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ABSTRACT

A century of plastic usage has led to an accumulation of plastic waste in waterways and oceans. Over time, these wastes break down into particles smaller than 5 microns – or "microplastics" – which can infiltrate human biological systems. Despite decades of research into this emerging source of environmental pollution, there is a lack of direct evidence on the health impacts of microplastics exposure at a population scale. This paper reports the first causal link between in-utero microplastic exposure and adverse birth outcomes. Our analysis is based on a compiled dataset of 3 million births that occurred in coastal areas of 15 countries spanning four continents, which we merge with a novel remote-sensing measurement of marine microplastic concentrations. We show that in-utero exposure to microplastics, particularly during the third trimester of pregnancy, leads to a significant increase in the likelihood of low birth weight. A doubling of exposure increases low birth weight hazard by 0.37 per 1,000 births, which implies over 205,000 cases per year globally can be attributed to microplastic exposure. We further show that aerosolization – whereby microplastic particles become airborne and inhalable due to seawater evaporation – is an important pathway for health impact, a challenge that is likely to escalate as ocean temperatures continue to rise.

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1. Introduction

Plastic has gained immense popularity since its invention in the 1900s. It was originally introduced for specialized uses such as in the production of electrical insulators and automotive parts, but the versatility of the material – its durability, light weight, and low cost – soon proved advantageous in a variety of applications, ranging from the lightest everyday uses such as garment making, to the heaviest industrial applications such as aviation (Andrady and Neal, 2009). Plastics production reached 400 million tons in 2022, with packaging being the largest market that accounts for approximately 40 percent of this total amount (Geyer, Jambeck, and Law, 2017).

The environmental impact of plastic wastes has long been debated. Most plastics do not decompose in the same way organic materials do. Instead, they *fragment*, breaking down into smaller and smaller pieces over time. This results in microplastics, tiny plastic particles that can persist in the environment indefinitely (Rillig, 2012). The technology of waste plastic composts and recycling are still immature, leading to an estimated 80 percent of waste ending up in landfills or the natural environment. Much of these wastes are mismanaged: they are either littered or inadequately disposed, with an estimated 4.8 to 12.7 million metric tons ending up in rivers and oceans (Jambeck et al., 2015). Tiny particles may eventually become airborne – a prediction that we will test in this paper – under the right environmental conditions and thus end up in the air that we breathe (Prata, 2018).

Decades of research have accumulated a substantial body of knowledge on emission sources of marine microplastics, their ambient presence and fate, and exposure risks of human and other organisms. In contrast, the availability of data and direct evidence on the health effects remain virtually absent to this date (Lim, 2021; Landrigan et al., 2023). In this paper, we provide the first global-scale estimate of the causal effect of microplastic exposure on infant health. This research addresses three main challenges. First, measurements of marine microplastic pollution are sparse, with most available datasets being cross-sectional. To overcome this, we use a space radar-based remote sensing method that detects microplastic presence on the ocean surface by observing the wind-dampening effect on surface roughness (Evans and Ruf, 2021). This new technique correlates well with in-situ sampling data and provides a longitudinal measurement

for microplastics at a high temporal frequency, which is crucial for health analyses that require measurement of individual exposure differences.

Second, microplastic concentration in a given location might be correlated with various environmental and economic factors – such as air temperature or water quality – that independently influence health outcomes. We develop an oceanography transport model that leverages idiosyncratic day-to-day variability in ocean current motions to predict how microplastics from the open sea (such as areas over 200 km from the coast) can be expected to reach coastal locations over time. This method helps tease out portions of coastal microplastic exposure that are quasi-random – those that are likely independent to any of the coastal-local confounding factors, which allows us to cleanly isolate out the causal impact of microplastics.

Third, detecting the health effects of microplastic exposure can be challenging due to the complexity of individuals' environmental exposure and the potential delay in health responses. We focus on infant health as mothers are generally stationary during pregnancy, allowing for more accurate exposure measurement (Currie, 2011). Unlike adults, infants do not have a long, complex history of environmental exposure, making econometric modeling of health outcomes and environmental risks—such as in-utero microplastic exposure—relatively straightforward. We compile individual-level data on birth weight from multiple data sources including the Demographic and Health Survey for developing countries, and administrative birth records from Brazil, Mexico, and the United States. Our compiled data includes 3 million individual births from 15 different countries, covering coastlines across four continents. The large sample size and wide geographic scope ensure that our estimates are statistically robust and representative of multiple regions worldwide. We use incidence of low birth weight (infants weighing less than 2,500 grams at birth) as our main infant health marker, a primary measure of infant health that has been linked to environmental stressors (Currie and Almond, 2011).

We find robust evidence that in-utero exposure to microplastics significantly increases the likelihood of low birth weight (LBW) among mothers living near the coast. In our pooled sample of births within 200 km of the shoreline, each doubling of microplastic concentration during pregnancy increases LBW incidence by 0.37 per 1,000 births. This effect is primarily driven by exposure during the second and third trimesters. In "placebo" exercises, we show that the same econometric model indicates no impact of microplastic exposure during the preconception and postpartum periods. The effect size we find is significant, suggesting that

microplastic variability accounts for about 2 percent of all low birth weight births. This rate ranges from approximately 1.2 percent in the USA to over 8 percent in certain developing countries in Africa and South Asia. Scaled linearly, our estimate implies that about 205,800 of LBW coastal births annually worldwide can be attributed to microplastic exposure.

These results remain robust across a range of alternative explanations and specification tests. First, we show that in-utero microplastic exposure shocks do not predict pre-determined maternal characteristics such as the mother's age, education, or the weather and economic conditions of her city of residence, suggesting our main findings are not explained by changes in the composition of mothers. Second, we test whether our results could reflect the transport of other offshore pollutants rather than microplastics *per se*. To do so, we run a permutation exercise: we hold the observed ocean current pathways as is, but randomly shuffle the microplastic concentrations across all open-sea grid cells. We then use each reshuffled distribution to re-estimate our baseline regression, thereby generating a "placebo" distribution of effect sizes. Our actual estimate—calculated using the true microplastic distribution—lies well outside this placebo distribution, suggesting that the health impact we observe is driven by the real spatial pattern of microplastics and not by any generic feature of ocean currents. Finally, our estimates hold up under a range of changes to the econometric specification and outcome construction, including different coastal distance bandwidths, varying the granularity of fixed-effects controls, and using alternative measures of adverse infant health outcomes such as the incidence of pre-term birth.

We explore possible underlying mechanisms, considering two general pathways: water and air. One intuitive channel of impact is the accumulation of microplastics in water and their subsequent consumption through microplastic-contaminated seafood. We leverage two different data sources from retail scanner data from the US and global Automatic Identification System fishing vessels tracking data to build proxies for seafood access. Using both of these proxies, we found no statistically significant evidence that areas with higher seafood access are associated with a larger health effect.

We next turn to the atmospheric pathway. As discussed above, plastic waste can break down into fragments small enough to become aerosolized. Our data shows a consistent pattern: variations in microplastic levels strongly predict the concentration of coastal aerosol particulate pollution, as measured by an independent satellite instrument. This relationship intensifies with

higher rates of evaporation in coastal regions. Combining this with established evidence that fine particulates can penetrate multiple organs via respiratory invasion, and specific evidence on the impact of in-utero exposure to ambient particulate matter on low birth weight outcomes, our data suggests aerosolization is a potentially important mechanism through which microplastic pollution affects infant health.

To our knowledge, this paper is the first to establish a direct link between ambient microplastic exposure and a human health outcome using large-scale data. Our findings corroborate decades of research across multiple fields warning about the potential risks of microplastic exposure. We are guided by knowledge from atmospheric and oceanic sciences that detect microplastics through remote-sensing techniques, from environmental toxicology that predicts the impact of microplastics on organisms, and from medicine that tests the health impacts of microplastics (See our review of current body of knowledge in Section 2). Our results support important hypotheses from prior literature, such as the relevance of microplastic aerosolization. We offer a much larger sample size for studying birth impacts, extending beyond the existing medical evidence that often relies on small sample clinical observations (Amereh et al., 2022).

