

# Geographic Life Environments and Coronary Heart Disease: A Literature Review, Theoretical Contributions, Methodological Updates, and a Research Agenda

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## Key Words

contextual analysis, eco-epidemiology, neighborhood, social-cognitive theories, social interactions

## Abstract

A growing literature investigates associations between neighborhood social environments and coronary heart disease (CHD). After reviewing the literature, we present a theoretical model of the mechanisms through which geographic life environments may influence CHD, focusing particularly on the social-interactive environment. We suggest that, in addition to the common notions of social cohesion or fragmentation and social disorder, eco-epidemiologists should consider neighborhood identities and stigmatization processes. We posit that neighborhood social interactions affect the wide set of affective, cognitive, and relational experiences individuals have in their neighborhoods, which in turn influence the psycho-cognitive antecedents of behavior and in the end shape health behavior. Finally, we discuss key methodological challenges relevant to the advent of a new generation of neighborhood studies, including the operational definition of neighborhoods, non-residential environments, ecometric measurement, model specification strategies, mediational models, selection processes and notions of empirical/structural confounding, and the relevance of observational versus interventional studies.

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**CHD:** coronary heart disease

**CVD:** cardiovascular disease

**SEP:** socioeconomic position

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## INTRODUCTION

A growing body of literature investigates whether social characteristics of residential environments are associated with the risk of coronary heart disease (CHD) and contribute to socioeconomic disparities in CHD, over and above the effects of individuals' sociodemographic factors (22, 33, 107).

After more than a decade of research on this issue, it is useful to summarize the emerging results, identify obstacles, and propose new lines of investigation. Do these studies converge to consistent results that confirm the existence of true neighborhood effects? Do we have sufficient knowledge on the underlying mechanisms at play to propose innovative interventions? If not, which strategies should we employ to penetrate the black box of neighborhood effects on CHD?

This article offers a narrative review of the literature on neighborhood social environments and CHD. Second, it proposes a theoretical contribution on the pathways through which geographic life environments may influence CHD, building on sociology and social and environmental psychology to supplement previous models (27, 30, 97). Third, it discusses a number of methodological challenges arising in the investigation of neighborhood effects on health.

## A NARRATIVE REVIEW ON NEIGHBORHOOD AND CHD

### Search Strategy

Inclusion and exclusion criteria were specified as follows: First, we focused on CHD outcomes defined at the individual level (prevalence, incidence, mortality, and survival after CHD—either overall mortality or CHD recurrence after a CHD event). However, we also included studies with overall cardiovascular disease (CVD) outcomes defined at the individual level (including CHD as a subcomponent) (11, 28, 32, 40, 52, 60, 61, 68, 109, 110). Studies on stroke were excluded. Second, we were interested mainly in the associations between area

social variables or urban environment variables and CHD-CVD. Studies focused on air pollution effects that did not consider any area social factor were excluded. Third, only studies estimating area effects adjusted for individual socioeconomic position (SEP) were considered. The others (96), even if modeling CHD-CVD at the individual level, were discarded.

We searched Pubmed for English-language articles published between January 1985 and September 2008 using relevant title expressions (listed in the footnote to **Table 1**), reviewed the reference list of the selected articles, and screened the papers citing the selected articles.

As shown in **Table 1** and in **Supplemental Appendix 1** online, 40 studies were retained (follow the Supplemental Material link from the Annual Reviews home page at <http://www.annualreviews.org>).

## Results

The literature review led to the following considerations.

**Location and population.** In this literature, study populations come from only a few countries: 35 of the 40 studies were from the United States, Sweden, or the United Kingdom. Most databases were drawn from the general population. Few studies relied on clinical samples (50, 81, 99).

**Designs and CHD-CVD outcomes.** Four studies considered subclinical vascular diseases (13, 77, 81, 89), 8 considered CHD-CVD prevalence (5, 29, 36, 52, 59, 61, 110, 113), 12 considered CHD-CVD incidence (21, 33, 56, 74, 82, 102–107, 111), 16 considered CHD-CVD mortality (4, 7, 8, 17, 31, 35, 38–47), and 4 considered post-CHD survival (21, 50, 99, 111). Whereas certain studies specifically validated incident CHD cases (e.g., 33, 42), many others identified them from national hospital or mortality registers (e.g., 21, 67, 68, 103, 105). Regarding analytic designs, considering first-ever incident cases through prospective follow-up decreases risk of health-related selective migration biases, which is a strength

of incidence and mortality studies (102), contrary to cross-sectional studies in which reverse causation may contribute to the observed associations. However, studies that may be best able to overcome these biases are those that focused on preclinical measures of atherosclerosis (13, 77, 81, 89). Very few studies investigated neighborhood effects on post-CHD survival (21, 50, 111). Moreover, few studies (21, 111) compared neighborhood effects on distinct outcomes, e.g., incidence, mortality, and survival, and none compared effects on a subclinical and clinical outcome.

**Area delimitations.** Most studies relied on census, administrative, electoral, or postal mail delimitations to define residential areas. Twenty-eight of the 40 studies reported explicit information on area population size. In most studies, neighborhoods had an average or median population size between 1000 and 5000, whereas 4 studies considered more local neighborhoods (17, 19, 20, 22) and 5 studies considered areas with more than 10,000 inhabitants (56, 67, 68, 82, 89).

**Assessment of geographic variations.** Nineteen studies reported measures of geographic variations of CHD, as previously recommended (70). The intraclass correlation (20, 40, 56, 59, 61, 89, 103, 105, 110), median odds ratio (20, 21, 61), and interquartile odds ratio (19–22) were used to quantify between area variations estimated from multilevel models. Few studies reported between-neighborhood variance from empty or minimally adjusted models (20–22, 61, 82, 104, 106), and they did so with different indicators and for different area definitions and outcomes, precluding any meaningful comparison.

**Individual-level adjustment.** In the 40 studies estimating area effects on CHD-CVD, models were adjusted for only 1 individual SEP variable in 11 studies, 2 variables in 8 studies, 3 in 18 studies, and 4+ in 3 studies (13, 59, 68) (see footnote to **Table 1** for a list of the individual SEP factors identified in these studies).

Thus, individual-level adjustment was often insufficient, not permitting the models to capture complexities of the socioeconomic trajectories over the life course. Only two studies also controlled for childhood SEP (13, 59). In Lawlor (59), the area SEP-CHD association adjusted for individual adult SEP was further reduced (but persisted) when controlling for childhood SEP. Only one study (32) used propensity score matching to mitigate the nonexchangeability of individuals (i.e., the nonoverlap of their propensity to reside in an exposed neighborhood) between contrasted neighborhood exposure categories.

**Neighborhood socioeconomic position.** Thirty-six studies considered area SEP as a contextual factor, and 25 took into account no other contextual exposure. Twenty-three studies relied on a composite SEP index. Only one study measured neighborhood SEP over the life course, but it made no attempt to separate childhood and adult area effects (13). Thirty-two studies documented, after individual-level adjustment, an increased CHD-CVD risk in deprived areas in at least one population subgroup (e.g., gender, ethnicity, etc.). Publication biases may of course contribute to this high percentage of positive findings. Only 2 of the 4 studies (81, 89) that focused on subclinical CVD documented a significant area SEP effect. Certain studies reported statistically significant cross-level interactions; data showed a higher area SEP effect on CHD-CVD mortality among the oldest (20, 109) and among women (11, 29, 105, 111) and a synergistic effect between low individual and low neighborhood income among women (102). One study also documented a stronger area SEP effect in urban than in periurban or rural territories (20).

