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ORIGINAL RESEARCH

Marine Microplastic Levels and the Prevalence of Cardiometabolic Diseases in US Coastline Counties

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BACKGROUND: Although micro- and nanoplastics are an emerging risk factor for the development of cardiovascular diseases, the association of marine microplastic levels (MMLs) with the burden of cardiometabolic diseases at a population level remains unexplored. We investigated the relationship between the mean microplastic concentration in ocean water within 200 nautical miles of the US coastline and the prevalence of type 2 diabetes, coronary artery disease, and stroke in nearby counties.

METHODS: Microplastic concentration data within 200 nautical miles of the US coastline were geospatially analyzed to calculate mean MMLs. The coastal counties were stratified into 4 categories based on MMLs in the adjacent ocean water body: low, medium, high, or very high. The Behavioral Risk Factor Surveillance System 2019 to 2020 survey data were used to identify the prevalence of type 2 diabetes, coronary artery disease, and stroke in these counties. Univariate and population-weighted multivariate quasi-Poisson regression, adjusted for county-level confounding variables, was used to evaluate the unadjusted and adjusted associations between disease prevalence and MMLs.

RESULTS: The mean age of the population was 43±6 years. After adjusting for age, sex, physician access, and socioenvironmental vulnerabilities at the county level, counties with very high MMLs exhibited an 18%, 7%, and 9% higher adjusted prevalence of type 2 diabetes, coronary artery disease, and stroke, respectively, compared with those with low levels.

CONCLUSION: Marine microplastic levels were significantly associated with the prevalence of cardiometabolic diseases in coastal counties. Comprehensive studies at an individual level are needed to further investigate the health impacts of microplastic pollution.

Key Words: coastline counties ■ coronary artery disease ■ microplastics ■ stroke ■ type 2 diabetes

Plastics are the most ubiquitous component in daily human life, partly due to their low cost and remarkable versatility. They are predominant materials for a wide range of applications. Plastic waste is often released into the environment directly or through improper reuse or recycling practices. When exposed to environmental conditions, plastic waste degrades into smaller particles known as micro- or nanoplastics (MNPs).[1] Microplastics are particles with a diameter of less than 5 millimeters, whereas nanoplastics are smaller, with diameters ranging from 1 to 100 nanometers.[1]

Beyond the well-established environmental threat associated with plastic-related pollution, there is a need to deepen the understanding of the possible consequences of the widespread use of plastics on

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RESEARCH PERSPECTIVE

What Is New?

- Coastline counties in the United States potentially exposed to higher levels of marine microplastics had a 18%, 7%, and 9% higher prevalence of type 2 diabetes, coronary artery disease, and stroke, even after accounting for county-level confounding factors

What Question Should Be Addressed Next?

- Future studies should focus on delineating and quantifying the routes and modes of microplastic exposure to humans, as well as studying their role as a novel component of exposome with respect to pathogenicity at the individual level

Nonstandard Abbreviations and Acronyms

CVI	Climate Vulnerability Index
MMLs	marine microplastics levels
MNPs	micronano plastics
PR	prevalence ratio
T2D	type 2 diabetes

human health. Although there are already warnings that plasticizers and other plastic-associated chemicals, such as bisphenol A and phthalates, promote adverse health outcomes through their endocrine-disrupting properties and other mechanisms, recent evidence suggests a possible deleterious role of MNPs. MNPs can interact with cellular components and cause damage through various mechanisms, such as oxidative stress, inflammation, apoptosis, and pyroptosis.[2] Recent studies have suggested potential associations between high exposure to MNPs and cardiovascular structural and hemodynamic abnormalities, including abnormal blood velocity, decreased cardiac output, altered heart rate, myocardial fibrosis, and endothelial dysfunction, leading to cardiometabolic disease phenotype.[3–7] Furthermore, emerging evidence indicates that patients with MNPs detected in atheromatous plaques had a higher risk of developing adverse cardiovascular events.[8] How these microvascular and hemodynamic changes translate clinically on an individual and population level remains unknown. Although some studies have suggested a possible association between MNPs and cardiovascular disease, these findings remain inconclusive and require further investigation.

In this study, we aim to address this gap by examining the county-level impact of marine microplastics on cardiometabolic diseases.

METHODS

The data supporting this study's findings will be made available upon reasonable request to the corresponding author.

Analytic Sample Collection and Assimilation

We used the National Centers for Environmental Information's marine microplastics geodatabase to obtain data on microplastic concentration in ocean water settings between January 1, 2015 and December 30, 2020.[9,10] The data were filtered using ArcGIS Online to include geopoints within 200 nautical miles (equivalent to 230 miles) of the US territorial sea margin. These limits correspond to the outer margin of an exclusive economic zone, defined by the United Nations Convention on the Law of Sea as an area of the ocean within which a coastal nation has sovereign rights to explore, exploit, conserve, and manage living and nonliving resources.[11] For our study, we focused on counties with boundaries adjacent to the coastline, identifying them as "coastal counties" or "coastline counties" in our analysis. The list of coastline counties is available from the US Census Bureau resource library.[12] Two layers were created, one consisting of coastal counties and another comprising the geopoints representing microplastic concentration in the ocean water sample within the exclusive economic zones. We used the built-in analysis tools in ArcGIS Online to first calculate the mean of geopoints, followed by the 'Join Nearby' function to assign them to the adjacent coastal counties.