Our findings on microplastic aerosolization highlight a new, pervasive source of airborne particulate pollution and raise important questions about their broader impacts. There is extensive scientific evidence on the health effects of particulate matter (PM) pollution, which has been linked to adverse outcomes across cardiovascular, respiratory, and mental health domains (e.g., Landrigan et al., 2018). This evidence has motivated regulatory actions on PM globally. Existing research has primarily attributed PM to direct emissions or secondary formations from burning and combustion processes. Our study, however, identifies microplastics as an additional source of ambient PM emanating from the waste sector. The extensive range of impacts and the social costs associated with microplastic-derived PM require further investigation.

From a policy perspective, our research emphasizes the natures in which microplastic pollution is a global challenge. Microplastic particles travel long distances: in our ocean current modeling analysis, a coastal area's microplastic variability can be explained by conditions hundreds of kilometers away in the open sea, which are themselves influenced by emission sources far away. On the health side, the adverse impacts of microplastics appear to be far more widespread than previously appreciated. We find significant effects in both the USA data and in

developing countries. The fact that the low birth weight effects do not appear through a seafood intake channel but through a broader atmospheric channel means population exposure is less selective than previously thought. Micron-sized particles are difficult to filter out and can easily penetrate indoor environments, rendering normal protective measures less effective (Chen and Zhao, 2011). Our findings highlight the interconnected nature of the global plastic problem—emissions from one site affect distant areas—and underscore the importance of global plastic waste management and reduction programs. The downstream benefits of such initiatives are currently underappreciated but have the potential to be enormous (Borrelle et al., 2020; He et al., 2023).

The economics of transboundary pollution has been extensively studied in the contexts of air and freshwater pollution. Pollutants transported by wind (Jia and Ku, 2019; Heo, Ito, and Kotamarthi, 2023; Dipoppa and Gulzar, 2024) and waterways (Kahn, Li, and Zhao, 2015; He, Wang, and Zhang, 2020) enable emitters to strategically choose when and where to release pollutants, posing challenges for environmental regulations. Ocean pollution and its transboundary impacts are increasingly important, as highlighted by numerous oil spill events, the Great Pacific Garbage Patch, waste from fishing and military vessels, and deep-sea mining activities. Our paper provides a framework to study transboundary ocean pollution, which has so far been largely unregulated except in specific economic activities like fishing zones or vessel congestion.

Our paper also contributes to the broad literature on in-utero exposure to environmental shocks and infant health outcomes (e.g. Almond, 2006; Jayachandran, 2009; Almond and Mazumder, 2011; Currie and Walker, 2011; Currie and Rossin-Slater, 2013; Currie, Greenstone, and Meckel, 2017; Rangel and Vogl, 2019; Hansen-Lewis and Marcus, 2022; Hill and Ma, 2022; Currie et al., 2022). We provide one of the first studies showing ocean pollution affects early-life health. Related to our study, Guimbeau et al. (2024) found ocean salinity affects childhood heights and weights through agricultural intensity and land use changes. Armand and Taveras (2021) documented ocean acidification increases neonatal mortality and affects parental investment on child development, and the channel lies in maternal malnutrition. These two papers focus on ocean chemical composition that is primarily changed by long-term climate change. In contrast, our focus on ocean microplastics has daily variation due to changes in anthropogenic sources and high-frequency ocean currents. To address endogeneity concerns, we

explore ocean current direction and speed and construct transported microplastics exposure. This proposed identification strategy is novel and provides a framework for future ocean studies.

Section 2 reviews scientific background. Section 3 describes data sources and methods. Section 4 presents the results. Section 5 concludes.

2. Background

Environmental and health scientists have been warning for decades about the presence and potential health effects of microplastics exposure. This section provides a concise review of existing research on microplastic emissions, exposure, and health effects. Appendix B provides a comprehensive survey of the scientific and clinical literature for interested readers.

Sources and Accumulation. Global use of plastics has grown exponentially since the mid-20th century. Annual plastic production has surged from just 2 million tons in 1950 to roughly 400 million tons in 2022. A significant share of this plastic becomes waste that pollutes oceans: on the order of 5–13 million metric tons of plastic debris enter marine environments each year (Worm et al., 2017). Figure 1, panel (a) summarizes some primary sources of microplastics. Roughly 80% of marine plastic originates from land-based sources (e.g. mismanaged waste, litter, and river discharge), with the remaining 20% coming from ocean-based activities such as fishing and shipping (Redford et al., 1997; Browne et al., 2010; Cole et al., 2011; Li et al., 2016). These plastics fragment over time via UV exposure and mechanical abrasion, breaking down into microplastics (typically <5 microns in diameter) that persist in the water. Ocean currents concentrate floating microplastic particles in gyres and convergence zones (the so-called garbage patches), and substantial amounts also settle into seafloor sediments and coastal areas.

This vast distribution raises concerns about ecological and economic impacts, as marine ecosystems and coastal economies bear rising costs from habitat damage, cleanup, and loss of amenity. Microplastics have been detected in a wide range of marine organisms, including commercially important species. Studies find these particles in many seafood items – from filter-feeding bivalves and crustaceans to various fish species destined for human consumption. See Figure 1, panel (b) for an example. Plastics tend to accumulate in digestive tracts, so seafood

consumed whole (e.g. oysters, mussels, small fish) poses a higher risk of introducing microplastics into the human diet.

Land and Atmospheric Pathways. Microplastic pollution is not confined to the oceans; substantial amounts reside in soils and are dispersed through the atmosphere. Terrestrial pathways begin with plastics released on land – for example, fragments from discarded plastics, synthetic textile fibers, tire wear particles, and microbeads in wastewater. Much of this material is carried by runoff into rivers and eventually to the sea, but a considerable fraction remains on land (Rillig, 2012; Lwanga et al., 2016; Li et al., 2019). Wastewater treatment plants, for example, capture microplastics in sewage sludge (Nizzetto, Futter, and Langaas, 2016). Soil studies have documented microplastic fragments in a range of terrestrial settings, from urban parks to remote croplands (Sajjad et al., 2022). Once in the soil, microplastics can be transported vertically and horizontally: earthworms and other soil biota have been shown to carry microplastic particles deep into soil profiles, and heavy rains can wash them into streams or groundwater (Rillig, Ziersch, and Hempel, 2017).

Airborne transport constitutes another major pathway for microplastic distribution. Recent research has overturned the notion that oceans are the "final sink" for plastics. Tiny plastic particles can become aerosolized and travel vast distances through the atmosphere, moving even faster than ocean currents. Field measurements find microplastics in the air above both densely populated cities and remote regions, carried by wind currents and weather systems. One mechanism for this aerosolization is the action of wind and waves at the ocean–atmosphere interface: breaking waves and sea spray can eject microplastic-laden sea foam and salt particles into the air (Prata, 2018; Liu et al., 2019). Raindrops striking a polluted water surface can fling microplastic-rich droplets upward; as those droplets evaporate, the plastic particles are left suspended in the atmosphere (Lehmann et al., 2021). Once aloft, microplastics eventually redeposit to land or sea far from their source. Atmospheric studies further reveal that microplastics are transported across vast distances by air currents, with sea spray and atmospheric circulation playing a significant role in their global distribution (Allen et al., 2020; Caracci et al., 2023). This atmospheric pathway implies a direct route of human exposure: people may inhale microplastics carried in windblown dust or sea breeze, just as they do other forms of particulate air pollution.

Evidence on Health Effects. Despite the omnipresence of microplastics in the environment, scientific understanding of their impacts on human health is still nascent. Environmental and medical researchers have warned about the potential risks, but direct empirical evidence remains limited (Lim, 2021).

Nonetheless, a growing body of laboratory and clinical research has begun to shed light on possible health hazards. Tiny plastic particles have been found in tap water, sea salt, and even the air in our homes, implying constant low-level ingestion and inhalation. See Figure 1, panel (c) for examples (Vianello et al., 2019). Studies have confirmed that some fraction of these particles can enter and persist in the body – for example, researchers have detected microplastic fibers in human lung tissue and plastic fragments in human stool and placentas (Zhang et al., 2025).