**Other contextual factors.** Fifteen studies considered area factors other than SEP: urbanicity degree (19, 42, 111), urban sprawl (36), income inequality (40, 102), and social-environmental variables such as residential stability (21), dwelling ownership (40), presence

**Table 1 Literature review<sup>a</sup> of studies investigating associations between area social factors and CHD or CVD, adjusted for individual SEP<sup>b</sup> (full details reported in Supplemental Appendix 1 online)**

First author, year	Location and date <sup>c</sup>	Study design and outcome(s)	Geographic scale	Contextual variable(s)
Garcia-Palmeri 1988 (42)	Northeast region of Puerto Rico; 1965–1968	Cohort; CHD mortality	Enumeration districts	Urban-rural status of enumeration districts
Jones 1995 (52)	United Kingdom; 1984–1985	Cross-sectional; self-reported symptoms of heart disease, high blood pressure, or stroke	Wards, parliamentary constituencies	Ward % living in high-rise or urban environment, ward deprivation, constituency average household income
Woodward 1996 (113)	Scotland; 1984–1986	Cross-sectional; prevalent CHD (diagnosed and self-reported)	Postcode sectors	Carstairs and Morris deprivation score
Diez-Roux 1997 (29)	4 U.S. communities; 1987–1989	Cross-sectional; CHD prevalence (diagnosed and self-reported)	Census block groups	Neighborhood education, income, house value, and occupation
Davey Smith 1998 (28)	Renfrew and Paisley, Scotland; 1972–1976	Cohort; CVD mortality	Postcode sectors	Carstairs and Morris deprivation index
Leclerc 1998 (60)	United States; 1986–1990	Cohort; heart disease mortality	Census tracts	Rate of female-headed families, % of blacks, median family income, public assistance, deep poverty, unemployment
Waizman 1998 (109)	United States; 1971–1974	Cohort; CVD mortality	Contiguous census tracts	Federally designated poverty areas
Diez-Roux 2001 (33)	4 U.S. communities; 1987–1989	Cohort; CHD incidence	Census block groups	Neighborhood socioeconomic summary score
Stjärne 2002 (56)	Stockholm county, Sweden; 1992–1994	Case-control study; first events of myocardial infarction	Metropolitan parishes	Class structure, social exclusion, poverty
Ewing 2003 (36)	United States; 1998, 1999, and 2000	Cross-sectional; self-reported CHD prevalence	Counties	Sprawl index
Franzini 2003 (40)	Texas, United States; 1991	Analysis among cases of death by heart disease; premature cardiovascular mortality	Census block groups, census tracts, counties	Census tract or block-group level: median house value, education, homeownership, own ethnic group density
Martikainen 2003 (68)	Helsinki, Finland; 1990	Cohort; mortality from diseases of the circulatory system	Small areas	County level: education, racial/ethnic composition, unemployment, median income, poverty rate, income inequality (90/10 ratio, Robin Hood index), crime, homeownership
Borrell 2004 (11)	4 U.S. communities; 1987–1989	Cohort; cardiovascular mortality	Census block groups	Proportion of manual workers, proportion of over 60-year-olds, social cohesion
Diez-Roux 2004 (32)	4 U.S. study areas; 1989–1990 and 1992–1993	Cohort; cardiovascular mortality	Census block groups	Neighborhood socioeconomic summary score

Horne 2004 (50)	Salt Lake City, United States; 1993–2000	Cohort of CHD patients; all-cause mortality; incidence of myocardial infarction	Zip code areas	Median household income
Marinacci 2004 (67)	Turin, Italy; 1971, 1981, and 1991	Cohort; CHD mortality	Large neighborhoods	Neighborhood deprivation index
Nordstrom 2004 (77)	4 U.S. communities; 1989–1990 and 1992–1993	Cross-sectional; prevalence of subclinical CVD	Census block groups	Neighborhood socioeconomic summary score
Steinland 2004 (101)	United States; 1992	Cohort; CHD mortality	Census blocks	Area-level summary socioeconomic summary score
Stjärne 2004 (103)	Stockholm county, Sweden; 1992–1994	Case-control study; first events of myocardial infarction	Small residential areas	Material deprivation (Townsend index), social fragmentation (Congdon index)
Sundquist 2004 (107)	Sweden; 1986–1993	Cohort; CHD incidence	Small area market statistics units	Neighborhood education, neighborhood income
Sundquist 2004 (105)	Sweden; 1996	Cohort; CHD incidence	Small area market statistics units	Neighborhood deprivation (Care Need Index)
Lawlor 2005 (59)	Great Britain; 1999–2001	Cross-sectional; CHD prevalence (diagnosed)	Electoral wards	Carstairs deprivation score
Leyland 2005 (61)	Scotland; 1998–1999	Cross-sectional; self-reported CVD prevalence	Post code sectors	Carstairs deprivation score, area occupational social class, area smoking
Southern 2005 (99)	Alberta, Canada; 1998–2001	Cohort of patients having undergone cardiac catheterization; survival	Enumeration areas	Median household income
Chaix 2006 (19)	Scania, Sweden; 1980–1993	Cohort; CHD mortality	Parishes	Population density, median income
Petersen 2006 (81)	Southwestern Pennsylvania; 1994–1996	Cross-sectional (untreated hypertensive men); intima-medial thickness, plaque occurrence	Census tracts	Community socioeconomic status summary score
Petrelli 2006 (82)	Turin, Italy; 1997	Cohort; CHD incidence, CHD mortality	Large neighborhoods, census tracts	Neighborhood deprivation score, census tract median income
Stjärne 2006 (102)	Stockholm county, Sweden; 1992–1994	Case-control study; first events of myocardial infarction	Small residential areas	Median income, socioeconomic homogeneity (Gini coefficient)
Sundquist 2006 (104)	Sweden; 1998	Cohort; CHD incidence	Small area market statistics units	Neighborhood linking social capital (proportion voting in local elections)
Sundquist 2006 (106)	Stockholm County; 1998	Cohort; CHD incidence	Small area market statistics units	Neighborhood violent crime, neighborhood unemployment
Carson 2007 (13)	4 U.S. communities; 1987–1989	Cross-sectional; intima-media thickness, ankle-brachial index	Counties (childhood SEP), census tracts (adulthood SEP)	Neighborhood cumulative SEP

(Continued)

