

# Residential Proximity to Environmental Hazards and Adverse Health Outcomes

Jean D. Brender, RN, PhD, Juliana A. Maantay, PhD, MUP, and Jayajit Chakraborty, PhD, MS

How living near environmental hazards contributes to poorer health and disproportionate health outcomes is an ongoing concern. We conducted a substantive review and critique of the literature regarding residential proximity to environmental hazards and adverse pregnancy outcomes, childhood cancer, cardiovascular and respiratory illnesses, end-stage renal disease, and diabetes.

Several studies have found that living near hazardous wastes sites, industrial sites, cropland with pesticide applications, highly trafficked roads, nuclear power plants, and gas stations or repair shops is related to an increased risk of adverse health outcomes.

Government agencies should consider these findings in establishing rules and permitting and enforcement procedures to reduce pollution from environmentally burdensome facilities and land uses. (*Am J Public Health*. 2011;101:S37–S52. doi:10.2105/AJPH.2011.300183)

## CONCERNS ABOUT HEALTH

and environmental hazards transcend the academic, scientific, and regulatory worlds; they are also of compelling interest to the public, who often recognizes a relationship between environmental hazards and health. In a 1999 national telephone survey among US voters,<sup>1</sup> 74% of respondents thought that environmental factors had an important impact on childhood cancer, and 73% thought these factors had an impact on birth defects. More than 50% of respondents felt that air pollution, contaminated drinking water, and toxic waste had a great deal of impact on a person's health. These concerns often resulted in public perceptions of disease clusters near hazardous waste sites, industrial facilities, and other potential sources of chemical releases. With the advent of geographic information systems, environmental scientists and public health researchers have been able to address these concerns more comprehensively and objectively with the use of various proximity analyses.

We undertook a systematic review of 94 studies that examined residential proximity to environmental hazards in relation to adverse reproductive outcomes, childhood cancer, respiratory and cardiovascular conditions, or other adverse health outcomes. In our review, unlike in previously published reviews, we focused on a wide range of health outcomes in relation to residential proximity to multiple environmental hazards and identified each study's

limitations. If the evidence indicates that residential proximity is associated with poorer health outcomes, regulatory agencies may need to factor in nearby populations when siting industrial facilities, municipal waste sites, incinerators, and other potential sources of emissions.

## APPROACH

We identified studies of environmental proximity analyses in relation to health through the National Library of Medicine's PubMed, using search terms that combined *proximity to* and adverse pregnancy outcomes (birth defects, fetal death, low birth weight, preterm birth, and spontaneous abortion), childhood cancer, cardiovascular and respiratory illnesses, end-stage renal disease, and diabetes. We also identified additional relevant studies in these studies' bibliographies. With respect to cancer, we focused our review primarily on childhood cancers. Given the relatively long induction and latent period of solid tumors in adults, studies would need to include residential histories for as many as 15 to 30 years before a cancer diagnosis to capture pertinent environmental exposures. Although several recently published studies have included extensive residential histories,<sup>2–4</sup> such studies are scarce. Even though various respiratory and cardiovascular conditions may originate many years before manifestation of overt disease, environmental exposures to pollutants could have acute

effects, for example, precipitating asthma attacks or myocardial infarction in susceptible individuals.

We summarized information from each study regarding target populations, type of study design used, health outcomes included, methods of proximity analyses and exposure assessment, major findings, and limitations. We also examined study results for evidence of racial or economic disparities in health outcomes in relation to residential proximity. In addition to discussing overall findings, we summarized the conclusions of studies that had minimal limitations with respect to exposure assessment and outcome.

A wide variety of methods were used in the reviewed studies to examine the relation between proximity to potential environmental hazards and adverse health outcomes, including spatial coincidence analyses (e.g., residence in a zip code with  $\geq 1$  hazardous waste sites), distance-based analyses (e.g., residence  $\leq 1$  mile of industrial facilities as defined by a 1-mile buffer), and pollution plume modeling (i.e., the dispersion footprint of the pollutant as a proxy for exposure). The most frequently used method was distance-based analysis.

## ADVERSE PREGNANCY OUTCOMES

We reviewed 49 studies that examined the relation between residential proximity to 1 or more potential environmental hazards and adverse pregnancy outcomes.

Few studies specifically examined health impacts in relation to race or socioeconomic status (Table 1). Those studies that did had mixed conclusions regarding health impacts on specific racial or income groups. In a study of maternal residential proximity to hazardous waste sites and chromosomal anomalies, increased risk of Klinefelter variants was confined to births to Hispanic women.<sup>6</sup> In the same study population, however, neural tube defects were associated with residential proximity within 1 mile of an industrial facility only among non-Hispanic White women.<sup>10</sup> Among the various ethnic and racial groups studied, Orr et al.<sup>8</sup> noted the strongest associations between maternal residence in a census tract with 1 or more National Priorities List hazardous waste sites and birth defects among American Indians and Alaska Natives. In Israel, Bedouin populations showed increased risk of major congenital malformations and perinatal mortality with residential proximity to an industrial park, but Jewish populations showed no increased risk.<sup>5,9</sup> In a Canadian population, risks for preterm and low birth weight births in relation to maternal residential proximity to highways were strongest among highly educated women and women who lived in wealthy neighborhoods.<sup>7</sup>

We summarize the findings of studies, including limitations, that examined the relation between residential proximity to various environmental hazards and adverse pregnancy outcomes in a supplemental table (available as a supplement to the online version of this article at <http://www.ajph.org>). In several studies, investigators noted positive associations between maternal residence near waste sites and central nervous system defects,<sup>8,11–14</sup> heart

defects,<sup>12,13,15–19</sup> surgical correction of gastroschisis and exomphalos,<sup>20</sup> hypospadias and epispadias,<sup>19</sup> and chromosomal anomalies in offspring.<sup>6,8,11,21</sup>

Fewer studies explored the relation between maternal residential proximity to waste sites and adverse pregnancy outcomes other than congenital malformations. Most of these studies reported minimal or no association except between maternal residential proximity to pesticide-contaminated waste sites and fetal deaths,<sup>22</sup> polychlorinated biphenyl (PCB)-contaminated sites and low birth weight among male births,<sup>23</sup> municipal solid waste landfills and low birth weight or small-for-gestational-age births,<sup>24</sup> and any hazardous waste site and low and very low birth weight.<sup>20</sup>

In Europe and Japan, pregnancy outcomes were examined in relation to maternal residential proximity to incinerators or crematoriums. Studies noted associations between these residential characteristics and risk of neural tube defects,<sup>25</sup> heart defects,<sup>25</sup> oral clefts,<sup>26</sup> renal dysplasia,<sup>26</sup> stillbirths,<sup>25</sup> and infant deaths.<sup>27</sup> However, Tango et al.<sup>27</sup> found no higher rate of deaths resulting from congenital malformations among births to mothers who lived near municipal solid waste incinerators in Japan.