Once in the respiratory or gastrointestinal tract, microplastics may interact with the body in harmful ways. Controlled experiments have shown that microplastic exposure in animals can provoke a variety of toxicological effects, including increased reactive oxygen species, metabolic disturbances, and tissue damage in the liver, kidneys, and intestines (Li et al., 2023). There are also indications of impacts on reproductive health: for example, male laboratory animals exposed to high microplastic doses have exhibited reduced sperm quality and fertility (Emenike et al., 2023). Epidemiological evidence connecting microplastic exposure to health outcomes in human populations is only beginning to emerge. To date, most human studies have been small in scale or observational, and results are not yet conclusive. Researchers have speculated about links between chronic microplastic exposure and conditions like respiratory illnesses, cardiovascular disease, or cancers, given that some inhaled particles can cause lesions and that plastic additives include carcinogens (Chartres et al., 2024). Our review of existing case studies notes that most have limited sample sizes or short follow-up periods, which highlights the need for more robust evidence (Appendix B).

The possibility that an emerging pollutant like microplastics could undermine population health invites comparisons to well-known pollutants (e.g. fine particulate air pollution) that have established economic significance. However, without rigorous population-level studies, the magnitude of microplastics' impact remains difficult to quantify. This gap in knowledge provides a strong motivation for our work.

3. Empirical Methods

3.1 Data Sources

Marine Microplastics. Our main measure of ocean microplastics comes from the CYGNSS Level 3 version 1.0 remote-sensing dataset (Evans and Ruf, 2021). This satellite-based method uses bistatic radar reflections to detect microplastic pollution indirectly. Surface-active pollutants like microplastics (and their associated surfactants) dampen the ocean's surface roughness that wind usually generates. The CYGNSS algorithm exploits this effect: when an ocean area exhibits less surface roughening than expected under given wind conditions, it infers a higher microplastic concentration in that location. The CYGNSS microplastic data are available daily on a 0.25° × 0.25° grid from April 2017 through September 2018. For each coastal birth in our sample, we assign an in utero microplastic exposure value based on the concentration in the nearest coastal grid cell during that pregnancy.

Figure 2, panel (a) maps the spatial distribution of microplastic concentrations detected by CYGNSS. Due to geometric constraints of the satellite radar, the measurements cover latitudes between 37°S and 37°N. Figure 2, panel (b) shows the aggregated weekly microplastic concentrations in seven coastal regions where our birth data are concentrated. A notable feature is the idiosyncratic temporal variation: microplastic levels follow distinct trends in different parts of the world. This heterogeneity is helpful for identification because it helps disentangle microplastic exposure from other factors (such as temperature) that follow more regular seasonal cycles.

We rely on the Evans and Ruf (2021) data as our primary microplastic measure because its temporal frequency is crucial for constructing prenatal exposure profiles. In contrast, most other available microplastic datasets provide only cross-sectional snapshot. As discussed in Section 2, in-situ measurements of marine microplastics remain relatively limited. Appendix Figure 1 shows that our remote-sensing metric aligns well with ground-based observations. In a cross-sectional comparison, the 2017–2018 average CYGNSS microplastic concentrations are significantly correlated with NOAA's NCEI in-situ microplastics database (over 8,000 water samples collected since 1972). This positive correlation holds for the full sample and also when restricting to post-2013 in-situ observations (which were not used to train the remote-sensing model).

Ocean Currents. We obtain ocean current data from NASA's Ocean Surface Current Analyses Real-time (OSCAR) product. OSCAR provides daily current direction and speed on a 0.25° spatial grid. These currents are derived by combining multiple satellite observations (sea surface height, surface wind vectors, and sea surface temperature) in a quasi-linear steady-flow ocean circulation model.

Coastal Ocean Chlorophyll. We use chlorophyll-a concentration as a proxy for coastal nutrient pollution, since chlorophyll indicates algal growth and thus nutrient levels in the water.[1] Chlorophyll concentration data are sourced from the Visible Infrared Imaging Radiometer Suite (VIIRS), which provides daily observations with a spatial resolution of 4km, measured in mg per m3. This satellite network monitors water bodies globally, including inland and ocean waters. We generate monthly nutrient pollution levels for coastal locations within a 100km ocean buffer.

Atmospheric Conditions. To test for aerosolization of microplastics, we use aerosol optical depth (AOD) data from MERRA2 reanalysis product. This product is based on bias-corrected AOD retrieved from AVHRR, MODIS, and MISR satellite sensors, along with ground observations of AERONET AOD data. The data is at a spatial resolution of 0.5° × 0.625°.

We use evaporation data from the NCEP/DOE (National Center for Atmospheric Research/Department of Energy) Reanalysis II product. This data measures the strength of water evaporation into the atmosphere, which captures the aerosolization process from marine microplastics. The data are at the monthly level with a spatial resolution of 2.5°.

We obtain meteorological condition data from the European Centre for Medium-Range Weather Forecasts Reanalysis 5 (ERA5) products. This dataset provides hourly information on temperature and precipitation, with a grid size of 0.25°. We aggregate the data to adm1-month level.

Seafood Access. We use two proxies to capture local seafood availability and consumption. First, we use household seafood purchase data from the Nielsen consumer panel in the United States. The Nielsen panel tracks grocery purchases for a representative sample of households over time using in-home barcode scanners, providing detailed information on each

[1] Chlorophyll-a can also be interpreted as a proxy for primary productivity in the ocean. Higher primary productivity often supports larger populations of zooplankton, which in turn support higher fish populations.

household's location (county and ZIP code), demographics, and purchase history. We aggregate seafood-related purchases to the county-by-month level as a measure of local seafood consumption.

Second, we incorporate fishing activity data from Global Fishing Watch to capture local fishery effort. This dataset tracks fishing vessels via automatic identification system (AIS) signals. Using the methodology of Rousseau et al. (2024), we translate the raw AIS signals into measures of fishing effort (hours)—leveraging vessel capacity and catch data for validation—and then aggregate total fishing hours within a 100 km radius of each community (defined as a DHS cluster, U.S. county, Mexican locality, or Brazilian municipality in our sample). This provides an indicator of the importance of fishing activity in each area. Appendix Figure 2 maps the geographic distribution of our seafood access metrics (household seafood purchases and fishing effort).

Birth. We compile birth outcomes from four sources. First, we use the Demographic and Health Surveys (DHS) for 12 coastal countries (across 14 survey rounds). For our main analysis, we restrict the DHS sample to mothers living within 200 km of the coast, yielding 10,545 births that we can match with microplastic exposure data. In robustness checks, we vary this coastal bandwidth selection. Our outcome of interest is an indicator for low birth weight (infants weighing below 2,500 grams at birth), constructed from the reported birth weight. Because the DHS only reports locations at the cluster level (each cluster is a centroid representing a roughly 10 km radius area), all households within a cluster share the same coordinates. Figure 2, panel (a) shows the locations of the DHS clusters in our sample (mostly in Southeast Asia and Africa) and highlights the variation in survey coverage across countries.

Second, we use U.S. vital statistics from the National Vital Statistics System (NVSS). The NVSS data include 934,081 births in 2017–2018 across 461 coastal counties.

Third, we use birth records from Mexico's national health system. This dataset covers all registered births in the country and reports each infant's birth weight, survival status, and the mother's demographic information and locality of residence (analogous to a U.S. county). We extract 725,726 births from 1,221 coastal localities in Mexico for our analysis.

Finally, we use birth records from Brazil's Live Birth Information System (SINASC). SINASC is a national registry of live births collected via a mandatory "Declaration of Live Birth"

form. The data include each newborn's birth weight, sex, the mother's age, and the municipality of residence. Our Brazilian sample comprises 1,344,769 births from 2,419 coastal municipalities.