**Table 1 (Continued)**

First author, year	Location and date <sup>c</sup>	Study design and outcome(s)	Geographic scale	Contextual variable(s)
Chaix 2007 (20)	Scania, Sweden; 1996	Cohort; CHD mortality	Local areas aggregating parcels	Area population density, area mean income
Chaix 2007 (22)	Scania, Sweden; 1986 and 1996	Cohort; CHD mortality	Local areas aggregating parcels	Neighborhood mean income
Chaix 2007 (21)	Scania, Sweden; 1996	Cohort; CHD incidence, CHD mortality, immediate case fatality, long-term survival after myocardial infarction	Local areas aggregating parcels	Neighborhood mean income, neighborhood residential stability, population density, distance to the hospital
Rosvall 2007 (89)	Malmö area, Sweden; 1991–1994	Cross-sectional; carotid plaque score	City areas	Area social deprivation index
Winkleby 2007 (111)	Sweden; 1995	Cohort; CHD incidence, case fatality	Small area market statistics units	Neighborhood deprivation index, urban/rural status
Augustin 2008 (5)	Baltimore, United States; 2001–2002	Cross-sectional; self-reported history of physician diagnosis of myocardial infarction	Meaningful neighborhoods defined by city planners	Neighborhood psychosocial hazard scale, Townsend deprivation index, index of neighborhood affluence
Chaix 2008 (17)	Scania, Sweden; 1999–2000	Cohort; acute myocardial infarction mortality	Local areas aggregating parcels	Neighborhood social cohesion, neighborhood shared feeling of insecurity, neighborhood income, population density, % of residents from low-income countries, residential stability, distance to the hospital
Morris 2008 (74)	Great Britain; 1992	Cohort; incidence of major nonfatal CHD events	Electoral wards	Carstairs deprivation score
Wight 2008 (110)	United States; 1993	Cross-sectional; self-reported physician-diagnosed CVD	Census tracts	Neighborhood socioeconomic disadvantage

<sup>a</sup>The Pubmed search was based on the following title expressions: “multilevel,” “neighborhood,” “area,” “community,” “geographic,” “context,” and “environment.” These expressions were used in conjunction with the following other title expressions: “mortality,” “cardiovascular,” “cardiac,” “heart,” “ischemic,” “coronary,” “myocardial infarction,” and “atherosclerosis.”

<sup>b</sup>Only the following variables were considered as indicators of individual SEP: education, employment status, occupation, income, public pension assistance, housing tenure, housing density, amenities at home and car accessibility during childhood or adulthood, insurance status, parents’ education, occupation, and housing tenure. Ethnicity was not considered as a SEP indicator.

<sup>c</sup>For cohort studies, we report the baseline year.



of female-headed households (60), crime (40, 106), neighborhood psychosocial hazards (5), electoral participation (104), and social cohesion (17, 68, 103). However, in six studies, the area effect was not adjusted for area SEP (5, 36, 42, 61, 104, 106). A recent study of the association between neighborhood psychosocial hazards and self-reported history of myocardial infarction did not control for neighborhood SEP, but it assessed whether neighborhood psychosocial hazards were more strongly associated with the outcome than was neighborhood SEP (as estimated from separate models) (5). Controlling for individual and area SEP, two studies reported statistically significant associations between urbanicity and CHD (19, 111). The other statistically significant predictors of CHD-CVD that were reported after adjusting for individual and area SEP are related to the social environment, i.e., residential stability (21), presence of female-headed households (60), crime (40), dwelling ownership (40), and social cohesion (17).

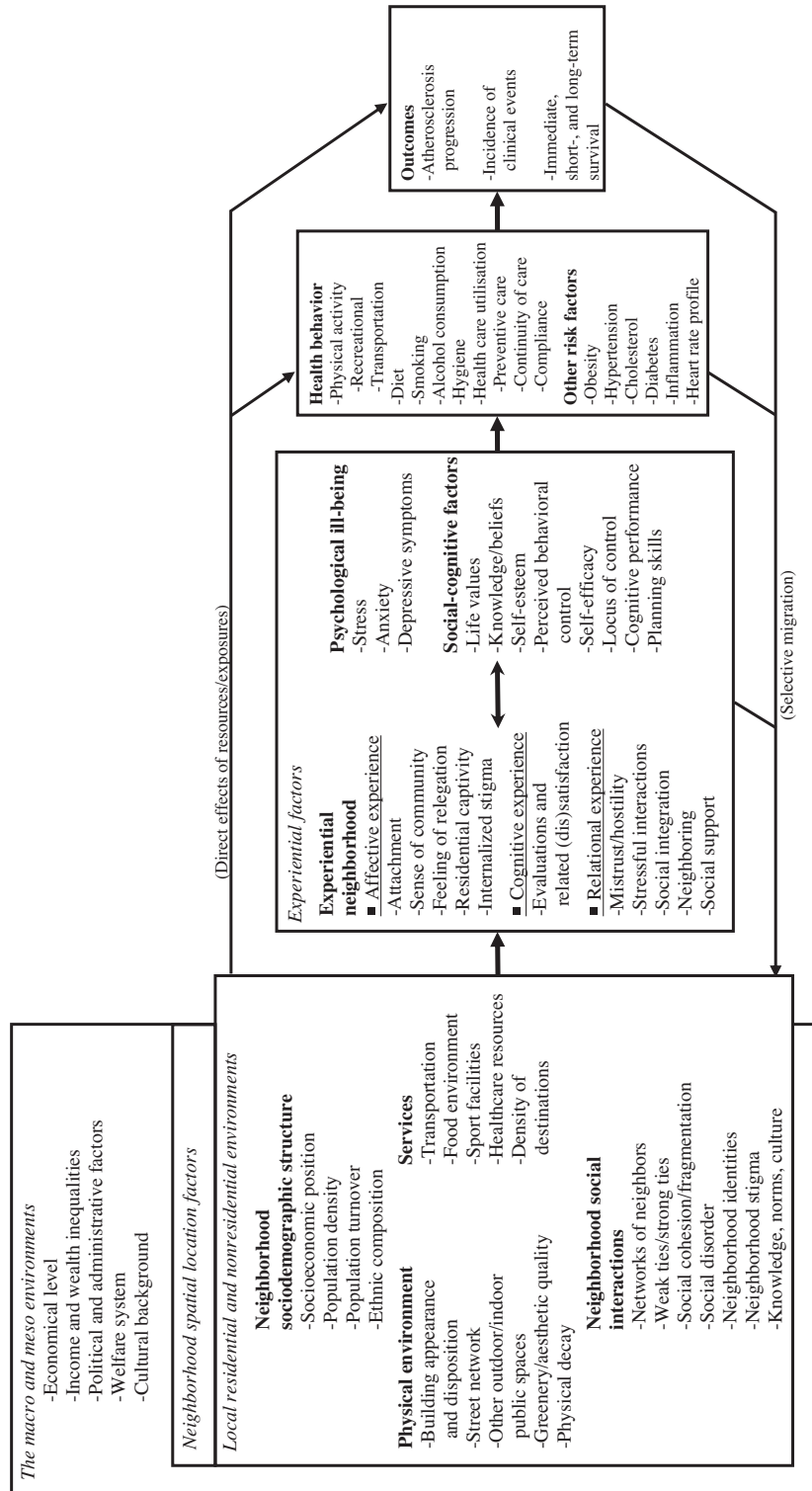
**Mediating mechanisms.** Twenty-four of the 40 studies accounted for some of the traditional CVD risk factors. However, only 15 of them introduced risk factors in a final step in a model with the area effect already adjusted for individual SEP (5, 11, 13, 17, 29, 32, 33, 50, 56, 59, 77, 89, 101, 103, 107), which allowed them to investigate their mediating role in the adjusted environment-health association. A recent study carefully noted, however, that some of the risk factors may be partly confounders rather than solely mediators if individuals with these factors tend to self-select in specific neighborhoods (5). In most studies, the associations between area factors and CHD-CVD remained unchanged or slightly decreased or increased after introducing the risk factors (29). Only a few studies noted a perhaps larger decrease in the area SEP-CVD association after adjusting for risk factors (59, 89, 103). In most studies, because risk factors were included all together, it is not possible to assess whether one of them had a stronger mediating role than the others. Notable exceptions include Lawlor's study (59), in which leg

length (a marker of childhood exposures) was found to have a nonnegligible mediating role in the neighborhood SEP-CHD association, or studies in which smoking was the only mediator investigated (56, 107). One study (103) considered individual social integration as a mediator of neighborhood social fragmentation effects on the incidence of myocardial infarction (social integration, however, was introduced together with the other risk factors). No study, to our knowledge, has investigated the mediating role of inflammatory and hemostatic variables or heart rate profiles. Thus we know very little about the mechanisms involved in the association between neighborhood SEP and CHD.