In several populations, maternal residential proximity to industrial complexes was associated with increased risk of adverse pregnancy outcomes, including central nervous system defects,<sup>5,10,28</sup> oral clefts,<sup>29</sup> chromosomal anomalies,<sup>6</sup> undescended testis,<sup>30</sup> perinatal mortality,<sup>9</sup> and low birth weight.<sup>31</sup> Vinceti et al.<sup>32</sup> noted that women living in an industrial area contaminated with lead were more likely to give birth to babies with cardiovascular, musculoskeletal,

and oral cleft defects than were women living away from this area.

Increased risks for low birth weight and preterm births were noted among offspring of mothers who resided near highways<sup>7</sup> and in areas with high traffic density,<sup>33</sup> although no statistically increased risk was noted for fetal and early neonatal deaths with this maternal residential characteristic.<sup>34</sup>

Mothers living near cornfields at delivery were more likely to give birth to babies with limb malformations,<sup>35</sup> an association that might be attributed to exposure to pesticides used on this crop. Rull et al.<sup>36</sup> noted elevated risks for neural tube defects among offspring of women who lived within 1000 meters of applications of pesticides classified as amides, benzimidazole, methyl carbamate, organophosphates, benomyl, or methomyl. Bell et al.<sup>37</sup> found elevated risk of fetal deaths from congenital malformations if mothers lived near areas of pesticide application.

We expected that studies that measured actual distances between the environmental hazard of interest and individual maternal residential addresses would have less exposure misclassification. With respect to congenital malformations, we considered studies that included fetal deaths or terminations as well as live births and that examined specific defects instead of all malformations combined as having stronger designs. Eleven of the studies on congenital malformations<sup>6,10,12,13,18,21,29,30,35,38</sup> and 6 of the studies on other adverse reproductive outcomes<sup>7,22,33,34,37,39</sup> met these criteria. In several of these studies, adverse reproductive outcomes were associated with maternal proximity to waste sites,<sup>6,12,13,18,21</sup> industrial sites,<sup>6,10,29,30,37</sup> areas with pesticide

applications,<sup>35,36,37</sup> roadways,<sup>7</sup> and dense traffic.<sup>33</sup>

## CHILDHOOD CANCER

Most published studies of childhood cancer and residential proximity to potential environmental hazards focused on leukemia, brain cancer, or all childhood cancers combined. We list the characteristics, findings, and study limitations of the 25 studies reviewed in Table 2.

Residential proximity to roadways and other indices of increased exposure to traffic-related pollution were associated with increased risk of childhood leukemia in a number of European studies<sup>43,44</sup> but were not noted in several US study populations<sup>50,54,55,62</sup> or in a Danish population.<sup>52</sup> Crosignani et al.<sup>43</sup> estimated traffic-related benzene emissions by means of a Gaussian diffusion model and observed an odds ratio (OR) of 3.91 (95% confidence interval [CI] = 1.36, 11.27) for childhood leukemia with benzene concentrations higher than 10 micrograms per cubic meter. Residential proximity to roadways or traffic emissions was also associated with Hodgkin's lymphoma in Danish children.<sup>52</sup>

Risk of childhood cancer was examined in relation to residential proximity to cropland and pesticide applications in US populations in California<sup>53,56,57</sup> and Texas.<sup>41</sup> A birth address within 1000 meters of cropland showed some association with germ-cell tumors, non-Hodgkin's lymphoma, and Burkitt lymphoma, although the elevated ORs were based on a small number of cases.<sup>41</sup> Using an exposure metric that consisted of residential proximity within 0.5 mile of pesticide applications (pounds per square mile), Reynolds et al.<sup>56</sup> noted

**TABLE 1—Studies of Residential Proximity to Environmental Hazards and Adverse Pregnancy Outcomes With Reported Disparities by Race and Ethnicity or Socioeconomic Status**

First Author, Year, and Country	Population	Pregnancy Outcomes	Disparities Examined	Environmental Hazard and Disparities
Bentov, 2006, <sup>5</sup> Israel	Beer-Sheva subdistrict, 1995–2000	Major congenital malformations combined and subcategorized into major congenital anomalies of central nervous system, chromosomal anomalies, and other major congenital malformations	Jewish populations (urban, urban satellite, and agricultural localities); Bedouin population (permanent localities and traditional tribal settlements)	Residential proximity to a regional industrial park was associated with increased rates of major congenital anomalies among the Bedouin population but not among the Jewish population.
Brender, 2008, <sup>6</sup> Texas	Live births and fetal deaths, 1996–2000	Chromosomal anomalies combined and categorized into 9 categories	Race or ethnicity (non-Hispanic White, Hispanic, African American, other)	Hispanic women who lived near hazardous waste sites were 7.9 times as likely (95% CI = 1.1, 42.4) to have offspring with Klinefelter variants.
Genereux, 2007, <sup>7</sup> Canada	All live singleton births, Montreal, 1997–2001	Preterm birth, low birth weight, and SGA birth	Maternal education (< 11 y, 11 y, 12–13 y, > 13 y); census tracts ranked into quintiles according to neighborhood poverty level	Proximity to highways associated with OR = 1.58 for preterm birth, OR = 1.81 for low birth weight, and OR = 1.32 for SGA births among women living in the most wealthy neighborhoods but not associated with these outcomes in less wealthy or poor areas; this residential characteristic was associated with preterm birth and low birth weight births in the most highly educated women but not in the less educated women.
Orr, 2002, <sup>8</sup> California	Live births and fetal deaths, 1983–1988	All congenital malformations combined and subcategorized into 9 defects or defect groups	Race and ethnicity (Hispanic or Latino, Black or African American, American Indian or Alaska Native, Asian and Pacific Islander)	Although the numbers of exposed cases and controls were small, the strongest association noted among American Indians and Alaska Natives was between a maternal residence in a census tract with 1 or more National Priority List hazardous waste sites and birth defects.
Sarov, 2008, <sup>9</sup> Israel	Beersheba subdistrict, 1995–2000	Perinatal mortality (fetal deaths, intrapartum death, and postpartum death within 28 d after delivery)	Stratified by ethnicity (Jews and Bedouins) and by type of locality	Residential proximity to an industrial park was associated with increased rates of perinatal mortality among Bedouins but not among Jews.
Suarez, 2007, <sup>10</sup> Texas	Live births and fetal deaths, 1996–2000	Neural tube defects	Ethnicity (non-Hispanic White, Hispanic)	Maternal residential proximity (< 1 mi) to 1 or more TRI industrial facilities associated with neural tube defects in births to White, non-Hispanic mothers (OR = 1.8; 95% CI = 1.1, 2.8) but not with births to Hispanic mothers (OR = 1.1; 95% CI = 0.8, 1.4)