After tabulating the geodata, we stratified coastal counties into 4 categories based on mean MMLs in the adjacent portion of exclusive economic zones: low (0–0.005 pieces/m^3), medium (0.005–1 pieces/m^3), high (1–10 pieces/m^3), and very high (≥10 pieces/m^3). We leveraged the 2022 Centers for Disease Control and Prevention's (Population-Level Analysis and Community Estimates) data set based on the Behavioral Risk Factor Surveillance System survey 2019 to 2020 and ACS (American Community Survey) 2015 to 2019 estimates to obtain the county-level prevalence of type 2 diabetes (T2D), coronary artery disease (CAD), and stroke.[13] We then investigated their association with marine microplastic concentration attributed to the county before and after accounting for potential confounding factors. As publicly available deidentified data were used, institutional review board approval from Lahey Hospital and Medical Center was not required per institutional policy.

County-Level Predictive Factors

Candidate factors considered for inclusion were those with available county-level data from 2015 to 2020 that are known to be associated with T2D, CAD, or stroke and could potentially confound the observed association. We used county-level measures of 3 domains—socioeconomic, infrastructure, and environment—in the baseline vulnerability theme of the Climate Vulnerability Index (CVI), the median age of the population in the respective counties averaged from 2016 to 2019 (from Centers for Disease Control and Prevention's National Environmental Public Health Tracking Network), sex, number of active federal and nonfederal physicians in a county per 10 000 population (from area health resource file 2019–2020) as covariates to adjust for demographics, socioeconomic-environment-infrastructure status, and health resources distribution.[14–16]

The US CVI is a comprehensive tool developed with the support of the Environmental Defense Fund, Texas A&M University, and many other partners. It integrates the climate impact at the census tract and county level with their respective environmental, health, infrastructure, and socioeconomic metrics.[15] The baseline vulnerability theme of the CVI includes factors that could weaken a community's resilience to socioeconomic, environmental, or infrastructure-related stressors. These factors reflect climate change's direct and indirect impacts on communities, incorporating 184 indicators collected between 2017 and 2019.

The baseline social and economic domain leverages the US Centers for Disease Control and Prevention and Agency for Toxic Substances and Disease Registry Social Vulnerability Index, further augmented by indicators such as redlining designations in urban areas, additional vulnerable populations (eg, homeless, veterans), crime and prison statistics, housing characteristics, and presence/lack of nongovernmental organizations. The baseline infrastructure domain incorporates transportation, energy, food, water, waste management, governance, and access to physical, digital, and financial resources. The baseline environmental domain includes indicators that characterize long-standing disparities of environmental exposure stressors and pollution, such as transportation, area and point sources generating air, soil, and water pollution, land use, and environmental health risk metrics. Figure S1 and Table S1 enlist the variables included in each of the CVI domains.

Statistical Analysis

We employed quasi-Poisson regression to examine the association between the county-level prevalence of cardiometabolic diseases—T2D, CAD, and stroke—and the category of MMLs in the surrounding ocean water body assigned to these counties. The quasi-Poisson regression analysis was preferred over linear regression using the absolute concentration of MMLs because of the following reasons: (1) MMLs and the prevalence of T2D, CAD, and stroke were nonnormally distributed across coastal counties (Figures S2.1 and S2.2) and (2) the assumption of constant variance of residuals across fitted values, necessary for fitting a linear regression model was not fulfilled (Figure S3). The differences in population characteristics among Q4 (very high MMLs) and Q1 (low MMLs) counties were estimated using quartile rate ratios computed by fitting a quasi-Poisson regression model with county-assigned MMLs as independent and individual demographic characteristics as a dependent variable (Weighted Quartile Rate Ratio; Table 1). Our analysis comprised three models constructed in order of increasing adjustment for potential confounders: Model 1 was unadjusted and depicts the crude association between MMLs and the prevalence of cardiometabolic diseases. Model 2 was adjusted for the county's median age, sex, and physician access. Model 3 used CVI's baseline socioeconomic, infrastructure, and environmental vulnerability domains as covariates in addition to the variables adjusted for in Model 2. Model 4 additionally adjusted for the proportion of self-identified Black and Hispanic individuals residing in the coastal counties, obtained from the ACS 2015 to 2019 5-year estimates. The baseline vulnerability health domain was not used due to the overlap of its components with the outcome variables. The quasi-Poisson method was used to account for overdispersion in the observed data. Climate change risks and their related domains were not used as covariates because some of the subdomains involved in its calculation are based on future predictions and do not fall within the study's timeline. Additionally, the models were weighted for the total population estimate of each county based on the 2016 to 2020 ACS. These adjustments were made to understand the independent association between microplastic concentration and disease prevalence. Statistical analysis was carried out using the R version 4.3.3, using packages 'tidyverse,' 'gtsummary,' 'AER,' and 'jtools' (R Foundation for Statistical Computing, Vienna, Austria. https://www.R-project.org/). Statistical analysis was conducted from June 2024 to July 2024.

Sensitivity Analysis

We conducted a sensitivity analysis using multivariable linear regression with heteroscedasticity-consistent robust SEs, adjusting for the same covariates as Model 4 discussed previously. To ensure comparability, we standardized MMLs using *Z* score normalization (mean = 0, SD = 1). Robust SEs were estimated using the Huber–White sandwich variance estimator from the 'sandwich' package in R version 4.3.3.