3.2 Econometrics

Our goal is to estimate the causal impact of microplastic exposure while in utero on birth outcome. The workhorse econometric equation is as follows:

$$\text{Low Birth Weight}_{i,t} = \beta \cdot \text{Log} \sum_{k\in[-1,-9]} \text{Microplastics}_{i,t+k} + \alpha_{adm1} + \alpha_{country-month} + X_{i,t}\gamma + \varepsilon_{i,t} \quad (1)$$

where $\text{Low Birth Weight}_{i,t}$ is an indicator variable for whether birth i occurring at month t is weighing less than 2,500 grams. To increase readability of the coefficient estimates, we multiply the dummy variable by 1,000, so that the outcome variable represents the incident of low birth weight per 1,000 births. $\sum_{k\in[-1,-9]} \text{Microplastics}_{i,t+k}$ captures i's exposure to microplastics while in utero, which equals the logged sum of microplastic levels observed at the nearest coastal sea grid to i's birth location over the 9-month period preceding the birth. For each birth, we match it to the 0.25 degree grid in the microplastic data that are the closest to the birth's location. Birth locations are measured by survey cluster latitude and longitude in the DHS data, county centroid in the USA data, municipality centroid in the Brazil data, and municipality centroid in the Mexico data.

α_{adm1} are region fixed effects. We define regions as "adm1" administrative level-1 subregion (e.g., state for the U.S. and Estados for Brazil and Mexico). In robustness specifications, we report versions with more stringent, administrative level-2 subregion fixed effects (e.g., county for the U.S. and Municipios for Brazil and Mexico). $\alpha_{country-month}$ are country by month-of-sample fixed effects that control for country-specific seasonal and secular trends. $X_{i,t}$ includes time-varying environmental covariates, which helps rule out confounding effects from other factors that might correlate with microplastic exposure while might also having independent impacts on birth outcomes, including temperature, precipitation, air pollution, and ocean chlorophyll-a. $\varepsilon_{i,t}$ is the error term. Standard errors are clustered at the admin1 level.

The key coefficient of interest is thus β, which captures the impact of in-utero exposure to microplastics on likelihood of low birth weight. The challenge in interpreting β as the causal effect

of microplastics lies in potential endogeneity: some third factors correlated with microplastics are directly affecting birth outcomes and are not captured by the fixed effects and co-pollutant controls. This concern is less likely to be substantial compared with other forms of environmental pollution because it takes at least several decades for microplastics to form. As a result, the third factors affecting microplastic generation may be correlated with socioeconomic conditions decades ago, but not recent ones, and are less likely to directly impact birth outcomes. Moreover, marine microplastics are often generated elsewhere, so the factors that produce microplastics tend to be distant from the areas of interest with birth outcomes. We address these endogeneity concerns in several ways.

First, we construct an ocean-current dynamic model to isolate exogenous variation in coastal microplastic levels originating from the open ocean. We use ocean currents to predict how microplastics from distant offshore areas (e.g. more than 200 km from any coast) can be transported to a given coastal location. Incorporating this "transported" microplastic measure into regression equation (1) helps alleviate endogeneity because the variation it exploits—microplastic fluctuations far from land, driven by ocean currents—should affect coastal infant health only via its influence on local microplastic exposure. By leveraging this plausibly random, distant-source variation, we aim to tease out the causal effect of microplastics on birth outcomes, minimizing confounding from local pollution or socioeconomic factors. The construction of this transported microplastic variable is detailed in Section 3.3.

Second, we estimate an augmented version of equation (1) that disaggregates exposure by gestational period. In place of a single in-utero exposure term (the 9-month sum), we include separate terms for each trimester of pregnancy. These trimester-specific exposure coefficients allow us to identify which stage of gestation is most sensitive to microplastic pollution, facilitating comparison with medical evidence. For example, because fetal weight gain occurs largely in the later stages of pregnancy, any effect of first-trimester exposure would be expected to be small or negligible. In addition, we include "preconception" and "postpartum" exposure terms, defined as microplastic levels in the three months before conception and the three months after birth, respectively. These serve as placebo tests: microplastic exposure outside of the pregnancy window should not affect birth weight outcomes unless the model is picking up spurious correlations. The augmented estimation equation is as follows:

$$\begin{aligned}\text{Low Birth Weight}_{i,t} = \; & \beta^{\text{Preconception}} \cdot \text{Log} \sum_{k\in[-10,-12]} \text{Microplastics}_{i,t+k} \\ & + \beta^{\text{1st Trimester}} \cdot \text{Log} \sum_{k\in[-7,-9]} \text{Microplastics}_{i,t+k} \\ & + \beta^{\text{2nd Trimester}} \cdot \text{Log} \sum_{k\in[-4,-6]} \text{Microplastics}_{i,t+k} \\ & + \beta^{\text{3rd Trimester}} \cdot \text{Log} \sum_{k\in[-1,-3]} \text{Microplastics}_{i,t+k} \\ & + \beta^{\text{Postpartum}} \cdot \text{Log} \sum_{k\in[0,2]} \text{Microplastics}_{i,t+k} \\ & + \alpha_{\text{adm1}} + \alpha_{\text{country-month}} + X_{i,t}\gamma + \varepsilon_{i,t} \quad (2)\end{aligned}$$

where the coefficients of interest are $\beta^{\text{Preconception}}$, $\beta^{\text{1st Trimester}}$, $\beta^{\text{2nd Trimester}}$, $\beta^{\text{3rd Trimester}}$, and $\beta^{\text{Postpartum}}$. We expect significant effects to emerge during pregnancy, but not before conception or after birth if the model is well-specified.

Third, our estimation sample contains data from multiple countries spanning different parts of the world. This means the characteristics of microplastic fluctuations vary significantly across locations in our sample (Figure 2), as do other environmental and economic conditions. Most importantly, what constitutes omitted factors in one area is unlikely to prevail in other countries. To the extent that we observe robust effect estimates in distinct parts of the world, it enhances our confidence that our model is capturing the genuine impact of microplastics.

3.3 Microplastic Transport Modeling

To quantify microplastics transported from the open ocean to coastal areas, we construct an oceanographic model that produces an ocean-current flow intensity matrix. Here we provide an intuitive overview of this modeling procedure and its key equations, while deferring detailed computational derivations and parameter calibrations to Appendix C for brevity.

Model. The model uses data from the Ocean Surface Current Analysis Real-time (OSCAR) product, which provides daily ocean current vectors (direction and speed) on a 0.25-degree global grid. Using these currents, we generate continuous flow trajectories (streamlines) that illustrate how water and any microplastics it carries moves through the ocean over time. We treat certain open-ocean grid cells as potential "far-sea senders" of microplastics. In our baseline specification, any ocean grid located at least 200 km offshore is considered a sender. We also experiment with using all ocean grids as senders in a robustness check. Including nearer-source grids improves the predictive coverage of coastal microplastic variation but may introduce endogeneity;

restricting to far-away sources sacrifices some predictive power in exchange for greater exogeneity of the variation. For each such sender grid i, we trace the path of a notional particle released into the ocean current and track its influence on each coastal "receiver" grid r over multiple days. Because microplastics from a distant source will take time (days or weeks) to reach a given coast, we allow for a multi-step (multi-day) transmission process rather than assuming an immediate one-day impact. In other words, using the pre-computed streamlines, we follow where the water from sender i flows on day 1, day 2, and so on, and how it progressively approaches different coastal locations. We accumulate this influence over a period of 90 days for each sender–receiver pair. This provides a downstream intensity measure capturing how strongly currents connect grid i to grid r over that interval.

Formally, we define a daily downstream flow intensity from sender i to receiver r at each step t as an exponentially decaying function of three factors: the time elapsed (which expands the area of influence), the directional misalignment between r and the current's heading from i, and the distance between the two grids. Let rad_t denote the effective search radius after t days of advection (with rad_0 equals 1 degree and growing by 0.05 degree per day to account for dispersion and trajectory uncertainty), let $|\theta|_{i\to r,d,t}$ be the absolute angle between the direction from sender to receiver and the ocean current vector at step t, and let $\mathrm{dist}_{i\to r,d,t}$ be the great-circle distance between sender and receiver after t days. We write the intensity at step t (starting on day d) as:

$$\mathrm{Current}_{i\to r,d,t} = \exp\{-\alpha \cdot \mathrm{rad}_t - \beta \cdot |\theta|_{i\to r,d,t} - \gamma \cdot \mathrm{dist}_{i\to r,d,t}\} \quad (3)$$

which decays with increasing radius, angular deviation, and distance (U.S. EPA, 2018; Phillips et al., 2021). We impose two cutoff criteria: $\mathrm{Current}_{i\to r,d,t}$ is set to zero if the receiver lies outside the radius ($d_{i\to r,d,t} > \mathrm{rad}_t$) or if the current is too misaligned ($\theta_{i\to r,d,t} > 0.4$ radian) at that step – meaning in those cases the particular receiver receives negligible influence from sender at that time. We iterate this calculation step-by-step for up to 90 steps for each sender i and for each starting day d in the sample period.[2]

[2] We use parameter values $\{\alpha, \beta, \gamma\} = \{0.8, 0.49, 0.23\}$. These numbers are empirically determined such that we would obtain a spatially continuous flow coefficient function through the successive steps, and that the directionality of the observed currents are respected through the flow coefficient representation. See Online Appendix C for more details.

