing circular and network buffers to examine the influence of land use on walking for leisure and errands. *Int J Health Geogr* 2007; 6: 1.
21. *The international statistical classification of diseases, injuries and causes of death. Ninth revision. Clinical modification*. Washinton DC: World Health Organization, 1979.
22. De Bruin A, Kardaun J, Gast F, et al. Record linkage of hospital discharge register with population register: Experiences at Statistics Netherlands. *Stat J UN Econ Comm Eur* 2004; 21: 23–32.
23. Paas GR and Veenhuizen KC (eds) *Research on the validity of the LMR* [in Dutch]. Utrecht: Prismant, 2002.
24. Ament P and Kessels W. *Report Regionaal Inkomens Onderzoek: Iitgebreide onderzoeksbeschrijving*. Voorburg: Centraal Bureau voor de Statistiek (CBS), 2008.
25. Smith D, Cummins S, Clark C, et al. Does the local food environment around schools affect diet? Longitudinal association in adolescents attending secondary schools in East London. *BMC Public Health* 2013; 13: 70.
26. Quan H, Li B, Couris CM, et al. Updating and validating the Charlson comorbidity index and score for risk adjustment in hospital discharge abstracts using data from 6 countries. *Am J Epidemiol* 2011; 173: 676–682.
27. Statistics Netherlands. Average income; private households to various characteristics, http://statline.cbs.nl/Statweb/publication/?DM=SLNL&PA=70843ned&D1=0,3-6&D2=0-1,8-9,15,18&D3=63-67&D4=l&VW=T (2017, accessed 6 April 2017).
28. Reitsma JB, Kardaun JW, Gevers E, et al. Possibilities for anonymous follow-up studies of patients in Dutch national medical registrations using the Municipal Population Register: A pilot study. *Ned Tijdschr Geneeskd* 2003; 147: 2286–2290.
29. Popkin BM and Gordon-Larsen P. The nutrition transition: worldwide obesity dynamics and their determinants. *Int J Obes* 2004; 28: S2–S9.
30. Zukin S. Urban lifestyles: Diversity and standardisation in spaces of consumption. *Urban Stud* 1998; 35: 825–839.
31. Netherlands Statistics. Lifestyle, preventive research; personal characteristics; 2010–2013, http://statline.cbs.nl/Statweb/publication/?DM=SLNL&PA=81177ned&D1=0-2,10,39-43,63&D2=54-58&D3=0&D4=l&HDR=T&STB=G1,G2,G3&VW=T,10,39-43,63&D2=54-58&D3=0&D4=l&HDR=T&STB=G1,G2,G3&VW=T (2017, accessed 6 April 2017).
32. Piepoli MF, Hoes AW, Agewall S, et al. 2016 European Guidelines on cardiovascular disease prevention in clinical practice: The Sixth Joint Task Force of the European Society of Cardiology and Other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of 10 societies and by invited experts). Developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation. *Eur J Prev Cardiol* 2016; 23: NP1–NP96.
33. Coyne E. Banning on fast foods near schools approved, *The Times*. https://www.thetimes.co.uk/article/ban-on-fast-food-near-schools-approved-prm823vpp (2016, accessed 11 August 2016).