Note. CI = confidence interval; OR = odds ratio; SGA = small for gestational age; TRI = Toxic Release Inventory.

slightly elevated ORs for leukemia with birth addresses near application of pesticides that were probable or possible carcinogens and nearby applications of organochlorine or organophosphate pesticides. Risk of acute lymphoblastic leukemia was elevated in children who lived within 0.5 mile (lifetime residences) of applications of organophosphates, chlorinated phenols, and triazines and pesticides classified as insecticides or fumigants<sup>57</sup>; acute lymphoblastic leukemia risk was associated with moderate but not high exposures.

Risk of childhood cancer was examined in relation to residential proximity to other sources of contaminants, including industries reporting under the US Toxic Release Inventory, petrochemical plants, gas stations, repair garages, nuclear power plants, and landfill sites and hazardous waste sites. Increased risk of childhood leukemia was found with residential addresses near gas stations,<sup>44,60,63</sup> repair garages,<sup>60</sup> and nuclear power plants.<sup>46,59</sup> Children whose mothers lived near industries covered under the Toxic Release Inventory during pregnancy were more likely to have brain cancer, especially if the mother lived within 1 mile of a facility with carcinogen emissions.<sup>42</sup>

We considered studies that examined incident cases of childhood cancer in relation to actual measured distances between 1 or more residences documented before diagnosis and the environmental hazard of interest to be less subject to exposure misclassification and selection bias. Of the reviewed studies, 10 met these criteria.<sup>41,42,50,52,55–57,61,62,64</sup> Residential addresses near cropland,<sup>41</sup> pesticide application,<sup>56,57</sup> and industries<sup>42,61</sup> and their associated emissions<sup>64</sup> were associated with

childhood cancer in several of these studies.

### CARDIOVASCULAR, RESPIRATORY, AND OTHER DISEASES

We conducted a comprehensive review of the literature to examine the relation between residential proximity to a wide range of environmental hazards and cardiovascular and respiratory disease, PCB toxicity, end-stage renal disease, diabetes, and adult cancers such as leukemia and non-Hodgkin's lymphoma. Online Supplemental Table 2 (available as a supplement to the online version of this article at <http://www.ajph.org>) summarizes the 20 research studies reviewed in this section, including limitations.

The results of 12 studies on respiratory illness suggested that residential proximity to both stationary sources of air pollution (industries covered under the Toxic Release Inventory, National Emission Inventory, hazardous air pollutants, petroleum refineries, etc.), and, with a few exceptions, heavily trafficked roads, was significantly associated with asthma hospitalizations.<sup>65–69</sup> An example is the case-crossover study by Smargiassi et al.,<sup>69</sup> which examined the effects of residential proximity to point-source air pollution on asthma among children. They collected asthma hospitalization data for children aged 2 to 4 years and calculated the risk of asthma episodes for residential postal codes for the east end of Montreal Island. Exposure was estimated using the American Meteorological Society–Environmental Protection Agency Regulatory Model, an air dispersion model, and sulfur dioxide emissions data from 2 petroleum

refineries and other point sources measured via 2 fixed air-monitoring sites. The model computed estimations of daily sulfur dioxide exposure at the centroid of each postal code as well as average hourly predictions and daily peaks. Smargiassi et al.<sup>69</sup> used logistic regression to evaluate sulfur dioxide exposure in relation to asthma hospitalization days versus control days using a time-stratified approach. Results revealed that short-term increases in sulfur dioxide were significantly associated with a higher number of asthma-related emergency department visits and hospital admissions in children residing near refineries.

In addition, several studies found that exposure to mobile sources of air pollution through residential proximity to major roadways increased the occurrence of chronic respiratory symptoms by exacerbating asthma or increasing self-reported chronic respiratory symptoms such as wheezing, pulmonary function, attacks, and the use of respiratory medicine.<sup>70–73</sup> Three other studies did not find a significant relationship between proximity to roads and asthma hospitalizations among children,<sup>74–76</sup> although English et al.<sup>74</sup> reported that the odds of residing in high traffic-flow areas were significantly higher for children experiencing more than 1 asthma hospitalization per year than for children having only 1 incident. Three studies on cardiovascular disease<sup>65,77,78</sup> also suggested a significant association between residential exposure to combined sources of air pollution and stroke mortality. For instance, Hu et al.<sup>77</sup> determined the observed and expected stroke mortality at the census tract–level for counties in northwest Florida. Air pollution was characterized by the presence of Toxic Release

Inventory sites and other stationary sources of air pollution (i.e., dry cleaners, sewage treatment plants, solid waste disposal, and Superfund sites), and roads with high average vehicle traffic counts. Using location, Hu et al. calculated air pollution density surfaces for point sources and traffic and used hierarchical logistic regression. The mean age-adjusted stroke rate in the study areas was more than 8 times the expected rate, and census tracts with high levels of air pollution had significantly elevated risks of stroke mortality. Maheswaran and Elliott<sup>78</sup> also looked at relationship between stroke mortality and residential proximity to main roads at the census enumeration district level in England and Wales. They evaluated mobile sources of air pollution by using road network data that characterized exposure as distance categories from the centroid of each census enumeration district to the nearest main road. Logistic regression controlling for age, gender, socioeconomic deprivation, and urbanization determined the associations between stroke mortality and distance categories. Census enumeration districts with distances of less than 200 meters to main roads had significantly higher stroke mortality rates than those with distances of more than 1000 meters. This association held when stratified by gender, and Maheswaran and Elliott determined a significant dose–response relationship for distance categories. Three studies using geographic information science examined the impact of residing near hazardous wastes sites, although the health outcomes of interest were different: cord blood PCB level, end-stage renal disease, and diabetes. Kouznetsova et al.<sup>79</sup> conducted an ecological study to determine