Figure 3 provides a visualization: the streamline emanating from the sender (red arrow) shows the path of current flow, while the expanding circular "buffer" illustrates the growing search radius rad_t over time. The visualization also explains why both the relative angle and distance variables (θ and $dist$) have starting day and step subscripts (d and t): we track where ocean currents originate and where they move to, and we compute each receiver grid's relative angle and distances dynamically.

After computing the stepwise current intensities, we aggregate them to obtain the cumulative influence over each starting day:

$$Current_{i\to r,d} = \sum_{d+t=d} Current_{i\to r,d,t} \quad (4)$$

We then average these daily intensities across all days within each month m to produce a monthly flow intensity $Current_{i\to r,m}$. The final step is to incorporate actual microplastic concentrations. Let $MP_{i,m}$ denote the observed microplastic concentration at the sender grid i in month m. We define the transported microplastic from i to r in month m as $MP_{i\to r,m} = MP_{i,m} \cdot Current_{i\to r,m}$. This represents the amount of microplastic (in concentration units) from source i expected to arrive at coastal location r during that month, according to the ocean current propagation model. Summing over all far-sea sender grids yields the total transported microplastic exposure for coastal grid r: $\sum_i MP_{i\to r,m}$. In the econometric analysis, we use $\sum_i MP_{i\to r,m}$ as an exogenous regressor – a proxy for microplastic pollution at r that originates from distant sources (and is thus purged of local contamination biases). We can then compare the results using this transported measure to those using the local microplastic concentration $MP_{r,m}$ directly.

Validation. To validate that our transport-based measure indeed captures transported microplastics, we investigate whether coastal microplastic levels rise and fall in tandem with far-sea microplastic sources when ocean currents strongly connect the two. Specifically, we estimate a regression of coastal microplastic concentration on the interaction of sender microplastic concentration with the transport intensity, allowing the effect to vary by the strength of ocean currents. Let $MP_{r,m}$ be the microplastic concentration at coastal grid and $MP_{i,m}$ that at a far-sea grid. We run:

$$\begin{aligned} \text{Log } MP_{r,m} = {} & \beta \cdot Current_{i\to r,m} \times \text{Log } MP_{i,m} \\ & + \gamma \cdot Current_{i\to r,m} + \delta \cdot \text{Log } MP_{i,m} + \alpha_{ir} + \alpha_m + \varepsilon_{i,r,m} \end{aligned} \quad (5)$$

where $\text{Current}_{i\to r,m}$ enters the regression in decile-bin dummies (with the weakest-current decile as the omitted category), and we include sender–receiver pair fixed effects (α_{ir}) and month fixed effects (α_m). Intuitively, this specification compares, for the *same* sender–receiver pair, months when the connecting currents are stronger versus weaker. Importantly, recall that our construction of the $\text{Current}_{i\to r,m}$ values did not involve microplastics information – they are purely based on ocean currents, and so there exists no mechanical correlation in the estimation of β. The estimation results are plotted in Figure 4, panel (a). They show a clear pattern: when ocean currents from i to r are stronger, changes in the sender's microplastic level translate into larger changes in the receiver's microplastic level.

In contrast, if we replace the outcome with an unrelated pollutant – for example, the log of coastal chlorophyll concentration – the transport intensity shows no systematic effect (Figure 4, panel b). This placebo test suggests that the ocean-current model is not simply capturing general environmental co-movements, but a targeted pathway for microplastic transport.

Finally, although we built this transport model to mitigate endogeneity, it is noteworthy that *ex post* our core results are quite similar whether we use local microplastic exposure or our transported exposure measures (using either all-source or ≥200 km sources) in the regression. In other words, the data indicate that omitted-variable bias due to local factors is not as severe as we might have feared. This consistency across specifications reinforces the credibility of our identification strategy and suggests that microplastic exposure has a robust influence on infant health regardless of the measure used.

4. Results

4.1 Main Results

Figure 5 provides a first look into our main estimation results based on equation (2), linking each individual's birth outcome (i.e., whether the birth has low birth weight of below 2,500 grams at birth) to in-utero exposure to microplastic pollution. From left to right, the coefficients represent the impact of microplastic exposure over the preconception quarter, the first, second, and third trimesters, and the postpartum quarter. We are mainly interested in the effects during the *actual* pregnancy (i.e., the three trimesters). These trimester terms provide

information on which trimester exposure seems to matter the most, in a way that we can compare to the medical literature. For example, we know that fetal weight is mostly determined toward the later periods of pregnancy, and therefore, we would expect a smaller effect, if any, during the first trimester (Resnik, 2002; Cunningham et al., 2018). Similar patterns have been observed in other causal studies on the impact of prenatal exposure to pollution (e.g., Currie, Neidell, and Schmieder, 2009; Lavaine and Neidell, 2017).

The preconception and postpartum terms measure microplastic "exposure" in the 3 months prior to conception and 3 months after birth. These coefficients provide useful *placebo* tests for our model specification: we do *not* expect microplastic concentration during these periods to matter for birth outcomes, unless there are model misspecifications that lead to spurious findings. Results in Figure 5 suggest that the impact on low birth weight is most attributable to microplastic exposure during the second and third trimesters, consistent with the notion that fetal development is mostly determined in these periods. Reassuringly, we do not find significant effects from the two placebo periods.

Table 1 reports more details on the microplastic-health links. Each coefficient reported in this table represents an estimate of β from equation (1) with varying microplastic measurement, sample restrictions, and control variables. Start with column 1. Here we report estimation results using pooled data. Each coefficient corresponds to a separate regression using a different right-hand-side microplastic measurement, as indicated by the row names. The number on the top row shows that a log increase in in-utero microplastic exposure, measured directly using observed local variation at the birth's coastal location, leads to an increase in the likelihood of low birth weight by 0.287 per 1,000 births. The number on the middle row shows the estimate when we use transported microplastics as the independent variable, where the effect size is a 0.442 per 1,000 births increase in low birth weight per log increase in microplastics. The bottom row also uses transported microplastics as the independent variable but restricts contributing grids to those that are at least 200 km away from the coastal location. Here the effect size is 0.381. The bottom section of column 1 shows that this regression does not include additional environmental controls (temperature, precipitation, atmospheric aerosol, and coastal ocean chlorophyll). The mean of the dependent variable is 27.6, meaning on average the rate of low birth weight in the pooled data is 27.6 per 1,000 births. The average effect sizes from these three regressions, (0.287+0.442+0.381)/3=0.370, therefore corresponds to about a 2 percent increase in the odds of

low birth weight for a log increase in in-utero microplastic exposure. The bottom row shows that the regressions include a little over 3 million individual births.

The rest of the columns are variants of the baseline in column 1. The estimation in column 2 is identical to column 1 except that it includes additional environmental controls. Columns 3-4, 5-6, 7-8, and 9-10 report subsample estimation results for the DHS samples, the USA sample, the Brazil sample, and the Mexico sample, respectively. We find statistically significant effects for the majority of the samples and econometric specifications. On the "percent change in likelihood of low birth weight per 1 log increase in in-utero microplastic exposure" scale, the effect sizes are 1.2 percent for the USA, 1.9 percent for Brazil, 2.1 percent for Mexico, and 8.1 percent for DHS countries. We are generally underpowered with the DHS data, which features a much smaller sample size (about 10,500), and so the noisy estimates may have exaggerated the true effect size, but they are of a similar order of magnitude to the other samples. Scaled linearly, our estimate implies that about 205,800 of LBW coastal births annually worldwide can be attributed to microplastic exposure.