Overall, a number of research areas, such as the environmental determinants of post-CHD survival, have been relatively neglected in the literature. Moreover, various methodological limitations compromise causal inference on the basis of the estimated associations. As emphasized below, these shortcomings include (a) considerable limitations in environmental exposure assessment (long-term as well as non-residential environmental exposures are neglected, environmental factors are often assessed within arbitrary administrative areas, and a limited number of environmental dimensions are considered); (b) uncertainties about the optimal adjustment strategies and failure to control for selective migration processes; and (c) inability to identify the mechanisms involved in the relationships between environmental factors and CHD-CVD.

## A THEORETICAL CONTRIBUTION TO THE UNDERSTANDING OF NEIGHBORHOOD EFFECTS: A FOCUS ON THE SOCIAL-INTERACTIONAL ENVIRONMENT

Building on previous contributions (27, 30, 97), **Figure 1** provides a theoretical model of the possible effects of geographic life environments on CHD. At the local level (see legend for a description of the spatial scales), our model



**Figure 1**

Theoretical model describing hypothesized processes between the geographic life environments and coronary heart disease. Four spatial scales are distinguished: the macro level defined on a regional or national scale; the meso level, or large territories within which individuals travel regularly in their activities; the local level with a collection of proximate geographic environments including the residential environment, the geographic work environment, environments for leisure-time activities; and the micro level at which individuals interact with their environment. Neighborhood spatial location factors refer to the central or peripheral position of each local neighborhood in the meso/macro organization.



distinguishes between neighborhood sociodemographic variables, the physical environment, services available in the vicinity, and the social-interactive environment. Because of space limitations, pathways related to the physical and service environments, which as described elsewhere may be of considerable importance (30), are not covered here. Similarly, biological pathways from risk factors or psychological states to CHD are out of the scope of the present contribution.

Instead, to efficiently complement previous theoretical contributions (27, 30, 97), we focus on neighborhood social interactions, with a particular interest in neighborhood identities, which have been relatively neglected. We posit that neighborhood experiences influence the psycho-cognitive antecedents of behavior, thereby mediating effects of neighborhood social interactions on behavior. We build on sociology and social and environmental psychology to explore these hypotheses. Such notions are developed here in a theoretical framework for CHD but are relevant to other health outcomes, as well.

### Neighborhood Social Interactions

Rationale for considering neighborhood social interactions as key variables in a model for CHD includes the consistent literature reporting associations between individual social support and CHD (72). Far from restricting the focus to the built environment (80), any ecopidemiologic model of CHD and its risk factors should consider social interactions, at least as potential confounders, modifiers, or mediators of physical or service environment effects. Confounding or mediation can occur because social interactions are shaped by the neighborhood sociodemographic, physical, and service environments. Indeed, (a) concentrated forms of habitat combined with socioeconomic disadvantage may contribute to feelings of overcrowding and may affect social relationships (35); (b) the availability of pedestrian areas may foster social interactions (30); (c) a high neighborhood population turnover may weaken local

networks (92, 95); (d) the presence of services may promote social participation among residents; but (e) a high concentration of services attracting nonresidents may result in colder relationships (unfamiliarity among pedestrians). Because social interactions (e.g., through collective efficacy) may reciprocally influence the physical or service environment, virtuous or vicious circles may operate between them (24).

**Social cohesion, social fragmentation, and social disorder.** Building on social sciences (92, 95) and social psychology (12), we define neighborhood social cohesion as the emerging property of a complex state of relationships in the neighborhood characterized by the presence of extensive, omnipresent, and interpenetrated networks of neighbors (41), shared feelings of attachment and belonging to the neighborhood (“we-ness”) (100), and the resulting residents’ ability to intervene collectively on behalf of the common good and provide support to each other.

In addition to the strong ties among friends, relatives, or close neighbors, some researchers have emphasized the importance of the weak ties that take place in public spaces (41, 46). Weak or bridging ties (98) have been depicted as the social cement that integrates a community by bringing together otherwise disconnected networks (93).

Conversely, socioeconomic disadvantage combined with high population turnover is known to dissolve networks of neighbors (92). A possible consequence of social fragmentation (115) is neighborhood social and physical disorder, because of neighbors’ diminished control over the deviant social behavior and physical decay of their environment (95). Social disorder (43, 87), whose definition depends to some extent on the cultural background of populations, refers to the perpetration of asocial behaviors combined with residents’ incapacity to intervene collectively to limit the damage. Disorder manifests itself through a number of visible signs that contradict a clean, well-maintained, safe, and healthy environment (prostitution, public drunkenness, vandalism, incivilities, drug

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#### Neighborhood social cohesion:

relationships characterized by the presence of interpenetrated networks of neighbors and feelings of attachment and belonging to the neighborhood

**Social disorder:** the perpetration of asocial behaviors combined with residents’ incapacity to intervene collectively to limit their damage

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### Neighborhood

**stigma:** neighborhood stigma may be defined by the co-occurrence of its components: labeling, stereotyping, separation, status loss, and discrimination

### Experiential

**neighborhood:** the experiential neighborhood refers to the wide set of affective, cognitive, and relational experiences individuals have in their neighborhood

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dealing, etc.) (87, 112). As such circumstances encourage residents to withdraw further from public spaces, vicious circles strengthening disorder are likely to operate (6, 93).

### Neighborhood identities: sources of reward or stigma.

In the aforementioned definition of neighborhood social cohesion, apart from its relational component (neighborhood networks) and affective component (attachment and belonging), a cognitive dimension was introduced, i.e., neighborhood identities (108) (all these dimensions are hypothesized to influence each other). Neighborhood identities imply a labeling of the neighborhood, delimitation of membership, and shared representations (endogenous or exogenous to the residents) characterizing it. Contrary to sociologists and environmental psychologists (58), health researchers, with few exceptions (63), have largely neglected neighborhood identities, focusing on more tangible realities such as neighborhood networks.

We argue that neighborhood identities may independently affect well-being and behavior. Neighborhood identities may convey a positive image of the neighborhood, which would be rewarding for residents, but may also contain negative representations. We believe a key factor to measure in future research is neighborhood stigma. Stigmatization processes are investigated for ethnic minorities, HIV patients, obesity, etc. (62, 64), but stigmatized neighborhoods have received almost no attention. Building on this earlier literature, neighborhood stigma may be defined by the co-occurrence of its components: labeling, stereotyping, separation, status loss, and discrimination.

### Neighborhood norms and capital of knowledge.

Through contagion processes, individual behavior may be affected by the neighborhood residents' health-related habits and norms (social modeling) and collective knowledge and beliefs (30). Even if other socialization contexts are more influential (e.g., family, work, nonneighborhood-based networks), local norms may affect individuals' habits through

observable behavior in the public space and through direct contacts with local friends and acquaintances.