Furthermore, to assess the robustness of our results and account for regional differences, we modeled cardiometabolic disease prevalence as a function of median age, sex, race, ethnicity, and social vulnerability index in coastal counties with very high MMLs and their adjacent noncoastal counties. The adjusted prevalence rates were then compared between coastal counties with very high MMLs and most adjacent noncoastal counties using Welch's 2-sample *t* test.

RESULTS

Baseline Characteristics

The baseline characteristics are presented in Table 1. In our analysis of 152 coastline counties, the mean age in the counties was 43±6years. The mean prevalence of T2D, CAD, and stroke was 11.7%, 7.5% and 3.6%, respectively in the coastal counties. Out of the 152 coastal counties included in our study, MMLs were categorized as low, medium, high, and very high in 35, 66, 21, and 30 counties, respectively. Within our study period and geographical inclusion criteria, the mean ocean surface MMLs along the Atlantic coast were estimated to be 122.057 pieces/m^3, whereas that along the Pacific coast was estimated to be 0.003 pieces/m^3. The mean prevalence of T2D, CAD, and stroke was higher in the counties located along the Gulf of Mexico (T2D: 12.89%, CAD: 8.83%, and stroke: 4.15%) compared with those along the Atlantic Coast (T2D: 11.88%, CAD: 7.31%, and stroke: 3.54%) and the Pacific Coast (T2D: 10.62%, CAD: 6.87%, and stroke: 3.23%) (Figure S4).

The mean Climate Vulnerability Index percentile rank was 42nd, with an SD of 25. The number of active federal and nonfederal physicians per 10000 people in counties ranged from 0 to 150, with a mean of 23 (SD: 20) physicians per 10000 population.

The mean age of people living in counties with very high MML was higher than those in low MML counties (quartile rate ratio Q4/Q1: 1.17 [1.13–1.22]). A greater proportion of Black adults resided in coastline counties with very high MML compared with coastline counties with low MML (quartile rate ratio Q4/Q1: 2.39 [1.74–3.31]).

Table 1. Demographic Characteristics of the Selected Coastal Counties Stratified by Marine Microplastic Levels

Characteristics*	Overall	Microplastics exposure				Quartile rate ratio (Q4/Q1)
		Low, Q1	Medium, Q2	High, Q3	Very high, Q4	
Total population	75992609	25338241	19680030	15951505	15022833	0.69 (0.30–1.50)
Male sex, %	49.6	50.1	49.4	48.6	49.9	0.98 (0.97–0.99)
Female sex, %	50.4	49.9	50.6	51.4	50.1	1.02 (1.01–1.03)
Age						
Mean age, y	43.1	42.4	42.6	41.2	46.5	1.17 (1.13–1.22)
18–44y, %	32.6	33.1	32.7	35.0	29.8	0.84 (0.80–0.89)
45–64y, %	27.3	26.7	27.5	27.7	27.5	1.03 (0.99–1.07)
≥65y, %	20.1	19.9	19.2	17.4	24.1	1.21 (1.05–1.40)
Race						
White, %	74.5	79.8	69.0	73.3	81.3	1.20 (1.09–1.32)
Black, %	13.2	6.2	17.9	11.0	12.5	2.39 (1.74–3.31)
American Indian and Alaska Native, %	0.6	1.4	0.5	0.4	0.3	0.34 (0.23–0.50)
Asian, %	4.4	4.3	5.3	5.5	1.8	0.21 (0.12–0.33)
Native Hawaiian and Other Pacific Islander, %	0.1	0.3	0.1	0.0	0.1	0.15 (0.07–0.28)
Other†, %	7.1	8.0	7.2	9.8	4.0	0.32 (0.23–0.43)
Ethnicity						
Hispanic or Latino, %	12.7	12.9	11.1	14.9	14.7	0.98 (0.92–1.05)
Not Hispanic or Latino, %	87.3	87.1	88.9	85.1	85.4	1.07 (0.97–1.18)
Physician access (physicians per 10000 population)	23	20	20	41	19	0.89 (0.66–1.18)
Climate Vulnerability Index percentile	42	35	44	34	50	1.38 (1.10–1.71)
Prevalence of type 2 diabetes, %	11.7	11.2	12.1	9.7	13.0	1.27 (1.19–1.35)
Prevalence of coronary artery disease, %	7.5	7.4	7.2	6.2	8.9	1.39 (1.29–1.49)
Prevalence of stroke, %	3.6	3.5	3.5	2.9	4.2	1.33 (1.24–1.42)

*All except total population expressed as mean.
†Other refers to the individuals who did not self-identify by any of the racial categories above.

Association Between Microplastic Concentration and Prevalence of Type 2 Diabetes

Counties with low MMLs had a significantly lower mean prevalence of T2D (11 200 cases per 100 000 people; SD: 1700) than those with very high MMLs (13 000 cases per 100 000 people; SD: 1300; $P<0.001$ using 1-way ANOVA; Figures 1 and 2). The unadjusted quasi-Poisson regression analysis revealed a significant association between very high microplastic concentration and the prevalence of T2D. Specifically, counties with very high microplastic concentrations had a 1.27 times higher prevalence of T2D compared with counties with low microplastic concentrations (prevalence ratio [PR], 1.27 [1.19–1.35], $P<0.001$). A positive trend in association with diabetes was observed in counties potentially exposed to moderate and high MMLs (Figure 3).