Our estimates using the local versus transported microplastic measures yield similar results, suggesting that the degree of endogeneity—such as omitted variable biases—may not pose a significant problem in equation (1). This is also supported by the fact that our estimates are largely unchanged after we condition on a rich set of environmental covariates.

4.2 Effect Size

To put our effect size into perspective, we compare it with established estimates from the literature on the causal effects of air pollution on low birth weight risks. Currie, Neidell, and Schmieder (2009) used natality microdata from the state of New Jersey and found that a 1 ppm increase in ambient carbon monoxide (CO) exposure during the third trimester led to an 8 percent increase in the risk of low birth weight (**an elasticity of 0.13**). Lavaine and Neidell (2017) studied a refinery strike in France that resulted in a significant reduction of sulfur dioxide (SO_2) by 15 $\mu g/m^3$, corresponding to a 33 percent reduction in low birth weight risks (**an elasticity of 0.08**). Hansen-Lewis and Marcus (2022) examined maritime shipping emission standards and showed that each 1 $\mu g/m^3$ reduction in $PM_{2.5}$ lowered low birth weight risks by 2.1 percent (**an elasticity**

of 0.19). Our estimated elasticity of 0.02 is smaller than these figures, but it is still substantial enough to suggest that microplastics pose a non-negligible risk to fetal health.

It is perhaps also helpful to consider research that does not study the impact of specific pollutants, but rather examines major air and water pollution events and documents their reduced-form effects on low birth weight risks. These studies may be helpful in putting effect sizes into perspective. Currie and Walker (2011) show that the introduction of E-ZPass reduces low birth weight risks by **11.8 percent** within 2 km of toll plazas. Imelda (2020) analyzes a large-scale clean cooking fuel program in Indonesia, which reduces low birth weight incidence by **19.7 percent**. Dave and Yang (2022) show that a lead-in-water crisis in Newark led to an **18 percent** increase in low birth weight risks. DiSalvo and Hill (2024) study the impact of drinking water contamination in Pennsylvania, showing that moving a mother from the 10th to the 90th percentile of water contamination increases low birth weight risk by 12 percent. Currie, Greenstone, and Meckel (2017) and Hill and Ma (2022) study the hydraulic fracking boom in Pennsylvania and show that for mothers living within 1 km of active wells, there is a **12–25 percent** increase in the probability of low birth weight, likely due to a combination of increased air and water pollution exposure during the in-utero period. Once again, the estimated impact of microplastics is small but not negligible relative to these major shocks, and given the continuing growth in plastic use, this suggests the importance of studying this emerging source of pollution further.

4.3 Confounders and Robustness

We examine several potential alternative explanations for our findings and conduct additional robustness checks.

Mother Characteristics. We first test whether our findings may be explained by changes in composition of pre-determined mother characteristics, in the spirit of a balancing test. We repeat our main regression from equation (1), but now use various maternal and location characteristics as the outcome variables: the mother's age; an indicator for the mother having less than a high school education; the distance to the coast; the city's average temperature and precipitation during the pregnancy; the city's per capita GDP; and the city's average nightlight

index during the pregnancy. While per capita GDP captures a city's overall level of development, the nightlight index better reflects economic conditions during the pregnancy period.

We summarize these results in Table 2. Across all outcome variables and using three different microplastic exposure measures, we find no evidence that higher in-utero microplastic exposure is associated with any significant differences in maternal characteristics.

Correlated Pollutants. Our identification strategy relies on variation in ocean currents to predict how much microplastic pollution from the open sea reaches coastal areas. One potential concern is that ocean currents could also carry other pollutants that independently affect infant health.

Two pieces of evidence alleviate this concern. First, as shown in Section 3.3 (and Figure 4, panel b), our ocean transport model suggests no evidence that microplastic pollution in the open ocean passes through to coastal chlorophyll levels—a proxy for general ocean pollution.

Second, we further conduct a permutation test. We reconstruct the transported microplastic exposure using actual ocean currents (as described in Section 3.3), but randomly shuffle the distribution of microplastics in the open sea, i.e., assign microplastic concentrations to ocean grid cells at random. For each such shuffled dataset, we re-estimate the main regression equation (1) to obtain a placebo coefficient. Conceptually, these placebo estimates represent the effect if ocean currents were transporting some arbitrary "placebo" pollutant instead of microplastics. Figure 6 shows the results of this exercise: the histogram plots the distribution of 1,000 placebo coefficients, and the vertical line indicates the actual coefficient using the true microplastic data. The actual estimate lies well outside the 95% range of the placebo distribution.

Alternative Infant Health Measures. Our main analysis focuses on low birth weight as the primary proxy for poor infant health. In Table 3, we report a series of regressions in which we substitute alternative infant health measures of varying severity for the outcome in equation (1). These include birth weight (in grams); an indicator for very low birth weight (below 1,500 g); gestational length (in weeks); an indicator for preterm birth (gestation < 37 weeks); and an indicator for very preterm birth (gestation < 32 weeks). We also construct a summary index of adverse infant health, which aggregates all of these measures along with our original low birth weight indicator. To create this index, we multiply birth weight and gestational length by –1 (so

that higher values correspond to worse outcomes), standardize each variable to mean 0 and standard deviation 1, and then take the average.

The results in Table 3 indicate that in-utero microplastic exposure tends to impair infant health across multiple measures. Moreover, the effects appear larger for more severe outcomes. For example, we find no statistically significant impact on average birth weight or gestational length, but we do observe strong effects on the incidence of low birth weight, very low birth weight, and very preterm birth. This pattern suggests that microplastic exposure disproportionately affects the lower tail of the birth outcome distribution.

Other Robustness Specifications. We further present a series of robustness specifications. Appendix Figure 3 reports the dose-response function estimation, allowing the LBW-microplastics estimates to vary based on the magnitude of the microplastic shocks, shown as residual log microplastics change on the horizontal axis.[3] The graphical patterns suggest a generally linear effect, indicating that the increase in LBW risk is roughly proportional to the order of magnitude of in-utero exposure to microplastics.

Our main analysis restricts the sample to mothers residing within 200 km of the coastline. In Appendix Figure 4, we show results using alternative coastal distance cutoffs from 100 km up to 500 km (in 100 km increments). As expected, the estimated effects diminish as we include mothers living farther inland, but our main findings are qualitatively insensitive to the bandwidth choice.

Appendix Table 1 presents a range of alternative specifications, varying features such as whether co-pollutant controls are included, whether we use admin level-1 or level-2 region fixed effects, and whether we use uniform month fixed effects or month fixed effects that vary by country. These specification checks yield results comparable to our baseline.

[3] We take equation (1), residualize both the LBW variable and the in-utero microplastics variable using the full set of fixed effects, and then plot the residualized variables in a decile bin scatterplot. The slope of the superimposed linear fit corresponds to the point estimates in Table 1, column 1.

4.4 Channels

How might in-utero exposure to microplastics cause low birth weight? In this section, we use additional data to explore two hypothesized pathways through which microplastic exposure could impact birth outcomes.

Access to Seafood. The first potential channel is via seafood consumption. Microplastics accumulate in marine organisms and biomagnify up the food chain, ultimately entering the human body when contaminated seafood is consumed. An intuitive test of this channel is to check whether microplastics have a larger impact on low birth weight in areas with higher seafood consumption. We construct two proxies for seafood consumption. The first is derived from Nielsen retail scanner data: county-level per capita seafood spending (available only for the USA). The second comes from Global Fishing Watch: the average fishing hours in an area during 2017–2018, which serves as an indicator of local seafood availability (available globally). In Table 4, we re-estimate our baseline model (Table 1, column 1) and include interactions between microplastic exposure and each of these seafood-consumption proxies separately. We find no evidence of substantial heterogeneity in microplastics' health effects between areas with high versus low seafood consumption.