## The Experiential Neighborhood

Beyond commonly considered experiences (e.g., feeling of insecurity), eco-epidemiologists should assess neighborhood experiences in a systematic way (6, 112), categorize them in a coherent framework, and derive psychometric tools to measure them. The next section relies on environmental psychology to draw a preliminary map of the experiential neighborhood, i.e., these interconnected, albeit distinct, experiences made in the neighborhood. We distinguish between affective, cognitive, and relational neighborhood experiences (even if these are indissolubly intertwined).

**Neighborhood affective experiences.** Place attachment is defined as positively experienced bonds that are developed over time from the behavioral, affective, and cognitive ties with the socio-physical environment (10). A related concept is that of a psychological sense of community (23), sometimes defined using four components (69): sense of belonging to the neighborhood, influence (a sense of mattering to the group), a feeling that one's needs will be met, and emotional connection with neighbors.

These constructs are relevant to eco-epidemiology as key to psychological well-being, but epidemiologists may be interested particularly in antagonistic feelings such as aversion to the residential environment, feeling of social relegation, or residential captivity [feeling powerless to escape from one's neighborhood because of financial reasons (88)].

In the present model, we propose a distinction among neighborhood-related experiences between safety- or life habit-threatening experiences and identity-threatening experiences. Only the former dimension is referred to as stressful environmental experiences because they imply a perceived or real threatening circumstance in the environment, with which the person feels powerless to cope. In

our broad definition, these stress-generating environmental demands are conceptualized as threatening the safety of the person, of her/his friends or relatives, and of their material goods, or disturbing any aspects of their life habits.

Regarding identity-threatening experiences, environmental psychologists have focused on place identification processes. As individuals often refer to themselves as members of their neighborhood, place identities may intervene in the self-construction process (108). Place identity, as a potpourri of cognitions on the local physical world, contributes to self-definition (84), providing an identity-enhancing context for one's biography (58). In that process, neighborhood residents acquire certain quasi-psychological characteristics associated with their neighborhood (58).

A critical aspect for eco-epidemiologists is that transfers between the image of the neighborhood and the self-image also operate in stigmatized neighborhoods. Living in a stigmatized neighborhood would preclude relying on one's neighborhood in the self-construction process. Furthermore, negative symbolic loads may be transferred from the neighborhood-stigmatized identity to residents' own identities, incorporating negative attributes in the self-concept. Internalized neighborhood stigma may result in feelings of shame about one's neighborhood, deteriorated self-esteem, fear of discrimination, and avoidance of social interactions with nonmembers of the stigmatized environment (thereby reducing life opportunities). Thus, eco-epidemiologists should focus on neighborhood stigma-induced identity threats.

**Neighborhood cognitive experiences.** We conceptualize individual perceptions of the environment as cognitive experiences because they imply a cognitive processing of environmental information. As such, they constitute relevant predictors of behavior in their own right rather than simple surrogates of objective environmental variables. Indeed, rather than being neutral assessments, cognitive evalua-

tions are associated with judgments on the quality of environmental features and satisfaction or dissatisfaction with them (mix of cognitions and affects) (6, 10) and should be surveyed as such. Discrepancies between objective indicators and the corresponding subjective evaluations of the environment (9) indicate that complex individual-environment interactions that may affect health behavior are at play, which is probably relevant to eco-epidemiologists (e.g., a shared feeling of insecurity in the neighborhood may contribute to a perceived lack of access to sport facilities, even when such facilities are available in the vicinity).

**Neighborhood relational experiences.** In assessing neighborhood relational experiences, useful distinctions can be made between their factual bases (social integration in neighborhood networks), the neighboring activities themselves (e.g., visiting local friends at their homes) (98), neighboring affects (e.g., trust in the neighbors), preferences regarding neighbors (neighborliness, i.e., the willingness or not to develop social contacts), cognitions (opinions on neighbors), and their product (e.g., social support). In addition to assessing strong ties, it is important to assess the weak ties individuals have in their neighborhood because these may be important in the genesis of neighborhood attachment (46) and provision of support.

However, neighborhood relational experiences also constitute sources of stress. In their assessment, researchers should consider not only the most severe offenses, but also common experiences present in a broader range of neighborhoods (e.g., conflicts with neighbors) (112). However, stress induced from social interactions may stem not only from direct interactions, but also from the visible cues of disorder reified in the environment (e.g., graffiti, vandalism, etc.) (6).

Ironically, neighborhood processes that contribute to the presence of sources of stress may at the same time dissolve the social networks residents would need to cope with them. Thus, these two intertwined processes may

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**Place identity:** place identity is a potpourri of cognitions on the local physical world that contributes to self-definition

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increase both exposure to stressors and environmental susceptibility to stress (34).

### **The Psycho-Cognitive Antecedents of Behavior**

Drawing on social-cognitive theories, we now emphasize that articulating neighborhood experiences with the psycho-cognitive antecedents of behavior may provide a path between neighborhood factors and behavior (91).

**The theory of planned behavior.** Ajzen's theory of planned behavior (1) states that intentions to perform a behavior are influenced by individuals' attitudes toward the behavior (positive or negative evaluations), subjective norms (perceived social pressure), and perceived behavioral control. In a second step, both intentions and perceived behavioral control contribute to predict action. Perceived behavioral control is defined as the perceived ease or difficulty of performing the behavior and incorporates perceptions of environmental impediments (e.g., limited access to sport facilities). The theory emphasizes that individuals' salient beliefs intervene as determinants of attitudes, subjective norms, and perceived behavioral control and proposes to construct measures of these three dimensions by aggregating survey information on the corresponding beliefs (belief-based measures).

Thus, the following two-step strategy may be considered to articulate neighborhood effects with constructs from the theory of planned behavior (44): first, investigating how the neighborhood environment shapes individual beliefs that influence attitudes, subjective norms, and perceived behavioral control for a specific health behavior (with a particular interest for beliefs that are related to the environment, e.g., perception of environmental impediments to active living); second, assessing whether belief-based measures of attitudes, subjective norms, and perceived behavioral control mediate part of the associations between contextual characteristics and health behavior.

**Self-efficacy, locus of control, and controllability.** In Bandura (7), perceived self-efficacy is defined as people's beliefs about their ability to exercise control over their own functioning and events. Perceived self-efficacy may influence choices of activities, preparation for an activity, effort expended during performance, how long people persevere in the face of difficulties, and whether their thoughts are self-hindering or self-aiding.

In contrast, according to Rotter's notions of internal versus external locus of control, a person will engage in a health-enhancing behavior only if she/he believes that her/his actions will lead to the expected outcome (health, well-being, etc.) (90). "Internals" perceive a causal relationship between their behavior and the outcome, whereas "externals" believe that forces beyond their control (fate, powerful others, the complexity of the world, etc.) determine the occurrence of the outcome.

Locus of control is a generalized expectancy that one's behavior influences desired outcomes (90), whereas perceived behavioral control and self-efficacy beliefs are more behavior- and situation-specific (1) and refer to control over the behavior rather than over its outcomes (2). Confronting these notions, some have called for the need to distinguish between confidence in one's motivation and ability to carry out a behavior (self-efficacy) and perceptions of control over required external resources (controllability) (2, 66).