Model 2, adjusted for age, sex, and physician access, showed that counties with very high microplastic concentrations had a 1.19 times higher prevalence of T2D compared with counties with low microplastic concentrations (PR, 1.19 [1.11–1.26], $P<0.001$) (Table 2). Model 3, adjusted for baseline socioeconomic, infrastructure, and environmental vulnerability of CVI, in addition to the variables adjusted for in Model 2, also showed a 1.18 times higher prevalence of T2D in counties with very high marine microplastics concentrations compared with those with low (PR, 1.18 [1.13–1.23], $P<0.001$).

Association Between Microplastic Concentration and CAD Prevalence

Counties with low MMLs had a significantly lower mean prevalence of CAD (7400 cases per 100 000 people; SD: 1500) compared with those with very high MMLs (8900 cases per 100 000 people; SD: 1500; $P<0.001$ using 1-way ANOVA; Figures 1 and 2). Unadjusted analysis revealed a significant association between very high microplastic concentration and

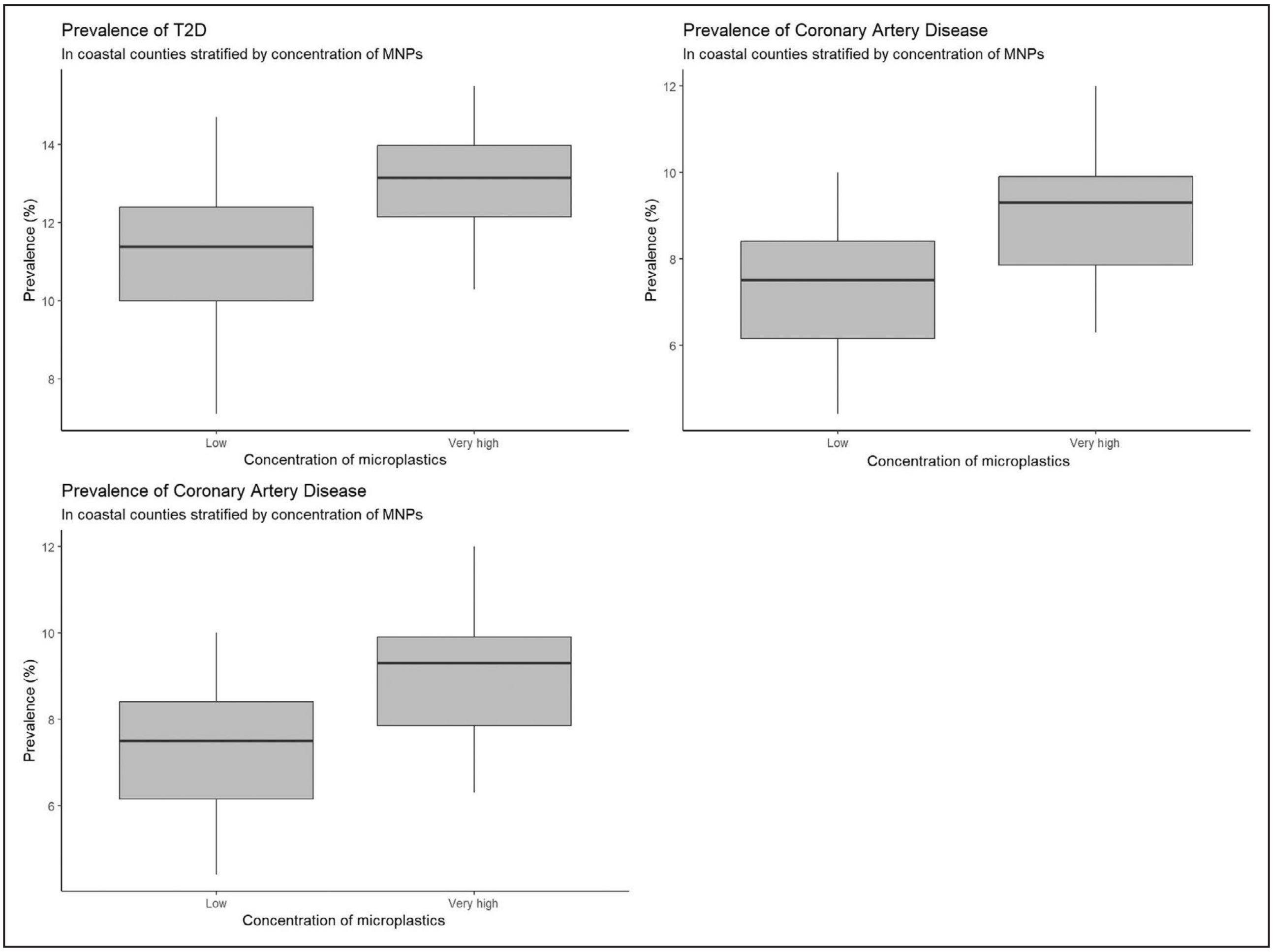

Figure 1. Boxplots showing the prevalence of T2D, CAD, and stroke in counties potentially exposed to very high MMLs in the adjacent ocean water bodies compared with low MMLs.
CAD indicates coronary artery disease; MMLs, marine microplastic levels; MNPs, micronano plastics; and T2D, type 2 diabetes.