**TABLE 2—Studies of Residential Proximity to Potential Environmental Hazards and Childhood Cancer**

First Author, Year, and Country	Study Design, Regional Description	Health Outcomes Included	Environmental Indicator and Exposure Description	Findings and Limitations
Carozza, 2008 United States <sup>40</sup>	Ecological study; US cancer cases in children aged 0–14 y diagnosed between 1995 and 2001 and reported to member registries of the NMACCR	All cancers combined and specific cancers diagnosed among children aged 0–14 y	Cropland: Percentage of cropland for each county, based on 1997 US Census of Agriculture; divided into <20% cropland (referent); 20%–<60% (medium), and 60% or more (high); also examined 6 leading US crops	Findings <sup>a</sup> : All cancers combined showed no association with percentage of cropland in counties; incidence rates of several specific cancers showed an association with medium or high levels of agricultural activity; risk estimates for childhood cancer varied by type of crop grown, with elevated risks noted in counties with corn, oats, and soybeans. Limitations: Potential for ecological fallacy, use of county of residence at time of diagnosis, potential for residual confounding
Carozza, 2009, United States <sup>41</sup>	Population-based case-control study; Texas childhood cancer cases and controls born 1990–1998	Childhood cancers reported among children aged <15 y to the Texas Cancer Registry	Cropland: Fields identified from digital orthophoto quadrangle data and Field Mass Index created to incorporate land area (cropland) and distance to each field from birth residence listed on birth certificate	Findings <sup>a</sup> : No association between a birth residence ≤1000 m of agricultural land use and all cancers combined. A birth residence near cropland showed some association with germ-cell tumors, non-Hodgkin lymphoma, and Burkitt lymphoma, but ORs based on few cases. Limitations: Small number of exposed cases and potential for residual confounding
Choi, 2006, United States <sup>42</sup>	Population-based case-control study; cases aged <10 y at time of diagnosis during 1993–1997 and residents of FL, NJ, NY (excluding New York City), or PA at diagnosis	Incident cases of primary brain cancer	TRI facilities & emissions: residential proximity to TRI during pregnancy (≤1 or ≤2 mi), whether carcinogens were emitted, and a comparative ranking system for TRI releases that combined toxicity information and total mass of release	Findings <sup>a</sup> : Increased risk of brain cancer among children aged <5 y at diagnosis observed for mothers living ≤1 mi of a TRI facility (OR = 1.66; 95% CI = 1.11, 2.48) and living ≤1 mi of a facility releasing carcinogens (OR = 1.72; 95% CI = 1.05, 2.82) Limitations: Quality of exposure data, potential for residual confounding from parental occupational exposures.
Crosignani, 2004, Italy <sup>43</sup>	Population-based case-control study; Varese Province	Childhood leukemia cases diagnosed 1978–1997	Roads and traffic density: Exposure distances of childhood addresses at time of diagnosis from highly trafficked roads; traffic densities in surrounding area; estimation of benzene concentrations with Gaussian diffusion model	Findings <sup>a</sup> : Relative to children whose homes were not exposed to road traffic emissions (<0.1 ug/m <sup>3</sup> benzene as estimated by the model), risk of leukemia with benzene concentration >10 ug/m <sup>3</sup> (OR = 3.91; 95% CI = 1.36, 11.27)

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TABLE 2—Continued

<p>Harrison, 1999, United Kingdom<sup>44</sup></p>	<p>Population-based case-control and retrospective cohort study designs, West Midlands</p>	<p>Childhood leukemia cases diagnosed between 1990 and 1994</p>	<p>Gas stations and roads: Exposure defined as an address at the time of diagnosis <math>\leq 100</math> m from gas station or a zone <math>\leq 100</math> m from a main road</p>	<p>Limitations: Potential for residual confounding from unmeasured confounders (parental occupation) and imperfect measurement (socioeconomic status assigned on the basis of municipality of residence), used addresses at time of diagnosis to assign exposure Findings<sup>a</sup>: From case-control study, ORs = 1.61 (95% CI = 0.90, 2.87) and 1.99 (95% CI = 0.73, 5.43) for living <math>\leq 100</math> m of a main road or gas station, respectively; incidence ratios from cohort analysis = 1.16 (95% CI = 0.74, 1.72) for proximity to roads and 1.48 (95% CI = 0.65, 2.93) for proximity to gas stations. Limitations: Risk estimates not adjusted for age or gender and addresses at time of diagnosis used to assign exposure.</p>
<p>Jarup, 2002, Great Britain<sup>45</sup></p>	<p>Ecological study that included cancer cases diagnosed 1983–1997</p>	<p>Childhood and adult leukemia; adult bladder cancer, brain cancer, and hepatobiliary cancer</p>	<p>Landfill sites: Constructed 2-km buffer zones around 9565 landfill sites using GIS techniques; Postal codes lying outside 2-km buffer were the referent areas.</p>	<p>Findings: With rate ratios adjusted for age, gender, and year of diagnosis, no excess of any cancer was found in relation to living <math>\leq 2</math>-mi buffer of landfills. Limitations: Potential for exposure misclassification to chemicals in landfills and potential for ecological fallacy</p>
<p>Kaatsch, 2008, Germany<sup>46</sup></p>	<p>Population-based case-control study of 41 counties in the vicinity of 16 power plant sites in western Germany</p>	<p>Leukemia and other cancers that were diagnosed in children aged <math>&lt; 5</math> y</p>	<p>Nuclear plants: Distance of residence at the time of diagnosis from the chimney of the nearest nuclear plant; residential proximity <math>\leq 5</math> km and <math>\leq 10</math> km</p>	<p>Findings<sup>a</sup>: For all leukemia cases combined, a dose-response effect was noted in which cases lived closer to sites than controls; residential proximity <math>\leq 5</math> km was associated with an OR = 2.19 (lower 95% confidence level = 1.51). Limitations: Potential selection bias resulting from differential response rates between cases and controls and between those who lived within 5 km of and outside the buffer zone; potential residual confounding: used address at time of diagnosis to assign exposure</p>