Another potential pathway is via seafood trade. A large share of the seafood consumed in the U.S. is imported, so U.S. birth outcomes could be influenced by microplastic pollution in exporting countries rather than by local microplastics. To test this trade channel, we use data on U.S. seafood imports from USA Trade Online (U.S. Census Bureau). Our key variable is the import value of live seafood (HS codes 0301 and 0302) at the state–exporting country–month level. For each birth record in state i in month m, we merge it with seafood exporters' microplastics, a weighted average of microplastics near each exporting country j in month m. This treatment measure is computed in three steps: i) We retrieve microplastics near each exporting country-month using a 200km buffer around country shorelines, i.e. $MP_{j,m}$; ii) Weight is calculated as importing value of live seafood, HS code under 0301 and 0302, at the state-country-month level, i.e. $ImSeafood_{i,j,m}$; iii) To aggregate over exporting countries, we calculate the weighted average: $\sum_j (ImpSeafood_{i,j,m} * MP_{j,m}) / \sum_j ImpSeafood_{i,j,m}$.

In Appendix Table 2, we include both local microplastic exposure and the constructed seafood-import microplastic measure in the regression (a horse race between the two). The

coefficient on local microplastics remains large, precisely estimated, and similar in magnitude to the corresponding estimates in Table 1 (columns 5–6), confirming that the relationship between local microplastic pollution and low birth weight is robust. By contrast, the coefficient on exporters' microplastics is near zero and statistically insignificant, suggesting little evidence that microplastic pollution in exporting countries affects birth outcomes in the importing states.

That said, the absence of detectable heterogeneity or exporter effects does not necessarily imply that microplastic exposure through seafood has no health consequences. For example, in-utero microplastic exposure might lead to other adverse outcomes manifesting later in life, even if birth weight at delivery is unaffected. Another possibility is that fishing efforts endogenously shift or product safety standards mitigate microplastic contamination in seafood, thereby limiting observable health effects. More scientific and econometric research is needed on the potential for mother-to-child transmission of microplastics, and on how microplastic accumulation in the fetus may impact short- and long-term health outcomes.

Aerosolization. The second channel we examine is microplastic aerosolization. Due to their small size, many microplastic particles can become airborne under suitable atmospheric conditions. Once aerosolized, microplastics can be inhaled and enter the body through the respiratory tract and, like other fine particulates, potentially penetrate into human organs. This pathway could be important, since prior studies have causally linked airborne particulate pollution to worse infant health outcomes (e.g., Jayachandran, 2009; Currie and Schwandt, 2016; Alexander and Schwandt, 2022; Hansen-Lewis and Marcus, 2022).

To test the aerosolization channel, we use satellite-based monthly aerosol optical depth measurements for the coastal locations in our sample. We then examine whether months with higher coastal microplastic concentrations also have higher aerosol pollution. To help isolate causality, we also leverage the transported microplastic measure from the open ocean (as defined earlier) to predict coastal aerosol pollution, exploiting variation driven by ocean currents. The estimation equation is:

$$\text{Log Aerosol}_{i,t} = \beta \cdot \text{Log Microplastics}_{i,t} + \alpha_i + \alpha_{\text{country}-\text{month}} + \varepsilon_{i,t} \quad (6)$$

where $\text{Log Aerosol}_{i,t}$ and $\text{Log Microplastics}_{i,t}$ are logged aerosol optical depth and microplastics concentration at coastal grid i in month t, respectively. α_i and $\alpha_{\text{country}-\text{month}}$ are grid and

country-by-month fixed effects. $\varepsilon_{i,t}$ is the error term and we cluster standard errors at the grid level.

Because vertical air movement is a key driver of aerosolization, we also estimate a model with an interaction between microplastic concentration and the local sea-surface evaporation rate (evaporation is largely temperature-driven and, conditional on seasonality, plausibly exogenous). The estimation equation is:

$$\text{Log Aerosol}_{i,t} = \beta \cdot \text{Log Microplastics}_{i,t} + \theta \cdot \text{Log Evaporation}_{i,t} + \delta \cdot \text{Log Microplastics}_{i,t} \times \text{Log Evaporation}_{i,t} + \alpha_i + \alpha_{\text{country-month}} + \varepsilon_{i,t} \quad (7)$$

and our hypothesis is that higher rate of evaporation exacerbates the degree of microplastic aerosolization. That is, the δ estimate in equation (7) should have a positive sign.

Table 5 presents results consistent with aerosolization: higher microplastic concentrations are associated with greater coastal aerosol pollution (columns 1, 3, 5), and this effect is stronger when evaporation rates are high (columns 2, 4, 6). These findings highlight the need to further understand the biological mechanisms by which microplastic particles and fibers affect respiratory and other health outcomes.

To gauge the quantitative importance of aerosolization, we assess how much of microplastics' overall effect on low birth weight this mechanism might explain. Specifically, in column 3 of Table 5, we find that each log increase in coastal microplastics concentration is associated with approximately a 4.5 percent rise in coastal particulate pollution levels. Using the elasticity estimate of 0.19 from Hansen-Lewis and Marcus (2022), who investigate the causal effect of fine particulate pollution on LBW in U.S. coastal areas, a 4.5 percent increase in airborne particulates would lead to an expected 4.5 percent * 0.19=0.86 percent increase in LBW. This increase accounts for roughly **54 percent** of the effect size we estimate in our primary LBW-microplastics model (Table 1, column 1), suggesting that aerosolization is a first-order channel underlying the impact of microplastics on low birth weight.

The interactive effect between sea surface evaporation and microplastic aerosolization also implies that population exposure to airborne microplastic pollution is expected to rise as climate change continues to cause a warm up of ocean temperature and evaporation (Abraham et al., 2013; Trenberth, 2011).

5. Conclusion

Plastic pollution has long been recognized as an environmental threat, but direct evidence of its impact on human health has been scarce. This study begins to fill that gap by demonstrating a measurable effect of microplastic exposure on infant health. By exploiting natural variation in ocean currents to isolate exogenous microplastic exposure, our analysis provides the first causal evidence that in-utero exposure to microplastics elevates the risk of low birth weight.

An important finding is that airborne transmission of microplastics likely plays a significant role in this health impact. We show that higher microplastic levels in coastal waters lead to increases in local particulate air pollution, consistent with microplastic particles becoming aerosolized and inhaled. This aerosol pathway appears to explain a substantial portion of the overall effect on low birth weight that we document. In contrast, we find little evidence that microplastics affect birth outcomes through the food chain (for example, via consumption of contaminated seafood), at least in terms of birth weight in the populations and time frame we study. This does not preclude other health damages from ingestion, but it suggests that the most immediate threat of microplastics to fetal health may operate through air pollution. Importantly, our results imply that the health burden of microplastic pollution may grow in the future: as climate change warms ocean surfaces and increases evaporation, the aerosolization of microplastics – and hence human exposure to them – is likely to intensify.

Our findings carry significance for both science and policy. From a scientific perspective, the evidence that microplastics in the environment can harm human health validates warnings raised by environmental and medical researchers. We contribute to a growing interdisciplinary literature by providing macro-level, population-based evidence to complement laboratory and clinical studies that have raised alarms about microplastics. Methodologically, we illustrate how high-resolution environmental data combined with natural experiment techniques can uncover previously undetected health externalities of pollution. For policymakers, these results highlight an emerging public health concern. Microplastic pollution should be viewed not only as an ecological problem but also as a potential contributor to poor health in communities. Policymakers may need to incorporate microplastics into air quality and water quality standards, monitor their levels more closely, and develop strategies to mitigate their release into oceans and atmosphere. Measures such as improved wastewater filtration, reductions in single-use plastics,

and better management of plastic waste could yield human health benefits in addition to environmental gains.

There are several avenues for future research. One is to investigate the long-run and life-course consequences of early-life microplastic exposure. While low birth weight is a critical predictor of infant health, exposed infants might suffer other subtler or longer-term effects that our study could not observe. Further work is also needed to explore health outcomes beyond birth weight, such as cognitive development or respiratory conditions in children, to fully capture the impact of microplastic pollution. In addition, our null results on the ingestion pathway suggest that future studies should examine under what conditions microplastic-contaminated food webs might impair health, and whether current food safety or behavioral adaptations are mitigating those risks. Finally, as microplastics are a global pollutant that travels across borders via oceans and the atmosphere, international policy responses and cooperative mitigation efforts warrant exploration.