Such notions are relevant for understanding pathways from neighborhood factors and related experiences to behavior. On one hand, a literature suggests that perceived social disorder and related stress (experienced when environmental demands seem to exceed capacities to deal with them) may contribute to a general feeling of powerlessness, which may have repercussions on health behavior (43). Deprived neighborhood residents, in experiencing a lack of control over their residential environment and an inability to move to another neighborhood, may learn that their own actions cannot produce the desired outcomes. On the other hand, as a distinct mechanism, neighborhood

deprivation and related stigmatized neighborhood identities may be associated with depressive symptoms (86) and threatened self-esteem and, in turn, with decreased feelings of personal efficacy. These two separate pathways, mediated by stress and threatened identity, may contribute to the link between neighborhood experiences and health behavior.

In this section, we did not aim to describe all the pathways depicted in our theoretical model in **Figure 1**. Our goal was to generate new hypotheses by focusing on a relatively neglected subpart of the model, i.e., how the neighborhood social-interactive environment influences neighborhood experiences and, in turn, the psycho-cognitive antecedents of behavior and health behavior.

## METHODOLOGICAL ADVANCES IN ECO-EPIDEMIOLOGY

We now discuss general methodological issues relevant to the investigation of neighborhood effects on CHD and other health outcomes.

### Delimiting Geographic Life Environments

**Theoretical considerations on neighborhood delimitations: toward assessment of exposure areas.** To guide us in the operational definition of neighborhoods, we posit that neighborhood delimitations should capture the environmental conditions to which individuals are exposed in their local environment, i.e., that neighborhoods should be viewed as exposure areas.

This criterion contradicts several commonly proposed approaches. First, some authors assume that neighborhoods should be defined as homogeneous in terms of the environmental exposure (49). However, because individuals may be locally exposed to heterogeneous environmental circumstances, there is no reason to select homogeneity as a criterion in itself. If individuals are locally exposed to heterogeneity, then our measures should reflect it.

Another definition proposed in the literature relies on resident-perceived neighborhood boundaries (as assessed with mental maps) (25). However, such boundaries do not necessarily match residents' objectively experienced neighborhoods (both being of interest for eco-epidemiologists) (47). Because one's neighborhood is a component of the self-concept, resident-expressed boundaries (what people would like them to be) may not permit reflect true exposures. For example, an individual may exclude the particularly deprived nearby block she/he crosses in her/his four-minute daily walk to the transportation station from her/his idealized neighborhood definition.

An adequate operational definition of objectively experienced neighborhoods should consider individuals' local activities by, e.g., drawing home-centered neighborhood boundaries on the basis of the 10 local destinations where individuals most frequently go. Such a definition of exposure areas based on interaction spaces implies that each factor should be measured on a specific scale, at the geographic level at which individuals effectively or potentially interact with it.

**GIS procedures to define neighborhood delimitations.** Developing automatic geographic information systems (GIS) procedures (49) to approximate objectively experienced neighborhoods is thus of high relevance. A distinction is made between fixed and sliding (or ego-centered) neighborhood boundaries. Ego-centered areas, i.e., areas centered on individuals' residences (18, 47, 83), may be circular or, more appropriately, may take into account the local street network (45, 47). Because activity patterns are limited by physical barriers (e.g., natural obstacles, major roads), combining sliding and fixed boundaries (by truncating the former using the latter) may be a relevant option. Sensitivity analyses comparing effects of variables on different scales and using measures of clustering as proposed elsewhere (70) may help investigators to draw inferences on the spatial scale of influence of the environmental factor investigated (18).

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**GIS:** geographic information systems

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**Nonresidential**

**environments:** the geographic work environment, leisure-time geographic environments, etc. (as opposed to the residential environment)

**Ecometrics:** used to characterize geographic or nongeographic life environments with geographic information systems, smoothing techniques, random effect regression models, etc., or a combination thereof

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However, individuals' activity patterns do not equally spread in every direction around their place of residence, i.e., their activities often take place in neighborhoods oriented in a particular direction. A GIS approach to operationalize such oriented neighborhoods may be to distend the neighborhood shape toward the closest major road, shops, or transportation station. Finally, because one's street is one's most immediate living space (people are more familiar with neighbors on the other side of their street than with residents from the other side of their block), precisely geocoded databases may be used to define environmental factors at the street level or street segment level (rather than at the block or block-group level as usual).

**Non-residential environments.** Considering that "real-life spatial trajectories" are not contained within local neighborhoods, Cummins recently warned us about the "local trap," defined as the methodological belief that "the 'local' is always the appropriate scale" (26). Obviously, it would be nonsense to measure, e.g., employment market characteristics or the availability of hospital services on such a local scale. However, although we share concerns about the exclusive reliance on "local residential environments," we feel that Cummins may have missed the right target, criticizing the "local trap" instead of the "residential trap." In contrast, we argue that daily trajectories could be operationalized as a collection of local geographic environments such as the residential neighborhood, the geographic work environment, and leisure-time geographic environments (51).

However, assessing nonresidential environments is no easy task. Should we assess (a) travel destinations over a short recall period, (b) salient nonresidential environments, i.e., those of which individuals spontaneously think when asked about their geographic life environments, or (c) the places where specific activities from a predefined list regularly take place (work, shopping, etc.)? Although the first approach is adequate to correlate, e.g., short-

term exposure to pollution with blood pressure, the third approach may be more appropriate to assess long-term exposures to aspects of the physical, service, and social-interactional environments that heighten CHD risk. Using these data, cumulative measures (combining residential and nonresidential exposures) may be constructed. Also, nonresidential exposures may be seen as confounding or modifying residential exposure effects (e.g., through compensation mechanisms) (51).

More attention is also needed to the temporal scale of neighborhood exposures, which should be made coherent with the temporal patterns of the disease (long-term exposures influencing atherosclerosis progression versus acute stressors triggering CHD events) (not further developed here).

### Assessing Environmental Characteristics

Upcoming challenges in eco-epidemiology imply an attention shift from modeling issues (e.g., multilevel models) to issues of measurement of the relevant exposures, mediators, and confounders. Building on a previously proposed concept (85), we employ the term ecometrics to designate a broad field of methodologies that are used to characterize geographic or nongeographic life environments using GIS, smoothing techniques, random effect regression models, etc. or a combination thereof, applied to survey, observation, administrative, and geographic data.

### Georeferenced administrative databases.

The increased availability of sociodemographic data from administrative sources geocoded at the building level (e.g., the census, tax registries, or housing price databases) allows us to characterize neighborhood socioeconomic characteristics within local, residence-centered areas using simple aggregation or smoothing techniques. Also of interest, a currently developed approach, which imports econometric techniques into ecometrics, is to rely on hedonic models applied to housing price data to



assess the degree of attractiveness of residential neighborhoods (54).

Recently released GIS data have allowed us to improve the measurement of built and service environment variables. Examples of refinements include measures of accessibility that are based on the street network and that incorporate information on destination attributes, and the definition of innovative indicators characterizing the built environment with two-dimensional data (e.g., the perimeter-to-area ratio to assess building compactness) or three-dimensional data (e.g., the street-width-to-building-height ratio as contributing to environment pleasantness).