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TABLE 2—Continued

Knox, 2000, Great Britain <sup>47</sup>	Migration study of 4385 children who died from cancer before age 16, 1953–1980	Tumors were classified into 11 groups.	Incinerators and landfill sites: Migration asymmetries of birth and death addresses and proximity of these addresses to municipal and hospital waste incinerators and landfill sites	Findings <sup>a</sup> : No systematic migration asymmetries noted for landfill sites; highly significant number of migrations away from birthplaces close to municipal and hospital incinerators  Limitations: No external control group or control for demographic characteristics or proximity to other environmental hazards; deaths instead of incident cases were used.
Knox, 2006, Great Britain <sup>48</sup>	Migration study of 5663 children who died from cancer before age 16, 1953–1980	Tumors were classified into 10 diagnostic subtypes.	Roads and other transportation-related entities: Examined birth and death addresses linked to location of railway stations, bus stations, ferry terminals, railways, roads, canals, and rivers and migration asymmetries of birth and death addresses.	Findings <sup>a</sup> : Significant migration asymmetries (close residential proximity at birth but not at death) noted for residential proximity to bus stations, railway stations, ferries, railways, and roads.  Limitations: No external control group or control for demographic characteristics or proximity to other environmental hazards; used cancer deaths instead of incident cases.
Knox, 1997, Great Britain <sup>49</sup>	Retrospective cohort study with 22 448 addresses at death (and available birth addresses) of children aged 0–15 y who died of leukemia and other cancers	Deaths from leukemia and other childhood cancers	Industries and transportation-related entities: Radial distances of home address postal codes (birth and death) from potential hazards, including industries, railway lines, motorways, airfields, and harbors	Findings <sup>a</sup> : Relative excesses of leukemia and solid tumors were found with residences close to a variety of industries and airfields, railways, motorways, and harbors; hazard proximities for birth addresses were stronger than for death addresses; among children who moved between birth and death, the proximity effect was limited to birth addresses.  Limitations: Population at risk not enumerated at various distances, no adjustment for age or gender, analyzed cancer deaths instead of incident cases
Langholz, 2002, United States <sup>50</sup>	Population-based case-control study in Los Angeles County, CA	Incident cases of childhood leukemia diagnosed during 1978–1984 among children aged 0–10 y	Traffic density. Integrated distance-weighted traffic density was computed for the residence of longest duration.	Findings: Although unadjusted ORs of the relation between quintile of traffic density and risk of leukemia suggested a linear trend, this trend was confounded by wire code.

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TABLE 2—Continued

<p>Liu, 2008, Taiwan<sup>51</sup></p>	<p>Population-based case-control study of 226 Taiwan municipalities</p>	<p>Brain cancer deaths from Bureau of Vital Statistics occurring in people aged 0–29 y; controls included deaths from all causes other than cancer and diseases with respiratory complications.</p>	<p>Petrochemical industry: Proportion of municipality's total population employed in petrochemical industry was used as indicator of petrochemical air emissions at residence at death.</p>	<p>Limitations: Potential exposure misclassification because traffic counts were obtained for 1990–1994, whereas cases were diagnosed during 1978–1984. Findings<sup>a</sup>: With the petrochemical indicator variable divided into tertiles, people in the highest tertile (with the lowest tertile as referent) had a significantly higher OR for death from brain cancer (OR = 1.65; 95% CI = 1.0, 2.73) with adjustment for age, gender, urbanization level of residence, and nonpetrochemical air pollution (<i>P</i> trend &lt; .01). Limitations: Used brain cancer deaths instead of incident cases; not clear whether data were available to distinguish primary from metastatic brain cancers; exposure metric based on municipality instead of individual distance from industry, thereby introducing exposure misclassification; death address may not be relevant for cases who changed residences between birth and death. Findings<sup>a</sup>: Risks of leukemia, CNS tumors, and all selected cancers combined not linked to exposure to benzene or nitrogen dioxide estimates; risk of Hodgkin's lymphoma associated with highest categories of exposure to benzene (OR = 4.3; 95% CI = 1.5, 12.4) and nitrogen dioxide (OR = 6.7; 95% CI = 1.7, 26.0). Limitations: Minimal; parental occupation not taken into account in the analyses Findings<sup>a</sup>: For all cancers combined, the RR for block groups with high propargite usage was 1.25 (95% CI included 1.0); with leukemia, the RR associated with propargite usage was 1.48 (95% CI = 1.03, 2.13); no association noted between usage density of pesticides classified as probable carcinogens ≥ 90th percentile and all types of childhood cancer combined (RR = 0.95, 95% CI = 0.80, 1.13)</p>
<p>Raaschou-Nielsen, 2001 Denmark<sup>52</sup></p>	<p>Population-based case-control study of Danish children aged &lt; 15 y diagnosed with selected cancers 1968–1991 and control children</p>	<p>Leukemia, tumors of the CNS, malignant lymphoma diagnosed in children aged &lt; 15 y</p>	<p>Air concentrations of benzene and nitrogen dioxide: Using Operational Street Pollution Use Model, linked addresses from 9 mo before birth to diagnosis of cancer or similar date for the matched controls to average concentrations of benzene and nitrogen dioxide at the dwelling's front door.</p>	<p>Limitations: Minimal; parental occupation not taken into account in the analyses Findings<sup>a</sup>: For all cancers combined, the RR for block groups with high propargite usage was 1.25 (95% CI included 1.0); with leukemia, the RR associated with propargite usage was 1.48 (95% CI = 1.03, 2.13); no association noted between usage density of pesticides classified as probable carcinogens ≥ 90th percentile and all types of childhood cancer combined (RR = 0.95, 95% CI = 0.80, 1.13)</p>
<p>Reynolds, 2002, United States<sup>53</sup></p>	<p>Ecological study using 1988–1994 childhood cancer incidence rates in CA</p>	<p>Cases of invasive cancer diagnosed in children aged &lt; 15 y during 1988–1994</p>	<p>Pesticide use in adjacent land area: Assigned census block groups to case residences at diagnosis; for each block group, estimated pesticide use density in lb/m<sup>2</sup> for 4 toxicologic groups, 4 chemical classes, and 7 individual pesticides.</p>	<p>Limitations: Minimal; parental occupation not taken into account in the analyses Findings<sup>a</sup>: For all cancers combined, the RR for block groups with high propargite usage was 1.25 (95% CI included 1.0); with leukemia, the RR associated with propargite usage was 1.48 (95% CI = 1.03, 2.13); no association noted between usage density of pesticides classified as probable carcinogens ≥ 90th percentile and all types of childhood cancer combined (RR = 0.95, 95% CI = 0.80, 1.13)</p>