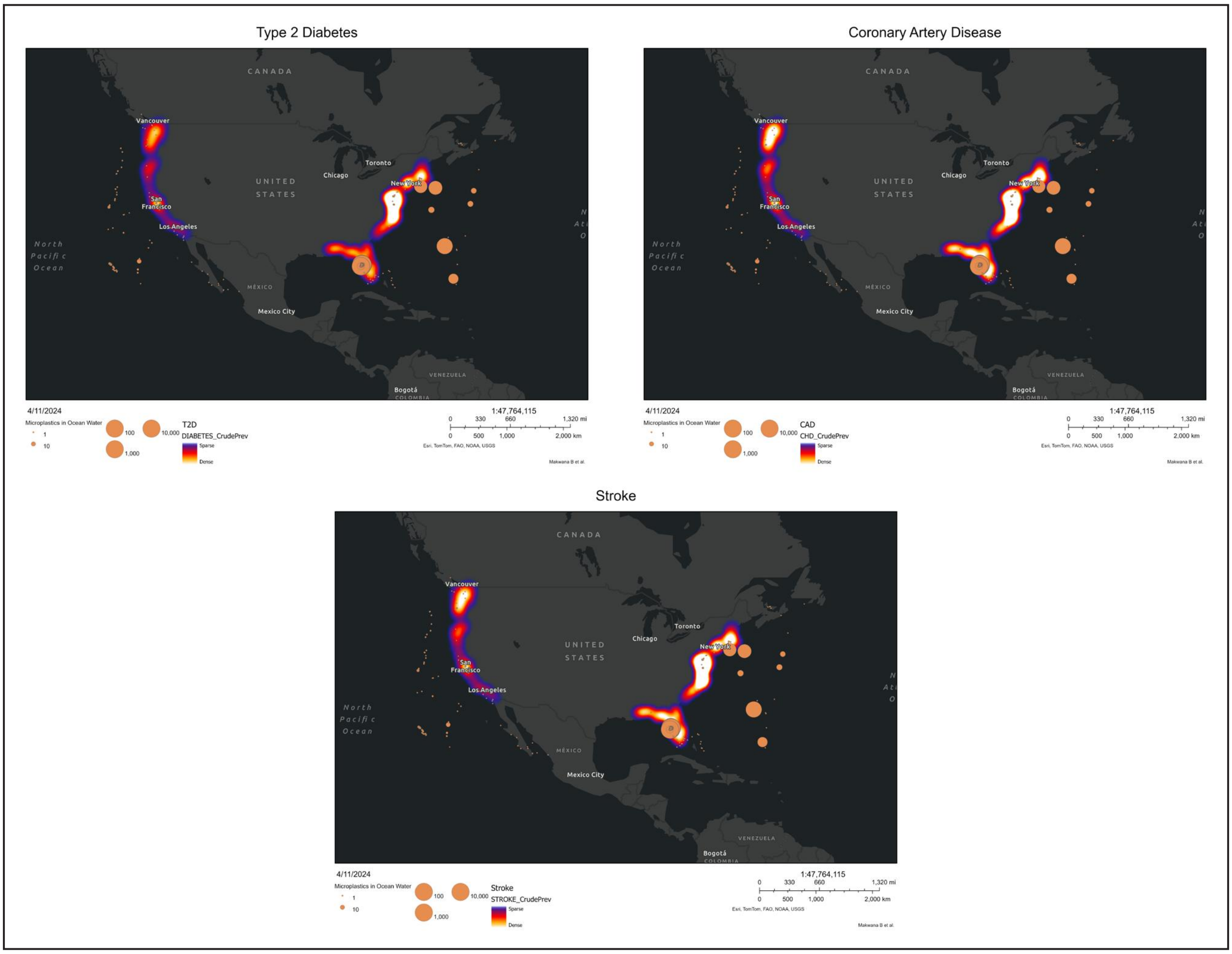

Figure 2. Choropleth maps showing the prevalence of T2D, CAD, and stroke, as well as the marine microplastic levels in the adjacent ocean water bodies.

The fluorescent areas along the coastline represent the prevalence of a particular disease, with higher fluorescence corresponding to a higher prevalence. The diameter of the bubble in the ocean is proportional to the absolute concentration of microplastic detected in the sample of ocean water collected at that site between 2015 and 2020. CAD indicates coronary artery disease; and T2D, type 2 diabetes.

coronary artery disease prevalence (PR, 1.39 [1.29–1.49], *P*<0.001). This association remained significant in Model 2 (PR, 1.11 [1.05–1.17], *P*<0.001) and Model 3 (PR, 1.07 [1.03–1.11], *P*<0.001) (Table 2).

Association Between Microplastic Concentration and Prevalence of Stroke

Counties with low MMLs had a significantly lower mean prevalence of stroke (3500 cases per 100000 people; SD: 700) compared with those with very high MMLs (4200 cases per 100000 people; SD: 700; *P*<0.001 using 1-way ANOVA; Figures 1 and 2). The univariate quasi-Poisson regression analysis revealed a significant association between counties with very high microplastic concentration and the prevalence of stroke. Counties with very high MMLs had 1.33 times higher prevalence of stroke than counties with low MMLs (PR, 1.33 [1.24–1.42], *P*<0.001).

Our adjusted models replicated these results qualitatively. Model 2 and Model 3 showed that counties with very high MMLs had 1.12 (PR, 1.12 [1.05–1.19], *P*<0.001) and 1.09 (PR, 1.09 [1.05–1.13], *P*<0.001) times the prevalence of stroke compared with counties with low MMLs, respectively. (Table 2).