### **Systematic observation/audit of resources.**

For constructs that are not measurable with administrative data, one alternative is to send trained raters with checklists to document aspects of the environment (94). This approach is particularly sound for the consistently observable and univocally interpretable aspects of the environment (15). However, it may be less reliable for dimensions of the social environment (14, 114). For example, many visible cues of social disorder cited in the literature are either intermittently observable (e.g., public drunkenness) or equivocal (graffiti may have complex meanings), or both (e.g., adults loitering). Moreover, neighborhood constructs that are not physically reified but exist only or mostly in residents' perspectives (e.g., neighborhood identities) are invisible to systematic observation (85).

**Survey of neighborhood residents.** Another approach is to rely on residents' evaluations to assess neighborhood exposures using complementary survey questions to assess each specific neighborhood dimension. Three-level random-effect models (survey items nested within individuals, and individuals nested within neighborhoods) with answers to these questions on the environment as their outcomes are employed to aggregate information at the neighborhood level to form environmental indicators (75, 85). Using this approach, the

neighborhood-level random effect estimated from the econometric model is subsequently used as an environmental variable to predict health outcomes. More recently, some authors have relied on geostatistical techniques to estimate continuous surfaces of these environmental attributes and to derive summary measures for buffers around individuals' homes (4, 31). Only one simplified application of these approaches has focused on CHD (17).

It is not clear whether the ideal design is to rely on the participants of the epidemiologic study, or on participants of a separate sample (3, 73, 75, 76), to answer to the econometric survey questions. That the same dimensions are available as both experiential variables at the individual level and environmental variables on the neighborhood scale is a strength of the first design. However, same-source biases may exist in this design (3) because the study participants provide information on both the environmental exposures and the health outcomes. An imperfect solution to this problem is to estimate the econometric environmental variable for each participant's neighborhood from an econometric model excluding that participant.

Future research will have to determine whether we can strengthen our econometric models by controlling for individual raters' characteristics that may bias their environmental assessment. Another promising avenue is to incorporate into the econometric models objective environmental variables as predictors of the individual answers to the questions on the environment (4). The aim of this innovative approach is to integrate individual perceptions of the environment with objective contextual data to form composite and more reliable neighborhood variables. However, the perceptual items and objective indicators should refer strictly to the same environmental construct (e.g., objective data on accessibility to parks and perceptions of park quality, crime summary data and individual fear of victimization). Thus including neighborhood SEP as a predictor in the econometric model, as recently done (4, 73), may artificially increase the correlation between the econometric indicator and SEP,

contradicting our initial aim of constructing more specific exposures than neighborhood SEP.

Moreover, in the simple econometric models that do not incorporate objective neighborhood predictors, an appealing consequence of neighborhood random effect shrinkage is that the neighborhoods in the most contrasted exposure categories of the econometric variable tend to be those with the largest number of individual raters. In contrast, neighborhoods with a low number of survey participants are pulled toward the intermediate exposure categories (17). This appealing property may be lost when objective environmental factors are introduced as predictors into the econometric models.

Finally, because econometric variables are model-based rather than observed quantities, it is necessary to account for the uncertainty in these estimates when modeling their associations with CHD, e.g., by simultaneously estimating the econometric model and the model for CHD with Markov chain Monte Carlo techniques.

## Identifying Specific Environmental Determinants of CHD

### Should we adjust for neighborhood SEP?

The need to consider more specific environmental factors than general neighborhood sociodemographic variables does not imply that neighborhood SEP can be eliminated from our models, as done in recent studies (3, 73, 76). We contend that, in many cases, at minimum, we should report associations between specific environmental factors and health adjusted for neighborhood SEP.

Building on directed acyclic graphs (37), we constructed a series of fictive graphs to guide us in our reasoning (see online Appendix 2 for details). In summary,

- Failure to adjust for neighborhood SEP could produce spurious associations between any causal antecedent or causal product of neighborhood SEP (e.g., the presence of a beauty salon) and CHD.

- We would not need to adjust for neighborhood SEP if a large number of environmental exposures/resources (some of them unmeasured) were not all correlated with each other in relation to SEP. However, basic observation suggests that they are.
- Because specific environmental exposures/resources are more proximal determinants of health than SEP, their effects should persist after controlling for neighborhood SEP if truly associated with CHD. Thus, adjustment for neighborhood SEP is a safe strategy.
- Neighborhood SEP is conceptualized as a convenient neighborhood adjustment factor because controlling for it may simultaneously adjust for a wide set of environmental confounders. Similar comments apply to other neighborhood structural factors such as the degree of urbanicity of the area.
- However, as there may be residual correlation among environmental exposures/resources conditional on neighborhood SEP, controlling for it is a useful but imperfect adjustment strategy.

Even if the specific environmental exposures/resources are conceptualized as components of neighborhood SEP, one should assess the effect of each component adjusted for the others. Thus, we recommend that studies investigating effects of specific environmental factors report associations adjusted for neighborhood SEP, at least as a complementary analysis.

### Plausibility of associations with CHD? To

understand the underlying mechanisms of the associations between the environment and CHD, is our strategy to incorporate in our models for CHD those contextual variables that are associated with physical activity, dietary behavior, and obesity? What can we expect from this approach?

Building on Daniel's perspective (27), we find it relevant to question the sociological, sociopsychological, and biological plausibility

of a number of hypothesized associations with CHD. For example, one could expect an association between the availability of sport facilities and physical activity. However, because of measurement error and an intrinsic weakness of the relationship investigated, it is much less likely that such an environmental effect on physical activity would translate into an analytically identifiable effect on CHD.

A number of such environmental effects on CHD may not reach statistical significance individually, but may, when combined into a cumulative effect, result in a nonnegligible influence. Thus, constructing variables combining different environmental exposures may be explored in future research (76). We would aim to replace neighborhood SEP (an opaque variable) with a composite environmental risk score constructed through a reasoned process.

### Testing Mediation Models

One reason for considering neighborhood experiences is because they fill a gap in the mediational chain between objective environmental factors and CHD (24), delineating a path for the embodiment of neighborhood influences. Second, these experiences may be less correlated with each other than are neighborhood factors, thus helping researchers to differentiate among the various mechanisms.

Overall, on the basis of **Figure 1**, the aim is to test a mediational model (24), suggesting that neighborhood sociodemographic variables influence CHD through specific environmental exposures/resources, resulting neighborhood experiences, psycho-cognitive antecedents of behavior, health behavior, and risk factors. However, it may be empirically difficult to test this theoretical model. The available data, with their measurement errors and imperfect temporality, may limit us to testing only simple mediational chains (16). Moreover, the decomposition of environmental effects into direct and indirect effects, despite its intuitive relevance in mediation analyses, may be valid only when applied to the linear model and when stringent assumptions are respected (53).

A major challenge will be to construct psychometrically sound instruments to assess neighborhood experiences, complementing those available (10, 12, 23, 69, 112). It will be key to define sufficiently separable experiential scales to disentangle the effects of the different interconnected experiences. Also, particular care is needed to differentiate between ecometric and psychometric items, e.g., between an ecometric assessment of neighborhood stigmatization processes and a psychometric assessment of residents' internalization of a stigmatized neighborhood identity.