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TABLE 2—Continued

Reynolds, 2002, United States <sup>54</sup>	Ecological study using 1988–1994 childhood cancer incidence rates in CA	All childhood cancers combined; leukemia; gliomas (brain cancer) diagnosed in children aged <15 y	Roads and traffic density. Assigned census block groups to case residences at diagnosis and used GIS to match addresses with a road network; estimates developed for vehicle, road, and traffic density; these 3 metrics were correlated with ambient measurements of carbon monoxide, nitrogen dioxide, PM10, benzene, and 1,3-butadiene.	<p>Limitations: Potential for ecological fallacy, potential for residual confounding, and exclusive use of residence at diagnosis to assign exposure</p> <p>Findings<sup>a</sup>: Rate ratios at the 90th percentile of traffic density were 1.08 (95% CI = 0.98, 1.20) for all childhood cancers combined, 1.15 (95% CI = 0.97, 1.37) for leukemia, and 1.14 (95% CI = 0.90, 1.45) for gliomas; minimal or no evidence of rate differences in these cancers in census block groups with high vehicle or road density; results were suggestive of an association between traffic density and Hovigkin's lymphoma, but a dose-response pattern was not observed.</p> <p>Limitations: Potential ecological fallacy, lacked data on potential confounding factors, used residence at diagnosis to assign exposure, which might not be relevant for children who changed residences.</p>
Reynolds, 2004, United States <sup>55</sup>	Population-based case-control study; CA statewide	Childhood cancer combined, leukemias, and CNS tumors diagnosed in children aged <5 y	Roads and traffic density. Case and control maternal residential address at delivery linked to road and traffic density in 500-ft radius of residence.	<p>Findings: For all cancers combined, for highest road density exposure category compared with lowest, OR = 0.87 (95% CI = 0.75, 1.0); for leukemia, OR = 0.80 (95% CI = 0.64, 1.01); and for CNS tumors, OR = 1.03 (95% CI = 0.75, 1.43). Similar ORs were found with traffic density, although for CNS tumors OR = 1.22 (95% CI = 0.87, 1.70)</p> <p>Limitations: Assignment of exposure limited to residence at birth</p>

Continued

TABLE 2—Continued

Reynolds, 2005, United States <sup>56</sup>	Population-based case-control study; CA statewide	Childhood cancer combined, leukemias, and CNS tumors diagnosed in children aged < 5 y	Pesticide use in adjacent land area: Case and control maternal residential addresses at birth linked to pesticides used on land area (lb/mi <sup>2</sup> ) ≤ 0.5 mi of residence.	Findings <sup>a</sup> : No clear risk patterns noted although mildly elevated ORs for leukemia associated with pesticides that were probable and possible carcinogens and with use of organochlorines or organophosphates. Limitations: Small numbers of children exposed to high-use areas, exposure assessment restricted to birth address, potential exposure misclassification
Rull, 2009, United States <sup>57</sup>	Population-based case-control study; selected counties in northern CA	Incident cases of childhood acute lymphoblastic leukemia diagnosed in children aged < 15 y 1995–2002	Pesticide use in adjacent land area: Case and control lifetime and 1st-year-of-life residences linked to pesticides used on land area ≤ 0.5 mi; pesticides categorized by toxicological effects, physicochemical properties, and target pests or uses.	Findings <sup>a</sup> : Increased risk of acute lymphoblastic leukemia with lifetime moderate exposure to pesticide applications of organophosphates, chlorinated phenols, and triazines and with pesticides classified as insecticides and fumigants; elevated risk not consistent with high exposures. Limitations: Small numbers of exposed cases and controls, climatic conditions not considered.
Sharp, 1996, Scotland <sup>58</sup>	Ecological study of populations near 7 nuclear sites, 1968–1993	Incident cases of leukemia and non-Hodgkin's lymphoma in children aged < 15 y	Nuclear sites: For each nuclear site, study zone constructed with a population centroid ≤ 25 km; each nuclear site examined separately; small-area statistical methods used.	Findings: No evidence of general increased incidence of childhood leukemia and non-Hodgkin's lymphoma noted around nuclear sites; only 1 site had appreciably more cases observed than expected (observed to expected ratio = 1.99). Limitations: Minimal information on potential confounding factors, residence at diagnosis used
Spix, 2008, Germany <sup>59</sup>	Population-based case-control study around all 16 major nuclear power plants	Leukemia including specific forms, CNS tumors diagnosed in children aged < 5 y during 1980–2003	Nuclear power plants: Metric of 1/(distance in km) used as measure of proximity; categorical analyses of 5- and 10-km zones versus outer zones.	Findings <sup>a</sup> : Effects modest except for the association between living in the inner 5-km zone and leukemia (OR = 2.19, lower one-sided 95% CI = 1.51).

Continued

TABLE 2—Continued

Steffen, 2004 France <sup>60</sup>	Multicenter, hospital-based, case-control study (4 centers) of newly diagnosed cases during 1995–1999	Acute leukemia in children aged 0–14 y	Roadways, repair garages, and gas stations: History of exposure to hydrocarbons (residential proximity to roadways, car repair garage, gas station) from date of conception to date of diagnosis (cases) or interview (controls); proximity information obtained by in-person interview; also obtained information about parental occupation.	<p>Limitations: Other sources of potential radiation exposure not accounted for, potential unmeasured confounders; used residence at diagnosis to assign exposure.</p> <p>Findings<sup>a</sup>: Association between residential proximity to a gas station or repair garage during childhood and risk of childhood leukemia (OR = 4.0, 95% CI = 1.5, 10.3) was stronger for acute nonlymphocytic leukemia (OR = 7.7, 95% CI = 1.7, 34.3).</p>
Tsai, 2006, United States <sup>61</sup>	Population-based case-control study of residents of CA, FL, NJ, MI, NC, and PA	Wilms's tumor diagnosed in children through age 9 y during 1992–1995	Hazardous waste sites: Maternal and paternal addresses in close proximity to a National Priorities List site during the 2-y period before the child's birth; residential history determined by parental interview.	<p>Limitations: Proximity to hazards ascertained by self-report, which could have introduced recall bias and inflated risk estimates.</p> <p>Findings: OR = 0.35 (95% CI = 0.12, 0.99) for Wilms's tumor with a maternal residence <math>\leq</math> 1 mi of National Priorities List site during pregnancy, OR = 0.39 (95% CI = 0.16, 0.98) with a residence <math>\leq</math> 1 mi of National Priorities List site during 2 y before birth; no association noted for paternal residence.</p>
Von Behren, 2008, United States <sup>62</sup>	Population-based case-control study in northern CA counties	Leukemia diagnosed in children aged < 15 y during 1995–2002	Traffic density: Traffic density $\leq$ 500-ft-radius buffer determined for each address at diagnosis, birth, and lifetime average.	<p>Limitations: Small numbers of exposed cases and controls and potential selection bias, with African Americans more likely to not participate than Whites</p> <p>Findings: OR = 1.17 (95% CI = 0.76, 1.81) for acute lymphocytic leukemia with residential traffic density &gt; 75th percentile (0 traffic density as referent) for residence at diagnosis, OR = 1.11 (95% CI = 0.70, 1.78) for residence at birth, and OR = 1.24 (95% CI = 0.75, 2.08) for average lifetime traffic density.</p> <p>Limitations: Potential selection bias, with control families having higher household incomes than case families</p>