Counties potentially exposed to moderate MMLs had a significantly higher prevalence of T2D and a lower prevalence of CAD (statistically significant) and stroke (statistically nonsignificant) compared with those with low MMLs. Those with high concentrations of MMLs compared with low had a higher prevalence of T2D but a lower prevalence of CAD and stroke, but these findings were statistically nonsignificant (Figure 3).

Adjustment for Race and Ethnicity

In addition to the variables included in Model 3, we further adjusted for the proportion of self-identified

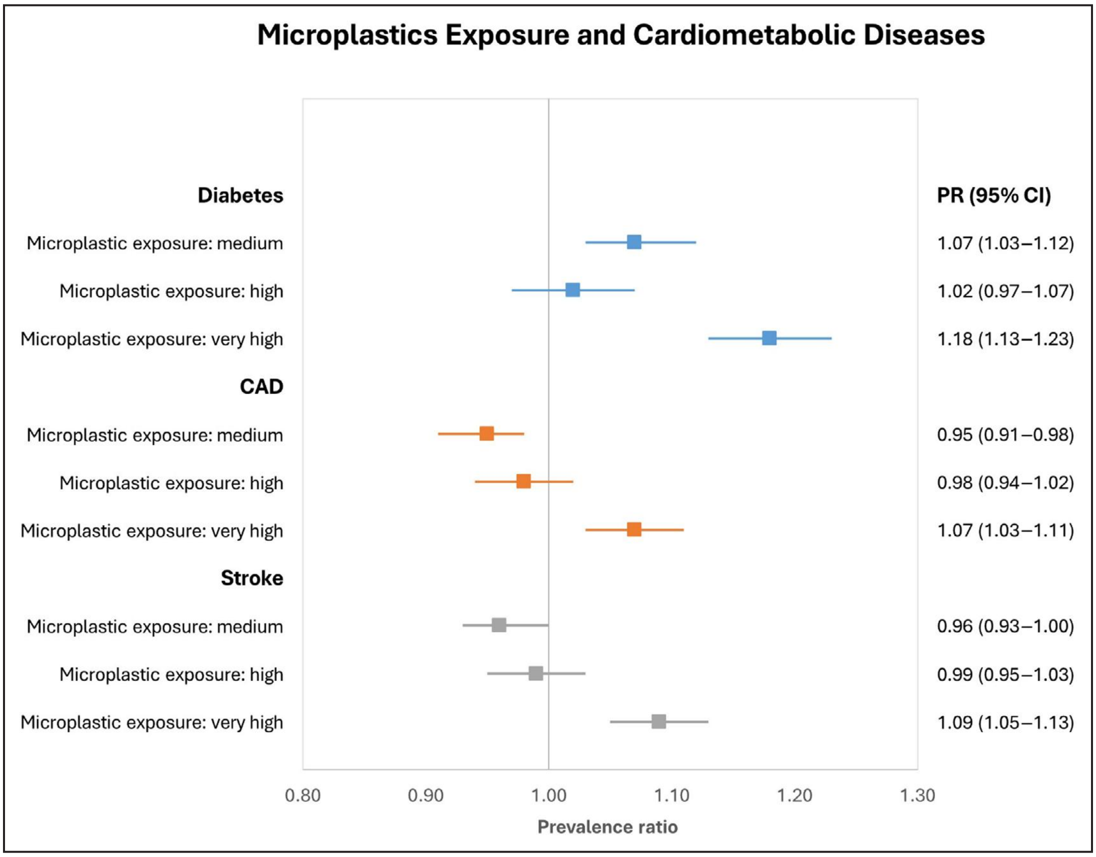

Figure 3. Forest plot showing prevalence ratios of T2D, CAD, and stroke among counties with medium, high, and very high MMLs compared with low MMLs in the fully adjusted model (Model 3) after adjusting for age, sex, physician access, and baseline community vulnerability of CVI.

CAD indicates coronary artery disease; CVI, Climate Vulnerability Index; MMLs, marine microplastic levels; PR, prevalence ratio; and T2D, type 2 diabetes.

Black and Hispanic individuals residing in the coastal counties (Model 4). The association between MMLs and county-level cardiometabolic disease prevalence remained statistically significant, with an adjusted PR of 1.05 (95% CI, 1.01–1.09; *P*=0.01) for T2D, 1.06 (95% CI, 1.02– 1.11; *P*=0.008) for coronary artery disease (CAD), and 1.04 (95% CI, 1.00–1.07; *P*=0.049) for stroke among counties with exposure to very high MMLs compared with low MMLs (Table 2, Model 4).

Sensitivity Analysis

Sensitivity analysis using multivariable linear regression with robust SEs from the heteroscedasticity-consistent variance estimator, showed consistent results. MMLs were significantly associated with prevalence of T2D, CAD and stroke after adjustment for age, race, ethnicity, sex, physician access, socioenvironmental vulnerabilities of communities (Table S2). Coastal counties with very high MMLs had a significantly higher adjusted mean prevalence of T2D, CAD, and stroke compared with their nearest noncoastal counterparts (Tables S3 and S4).