### Selection Processes and Empirical/Structural Confounding in Observational Studies

**Selective migration and the selective location of resources.** Epidemiologists have started to recognize that causal inference in observational neighborhood effect studies is plagued by selection processes (78, 79). At least two selection processes may be distinguished. First, associations between the availability of resources and health outcomes may be biased by the selective location of resources by economic or public agents near populations willing to use them (30). A second selection process refers to the (voluntary or constrained) migration of individuals with specific characteristics affecting the outcome toward particular neighborhoods. Selective migration processes, and the magnitude of confounding, differ according to the specific individual characteristic selectively brought into particular neighborhoods: the outcome itself or one of its more or less proximal determinants, e.g., another health variable, a health behavior, or more distal factors.

**Empirical or structural confounding of neighborhood effects?** It has been argued that selection processes may suppress the exchangeability of individuals between contrasted neighborhood exposure categories, hindering the counterfactual assessment of causality (65). To grasp Oakes's criticism of observational neighborhood studies (78, 79), we distinguish

between empirical confounding (a model limitation) and structural confounding (a limitation in the data). Empirical confounding refers to the lack of adjustment of a neighborhood effect for individual characteristics that are empirically adjustable in a well-performing model. Conversely, structural confounding is our inability to properly control for important individual confounders because of the almost complete separation of the distributions of these confounders in the contrasted exposure groups. If that were true, we would be left with a choice between a well-performing but incorrectly adjusted model and a model making excessive extrapolations to disentangle inseparable effects.

To strengthen evidence, supporters (78, 79) of this argument should empirically explore, in *different* populations, the extent to which there is or is not significant overlap in *various* individual self-selection factors across categories of *diverse* neighborhood exposures (i.e., not only in the United States, not only individual SEP, and not only neighborhood SEP).

#### **Addressing empirical confounding.**

Propensity score matching has been applied (32) to ensure that adjustment of regression models for individual self-selection factors is not based on excessive extrapolations (79). This approach, however, does not address unmeasured confounders.

Overall, no methodological shortcut may exempt us from collecting precise information on selective migration processes. Even instrumental variables may offer only a partial solution in the absence of specifically collected data (38). Thus, to the extent that these are sources of confounding, we need to explore, identify, and measure the complex mechanisms influencing the propensity to move in one neighborhood or another.

Recent groundbreaking papers (39, 48) suggest it is feasible to assess neighborhood selection factors (reasons for selecting a particular neighborhood), enabling investigators to separate truly environmental effects from confounding influences predisposing study participants to both the exposure and the out-

come. Building on Frank's framework (39), it may be relevant to distinguish among (a) general life values, (b) general neighborhood preferences, and (c) neighborhood selection factors. Of course, assessing the relevant selection factors may be more complex for CHD than for a definite health behavior such as physical activity.

**Interventions as the key?** Some researchers have argued that randomized community trials offer a "superior research strategy" to perform causal inferences on neighborhood effects (78, 79). Although intervention studies are of course needed to assess intervention effects, we argue that, in most cases, they do not allow researchers to draw causal inferences on the neighborhood effects that operate out of an intervention context (71). First, to maximize efficiency, interventions generally combine multiple components (8), prohibiting investigators from making inferences on a specific neighborhood process. Second, residents' awareness of an environmental intervention occurring in their neighborhood and their involvement in the process are often seen as keys to the intervention success (57), resulting in social placebo effects. Therefore, an intervention specifically designed to draw inferences on everyday world neighborhood effects (i.e., a single-component intervention that is blind to the target populations) may be rather inefficient. We thus argue that inducing behavior change and performing inferences on everyday world neighborhood effects are antagonistic objectives.

Aside from community trials, another strategy is to randomly assign individuals to distinct neighborhoods through relocation programs (55). However, we are skeptical that the specific "treatment" investigated (winning a lottery that artificially relocates an individual to a more affluent neighborhood, which is a rather unnatural scenario) could provide any causal information on neighborhood effects as they operate out of an intervention context.

But we have a more serious objection. Proponents of the use of intervention studies in

eco-epidemiology argue that a benefit of randomization is to dissolve the link between neighborhood exposures and the associated unmeasured individual variables biasing the causal contrasts estimated (78, 79). We argue that socioepidemiologists need to do the exact opposite, i.e., investigate, understand, and adjust for, rather than artificially dissolve, these potential sources of bias (71). For example, through selective migration, general life values may confound neighborhood effects. However, these cognitive constructs may also modify neighborhood influences (39). Overall, we need to know how neighborhood exposures affect the actual people residing in places where they operate—with their specific resources and vulnerabilities—rather than how they would affect exchangeable individuals artificially randomized to these neighborhoods.

## CONCLUSION: A RESEARCH AGENDA

On the basis of our review and theoretical and methodological considerations, a research agenda to improve our understanding of the mechanisms through which geographic life environments may influence CHD should include the following tasks:

1. Strengthen theories of the mechanisms through which geographic environments may influence CHD;
2. Attempt to define ego-centered neighborhood delimitations capturing objectively experienced environmental exposures;
3. Verify that individual propensities to reside in an exposed neighborhood show

some overlap across neighborhood exposure categories;

4. Strengthen models' adjustment by controlling for residual sources of confounding at distinct levels, e.g., childhood SEP and neighborhood SEP;
5. Develop questionnaires to quantitatively describe selective migration processes;
6. Compare the various methods to control for selection processes;
7. Conduct comparative investigations of neighborhood influences on atherosclerosis progression, CHD incidence and mortality, and post-CHD survival;
8. Develop various strategies, organized in the econometric toolbox, to measure environmental factors;
9. Assess nonresidential environments and incorporate them into the analyses;
10. Construct psychometric tools to assess the wide set of neighborhood experiences;
11. Compare objective environmental indicators with the corresponding subjective perceptions, assuming that discrepancies between them may stem from complex individual-environment interactions possibly relevant to health behavior;
12. Incorporate neighborhood identities and stigma, and their experiential repercussions, in the analytical models; and
13. Test mediational models to investigate the underlying mechanisms of neighborhood effects.

These approaches will contribute to the advent of the next generation of neighborhood and health studies.

## SUMMARY POINTS

1. Approximately 40 published studies have investigated associations between social characteristics of the residential environment and coronary heart disease or cardiovascular disease, a significant number of them reporting an increased risk among residents of socially deprived areas after controlling for individual socioeconomic characteristics.
2. Our theoretical model posits that, in addition to the physical and service environments, neighborhood social interactions may contribute to the socioeconomic disparities in coronary heart disease and its risk factors observed among neighborhoods.

3. In addition to the common notions of social cohesion, social networks, and social disorder, eco-epidemiologists should consider neighborhood identities and stigmatization processes as possibly relevant to health.
4. The experiential neighborhood, i.e., the wide set of affective, cognitive, and relational experiences individuals have in their geographic environments, could mediate part of the associations between these environments and coronary heart disease and its risk factors.
5. Early studies on neighborhoods and health have focused on modeling issues (e.g., multi-level models). Upcoming challenges in eco-epidemiology imply an attention shift to issues of measurement of the relevant exposures, mediators, and confounders, with particular attention given to the delimitation of neighborhoods as exposure areas, to nonresidential geographic life environments, to the ecometric assessment of environmental factors and psychometric assessment of neighborhood experiences, and to the identification and measurement of selection processes.

## DISCLOSURE STATEMENT

The author is not aware of any biases that might be perceived as affecting the objectivity of this review.

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33. The first study to report that neighborhood socioeconomic disadvantage was independently associated with the incidence of coronary heart disease.

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39. A key attempt to conceptualize and measure self-selection factors and incorporate them into the analysis of neighborhood effects.

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