Continued

TABLE 2—Continued

Weng, 2009, Taiwan <sup>63</sup>	Population-based case-control study of all deaths	Leukemia deaths in children aged <15 y, 1996–2006	Gas stations: Gas station density in municipalities in which the residents lived at the time of death	Findings <sup>a</sup> : AOR = 1.91 (95% CI = 1.29, 2.82) for leukemia (death) associated with living in municipalities with the highest gas station density; a significant trend was noted between increasing gas station density and risk of death from childhood leukemia. Limitations: Used deaths instead of incident cases of leukemia; exposure classification restricted to municipality of residence at death; gas station density metric based on 2008 data, which might have introduced exposure misclassification; potential for residual confounding.
Yu, 2006, Taiwan <sup>64</sup>	Population-based case-control study in Kaohsiung, 1997–2003	Incident leukemia diagnosed in people aged <30 y, 1997–2003	Petrochemical plants: Exposure opportunity score assigned, based on residences ≤2 years before birth, that took into account residential mobility, length of stay in each residence, distance to petrochemical plants, monthly prevailing wind direction, and multiple petrochemical pollution sources.	Findings <sup>a</sup> : No overall association noted with acute lymphocytic leukemia in the group aged 0–19 y who had a higher exposure opportunity score (OR = 1.21; 95% CI = 0.89, 1.65); positive association seen between residential petrochemical exposure and leukemia among people aged 20–29 y (1 unit increase in log-transformed exposure score: OR = 1.54; 95% CI = 1.14, 2.09). Limitations: Potential for selection bias because of differential participation between case and control participants.

Note. AOR = adjusted odds ratio; CI = confidence interval; CNS = central nervous system; GIS = geographic information system; NAACCR = North American Association of Central Cancer Registries; OR = odds ratio; PM10 = particulate matter ≤10 μm; RR = relative risk; TRI = Toxic Release Inventory.

<sup>a</sup>Positive associations found between environmental entity and health outcomes under study.

whether exposure to persistent organic pollutants (POPs) found near hazardous waste sites was associated with diabetes risk in New York. They used state hospitalization data to calculate diabetes hospitalization rates in patients aged 25 to 74 years by zip code, controlling for age, gender, race, income, and urban or rural population density. Hazardous waste sites were grouped into exposure categories by zip code: POP sites (dioxins and furans, PCBs, persistent pesticides), non-POP sites (volatile organics and metals, etc.), and clean sites. They found significantly higher diabetes hospitalization rates in POP zip codes versus both clean and non-POP sites. With stratification, the rate ratios were highest for Blacks and older age groups.

Some evidence has linked residential proximity to hazardous waste sites and adverse health impacts,<sup>79–81</sup> but the dearth of literature makes cross-study comparisons difficult. Although exposure to hazardous waste sites may be associated with outcomes such as PCB toxicity, end-stage renal disease, and diabetes, more research is needed.

We reviewed three studies using geographic information science to explore the relationship between environmental burdens and adult cancer. The environmental exposures of interest in these studies included air pollution from industrial plants and a nuclear facility's radioactive emissions; the findings were mixed. Leukemia was significantly associated with proximity to the Pilgrim nuclear power plant among women,<sup>82</sup> and the odds of leukemia increased with proximity to the plant. Wilkinson et al.<sup>83</sup> calculated the observed and expected cancer incidence and death rates for electoral wards using a distance decline model around the

pesticide- and fertilizer-producing Pan Britannica Industries Plant. Although they found significant differences between observed and expected rates of cancer incidence and mortality for distances from 0 to 1 kilometers and 0 to 7.5 kilometers around the plant, evidence of a dose–response relationship was mixed, and the rate ratios were within the range for the region, although the observed incidence of melanoma and cancers of the lung, pancreas, and stomach was significantly higher than expected values. Another study<sup>84</sup> found significant associations between other industrial plants and some cancers; for instance, non-Hodgkin's lymphoma was significantly associated with proximity to a copper smelter and sulfite pulp mill.

Similar to the hazardous waste site studies, studies assessing cancer risk from exposure to various environmental hazards suffered from comparability problems. Residential proximity to certain industrial plants appears to be linked to cancer risk, although the significance of the results depends on the specific type of plant, population subgroup, and cancer types.

We would expect studies that measured the distance between the environmental hazard of interest and individual residential addresses to have fewer exposure misclassifications and therefore stronger designs. Of the 20 studies we considered, 11 met that criterion.<sup>65–68,70,71,74,80–82,84</sup> In 9 of these 11 studies, residential proximity to roads<sup>66,70,74</sup>, to cokeworks, copper smelters, refineries, nuclear power plants, or other stationary point sources of air pollution<sup>65,67,68,82,84</sup>, or to hazardous waste sites<sup>81</sup> were associated with asthma, respiratory illnesses, heart disease, non-Hodgkin's lymphoma,

end-stage renal disease, or adult leukemia.

### LIMITATIONS OF EVIDENCE

Without exception, the studies included in this review had 1 or more limitations that should be considered in the interpretation of results and implications for environmental and public health. Several studies used an ecological approach in which the investigators compared rates of adverse health outcomes with census-defined or administrative boundaries (e.g., zip code) as a proxy for residential location when individual addresses were unknown. This approach, in which exposure is assigned on the basis of whether a specific environmental hazard is present within a particular unit of aggregation<sup>85</sup> (called a container approach or spatial coincidence analysis), implies that all residents within a particular boundary are equally affected by the hazard of interest without accurate assessment of individual exposure.<sup>85</sup> For example, a case participant may live next to a particular hazard of interest, but if the hazard is located outside the researcher's unit of analysis (e.g., zip code), then that case participant would not be defined as exposed. Such a design can lead to an ecological bias in which associations at an aggregate level do not represent exposures at the individual level among people with and without adverse health outcomes.<sup>86</sup> In general, the larger the unit of spatial aggregation is, the more likely it is that bias will be introduced as a result of heterogeneity across and within these units,<sup>85</sup> and ecological fallacy may result.