DISCUSSION

In this analysis of 152 coastal counties, we found that coastal counties potentially exposed to very high marine microplastic levels based on adjacent ocean water samples collected from 2015 to 2020 around the United States were associated with a higher prevalence of T2D, CAD, and stroke after adjustment for the median age of county population, sex, race, ethnicity, physician access, and the baseline socioeconomic, infrastructure-related and environmental vulnerabilities of the communities residing in these counties.

Both experimental and real-world models highlight the correlation between microplastics in the local marine environment and groundwater. Seawater intrusion, defined as the natural phenomenon of mixing seawater with groundwater resources, is widely described in geological studies.[17,18] This phenomenon, reported extensively in coastal areas, results in a high concentration of various contaminants, including microplastics, in the groundwater aquifers near the coastal areas.[19–23] Experimental studies with visible flow chamber systems

Table 2. Prevalence Ratios and CIs for Study Outcomes

Exposure	Outcome	Model	Prevalence ratio (95% CI)	*P* value
Marine microplastic level: very high	Type 2 diabetes	Model 1	1.27 (1.19–1.35)	<0.001
		Model 2	1.19 (1.11–1.26)	<0.001
		Model 3	1.18 (1.13–1.23)	<0.001
		Model 4	1.05 (1.01–1.09)	0.01
	Coronary artery disease	Model 1	1.39 (1.29–1.49)	<0.001
		Model 2	1.11 (1.05–1.17)	<0.001
		Model 3	1.07 (1.03–1.11)	<0.001
		Model 4	1.06 (1.02–1.11)	0.008
	Stroke	Model 1	1.33 (1.24–1.42)	<0.001
		Model 2	1.12 (1.05–1.19)	<0.001
		Model 3	1.09 (1.05–1.13)	<0.001
		Model 4	1.04 (1.00–1.07)	0.049

Model 1: unadjusted. Model 2: adjusted for age, sex, and physician access. Model 3: adjusted for age, sex, physician access, and percentile rank of baseline socioeconomic, infrastructure, and environmental vulnerability of Climate Vulnerability Index. Model 4: Adjusted for age, sex, physician access, percentile rank of baseline socioeconomic, infrastructure, and environmental vulnerability of Climate Vulnerability Index, and the proportion of self-identified Black and Hispanic individuals residing in the coastal counties.

showed that 0.2, 1, and 2 μm plastic particles could easily translocate from seawater to groundwater via coastal aquifers.[24] A real-world assessment in the Laizhou Bay area suggests that microplastic concentration in coastal groundwater is indeed correlated with levels found in their local marine environments.[25] Microplastics have also been detected in capped groundwater bores, presumably via permeation through soil.[26,27] Because groundwater supplies 35% of drinking water in the United States, its contamination with microplastics provides a direct portal of entry into human bodies.[28]

The contamination of seafood with microplastics is a common occurrence. Microplastics have been detected in approximately 45% of fish ingested globally.[29] Masury and Schumann found that seafood availability decreased from coastal to inland markets, suggesting higher seafood consumption near the coast.[30,31] Microplastic fibers, fragments, and films were found in almost 98% of seafood samples collected from the Oregon coast and Oregon retail markets in 2021.[32] Altogether, although still an underestimate, Americans may ingest 39 000 to 121 000 microplastic particles annually through food and inhalation, with bottled water contributing up to 90 000 additional particles per year.[33]

Previous studies on the distribution of microplastic pollution have reported higher total particle counts in the Northern Pacific Ocean compared with the Northern Atlantic, with accumulation primarily occurring in the open ocean rather than along the US coastline.[34] In contrast, our analysis, which focused on microplastic concentrations within 200 nautical miles of the US coastline, identified higher levels near the Atlantic coastline. This discrepancy may be attributed to differences in study design, spatial coverage, and oceanic transport dynamics, highlighting the need for further investigation into regional microplastic deposition patterns. We observed that the prevalence of cardiometabolic diseases was higher among the counties located in the east and south coast, compared with the west coast. This aligns with the reports of geographic variation in the cardiometabolic diseases prevalence and associated risk factors, with individuals in the Western United States exhibiting better cardiovascular health and lower prevalence of traditional risk factors compared with those in the Northeast, Midwest, and South.[35] A combination of factors, including socioeconomic status, dietary patterns, and health care access, among others, may contribute to the higher burden of disease observed in the Northeast compared with the West.[35]

Our findings are consistent with previous animal studies and experimental reports on the association between microplastic exposure and cardiometabolic diseases. Microplastics may affect the cardiac and vascular systems both directly and indirectly. Animal studies have shown that short-term exposure to 8 to 12 μm polystyrene particles on isolated hearts of adult zebrafish led to a dose-dependent decrease in ventricular contraction frequency and subsequent bradycardia.[2] The effects persisted even on waterborne zebrafish larvae and embryos. Furthermore, polystyrene microplastics have been found to induce myocardial inflammatory injury in chicken cardiomyocytes and promote cardiomyocyte apoptosis by activating caspases and reactive oxygen species.[36]

Lu and colleagues discovered that nanoplastics interact with human umbilical vein endothelial cells, causing damage to the endothelial cell membrane.[37] Additionally, internalized nanoplastics disrupt autophagic processes in vascular endothelial cells, leading to cell injury and death.[37] When added to plasma in vitro, carboxyl-modified nanoplastics activate the intrinsic pathway of blood coagulation in plasma.[38] Hence, microplastics create a proinflammatory and prothrombotic milieu in blood vessels, accelerating atherogenesis. A prospective study by Marfella and colleagues further strengthened the mechanistic evidence in humans, showing that individuals with carotid artery plaque containing MNPs had a higher risk of developing myocardial infarction or stroke over 34 months of follow-up compared with those without.[8] Interestingly, microplastics were also detected in postmortem samples of the human brain, liver, and kidney, with a significantly higher deposition in cerebrovascular walls and immune cells of decedent brains with documented dementia diagnosis.[39]