Distance-based methods greatly improve on this approach by measuring the actual distance

between residential addresses and environmental hazards. However, residential exposure to site contaminants, industrial emissions, traffic emissions, and pesticide applications will also vary by the climatic and topographic characteristics of the geographic area. The distance-based studies we reviewed rarely considered these conditions; the exceptions were those by Crosignani et al.,<sup>43</sup> Goldberg et al.,<sup>24</sup> Maantay et al.,<sup>68</sup> Raaschou-Nielsen et al.,<sup>52</sup> Vinceti et al.,<sup>38,87</sup> Wilhem and Ritz,<sup>33</sup> and Yu et al.<sup>64</sup> Residential proximity to environmental hazards can only serve as a crude proxy for exposure and does not accurately represent individual exposure to ambient conditions or body or target organ dose. However, pollution plume modeling, a method that combines data on chemical emissions and local meteorological conditions to model the environmental fate and dispersion of pollutants, can more accurately predict exposures in the ambient environment.

With respect to residential proximity and adverse pregnancy outcomes, many studies used maternal address at delivery to assign exposure rather than address around conception and during the first trimester. Although address at delivery might be relevant for some adverse pregnancy outcomes, it can be problematic for assigning exposure in studies of chromosomal and nonchromosomal congenital malformations, for which the time around conception and earlier or in the first trimester of pregnancy are, respectively, the most relevant. The estimated percentage of women who change addresses between the time of conception and delivery has ranged between 12% and 33% depending on the maternal population examined.<sup>88–91</sup>

Childhood cancer studies frequently based exposure assignment on 1 address such as address at birth, diagnosis, or death, which potentially introduced exposure misclassification by not accounting for residential mobility. In a California study of childhood leukemia,<sup>92</sup> 65.8% of the study children moved between birth and date of diagnosis, with more than one third of those who moved living in at least 1 residence outside of the county of birth.

Residual confounding can also be an issue; information regarding factors that are related to both exposure and outcome are measured imprecisely or are not available. Residential addresses of mothers, children, and adults are not randomly distributed; rather, they depend on several factors such as socioeconomic status, parental occupation, and race or ethnicity. These same factors may also increase or decrease the likelihood of living near potential environmental hazards. For instance, race and ethnicity,<sup>93–95</sup> maternal education,<sup>95</sup> and maternal occupation<sup>96</sup> have been associated with maternal residential proximity to sources of pollution. These factors are also risk factors for several adverse pregnancy outcomes.

### CONCLUSIONS AND RECOMMENDATIONS

Research using geographic information science and other geospatial techniques to explore the public health burdens of residential proximity to environmental hazards is in its infancy. We investigated the associations between only a few environmental exposures and health outcomes, and our review is not by any means meant to be considered exhaustive. In addition, differences in study design and methodology

may result in a lack of consistency across studies. Although their results are mixed, many studies found significant relationships between residential proximity to environmental hazards and adverse health outcomes, such as adverse pregnancy outcomes (including increased risks for central nervous system defects, congenital heart defects, oral clefts, renal dysplasia, limb malformations, chromosomal anomalies, preterm births, low birth weight, small-for-gestational-age births, fetal deaths, and infant deaths), childhood cancers (including leukemia, brain cancer, germ-cell tumors, non-Hodgkin's lymphoma, and Burkitt lymphoma), asthma hospitalizations and chronic respiratory symptoms, stroke mortality, PCB toxicity, end-stage renal disease, and diabetes. Although populations living close to environmental hazards appear more likely to have adverse health outcomes, proximity does not necessarily equate to individual-level exposure. Few studies have examined whether such exposures are more or less likely to increase risk of adverse health outcomes among minority and lower-income populations. This dearth of studies is possibly the result of a limitation of the available health data, which often do not accurately or completely report race and ethnicity for the health outcome cases.

Given these conclusions, which are based on previous evidence of disparities by race and income in relation to proximity to environmental hazards,<sup>97–100</sup> the adverse health outcomes for populations in close proximity to environmental hazards, and acknowledgment of the health disparities experienced in general by communities of color and lower-income communities, we suggest that government agencies consider these findings in

siting of environmentally burdensome facilities and land uses, in regulatory and enforcement efforts concerning pollution, and in the active promotion of environmental health justice and environmental health protection.

The evidence at this time is sufficient to justify the application of the precautionary principle to protect people from the deleterious effects of living near environmental hazards. Even in the absence of complete scientific proof, enough evidence of potential harm being done exists to justify taking steps to rectify the problem and to protect the public from potentially harmful exposures when all available evidence points to plausible risk. Although economic and political forces will likely require stringent proof that specific recommendations (e.g., establishment of protective buffer zones around noxious land uses) will be effective, some practical applications should be obvious. For example, prohibiting the siting of schools near highways and being cognizant of pesticide drift when planning residential locations or other sensitive land uses, fall into the category of commonsense guidelines and constitute approaches that would be difficult to argue against.

### About the Authors

Jean D. Brender is with the Texas A&M Health Science Center, School of Rural Public Health, College Station, Texas. Juliana A. Maantay is with the Geographic Information Science Program and Urban Geographic Information Science Lab, Department of Environmental, Geographic, and Geological Sciences, Lehman College, City University of New York, Bronx, NY. Jayajit Chakraborty is with the Department of Geography, University of South Florida, Tampa, FL.

Correspondence should be sent to Jean D. Brender, RN, PhD, School of Rural Public Health, 257 SRPH Administration Building, TAMU 1266, Texas A&M Health Science Center, College Station, TX 77843-1266

(e-mail: [jdbrender@srph.tamhsc.edu](mailto:jdbrender@srph.tamhsc.edu)).

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### Contributors

J. D. Brender, J. A. Maantay, and J. Chakraborty conceptualized the analytic review. J. D. Brender and J. A. Maantay completed the systematic reviews of the literature and shared supervision of the article's overall content. J. Chakraborty reviewed the article and contributed to the conclusions, implications, and recommendations.

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This work did not involve human research participants.

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