Animal studies suggest an association between microplastics and metabolic derangements. Mice exposed to microplastics in their diet for 10 weeks demonstrated a significant decrease in the expression of IRS1 (insulin receptor substrate 1) and p-IRS1 (phosphorylated insulin receptor substrate 1), culminating into insulin resistance, as compared with mice fed with normal chow diet.[40] In pregnant women, urinary concentrations of microplastic compounds like phthalates, often used as plasticizers in consumer products, were positively correlated with impaired glucose tolerance and gestational diabetes.[41]

Although marine microplastics can enter the human body by consumption of contaminated seafood or water, the modes of exposure may not be limited to ingestion. Microplastics can become airborne and be inhaled, particularly in coastal areas where sea spray and wind can disperse them. Direct contact with marine microplastics, such as through recreational activities on contaminated beaches, could also pose a risk of exposure. However, a conundrum exists with regard to the exposure–response relationship across different modes of microplastics exposure to humans. Analyzing fish ingestion among people through a questionnaire could be a valuable approach to understanding potential exposure to microplastics through ingestion and assessing associated health risks, risk communication strategies, and policy implications related to seafood consumption, ultimately supporting efforts to address the environmental challenges posed by microplastic pollution.

Our work could drive translational studies to elucidate cellular and molecular mechanisms of microplastic-mediated damage in human tissues. The mechanisms underlying the association between microplastics and human health, including but not limited to cardiovascular diseases, should be further distilled and solidified in order to promote evidence-based decision-making and influence regulatory policies at governmental levels, leading to stricter regulations on plastic production, use, treatment, and disposal to minimize the release and accumulation of microplastics into the environment as well as to develop interventions to reduce current levels in the environment. Individual-level exposure assessments, such as seafood consumption surveys and water quality analyses, may help establish threshold levels of exposure associated with human disease risk. Altogether, a foundation of causality based on robust evidence may guide efforts to establish a maximum permissible limit to the level of microplastics in drinking water supply, packaged water, and seafoods at the supply source and consumers' end, akin to the recently developed legally enforceable drinking water standard in the United States that includes measurement for per- and poly-fluoroalkyl substances and numerous other drinking water guidelines to protect communities from exposure to the harmful effects of their exposure, particularly in high-risk coastal areas.[42–46]

Limitations

Our study has several limitations. First, although MNPs can be detected in marine and terrestrial ecosystems worldwide and be ingested and accumulated by animals, which can then be passed down along the food chain, our study is ecological and considers only the concentration of MNPs in ocean water, a part of the marine ecosystem. Although consuming groundwater or seafood containing microplastics may lead to human exposure, it is difficult to precisely quantify the microplastic fraction entering human bodies and accumulating in our tissues and define a threshold of level and chronicity of microplastics in human body that leads to diseases, in the absence of good-quality evidence.

The unit of analysis for our study is a county. Hence, individual-level associations cannot be drawn from our study results. Due to data source restriction, we were not able to substratify outcomes based on microplastic type. Being cross-sectional, our study cannot determine the temporality of association. The population migration across the counties is unknown and unaccounted for during the study period. The relationship between the prevalence of cardiometabolic diseases and coastal pollution policies may be bidirectional, indicating that a greater burden of disease could necessitate the implementation of stricter environmental regulations and mitigation measures. This underscores the importance of adopting a longitudinal study design in future research. The interaction of other unknown confounders and nonstandard modifiable risk factors that directly or indirectly affect the study outcomes may have been missed.

Leaving apart the counties included in the sensitivity analysis, our analysis did not include counties that did not share a boundary with the coast. Although not directly in contact with ocean water, people residing in these counties can still be exposed to MNPs through other routes, such as direct ingestion or dermal contact. There remains a lack of established pathogenicity thresholds for microplastics in human disease. Although our findings suggest an association between microplastic exposure and cardiometabolic diseases, the precise exposure–response relationship remains unclear. Given that microplastics can act as vectors for toxic substances, even low environmental exposures may have cumulative or synergistic effects over time.[45] Future research should focus on defining exposure thresholds that drive pathogenic mechanisms and contribute to disease risk. Due to database restrictions, we could not explore the results of diverse types of microplastics individually. Lastly, our results do not imply causation due to the study's cross-sectional and ecological nature.

Even with inherent limitations, our findings may be, at best, the underestimation of the hazard posed and raise the need to investigate further and intervene to address this emerging environmental hazard.

CONCLUSIONS

The coastal counties with a higher concentration of marine microplastics in adjacent ocean water showed a higher prevalence of T2D, CAD, and stroke compared with those with lower concentrations. This association remained significant after adjusting for age, sex, physician access, and baseline community vulnerabilities of the counties. Together with other bodies of evidence studying the health concerns of microplastics to humans and the environment, our study supports the need for policies to protect against microplastic exposure and reduce marine water pollution.

ARTICLE INFORMATION

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Supplemental Material

Tables S1–S4
Figures S1–S4

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