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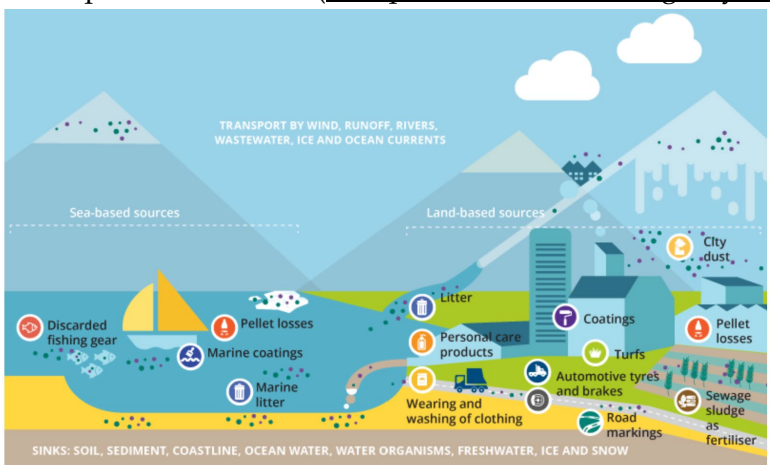
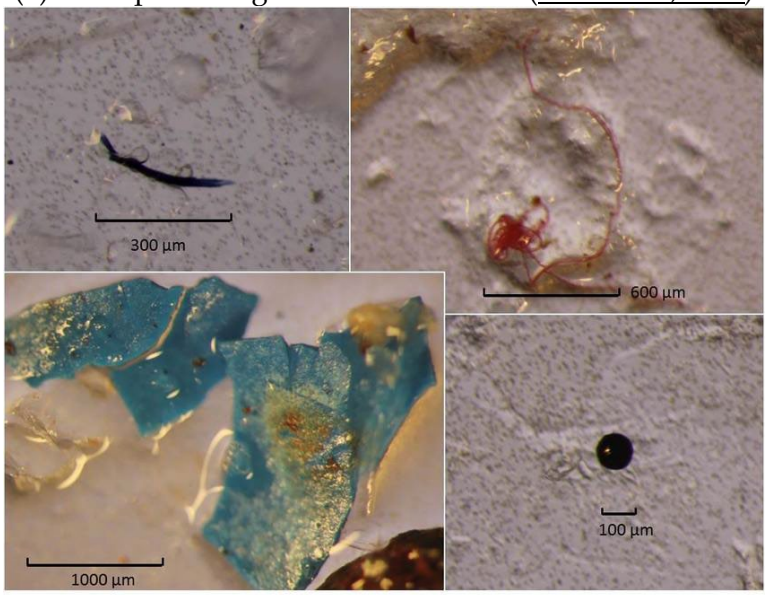
Figure 1. Plastic Pollution Sources, Ambient Presence, and Exposure

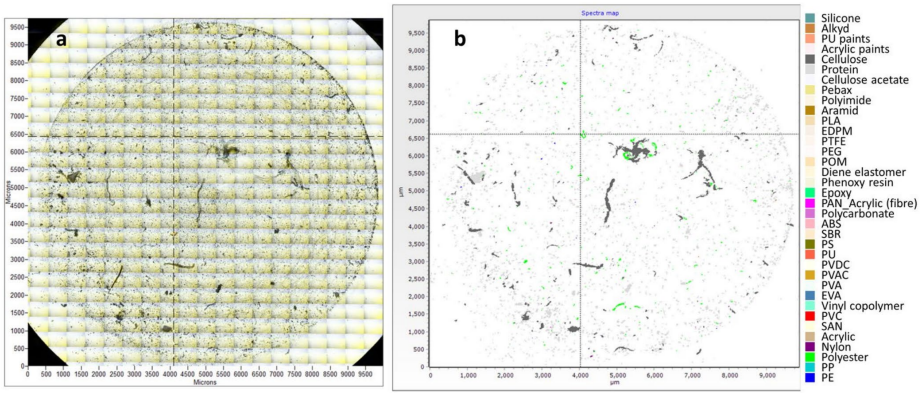
(a) Plastic pollution sources (European Environment Agency, 2022)



(b) Microplastic ingestion in fish larvae (Steer et al., 2017)

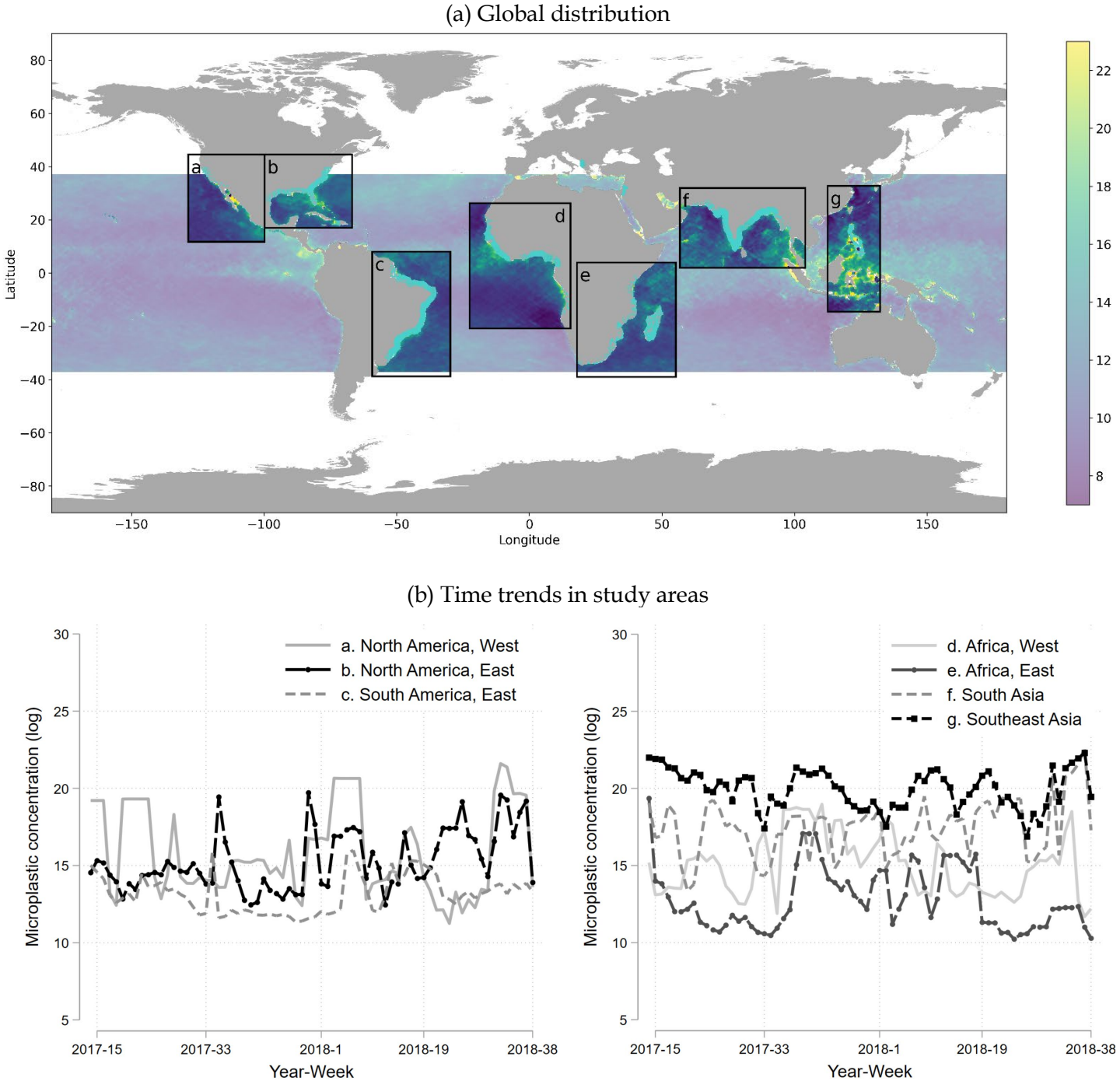


(c) Airborne microplastic (Vianello et al., 2019)



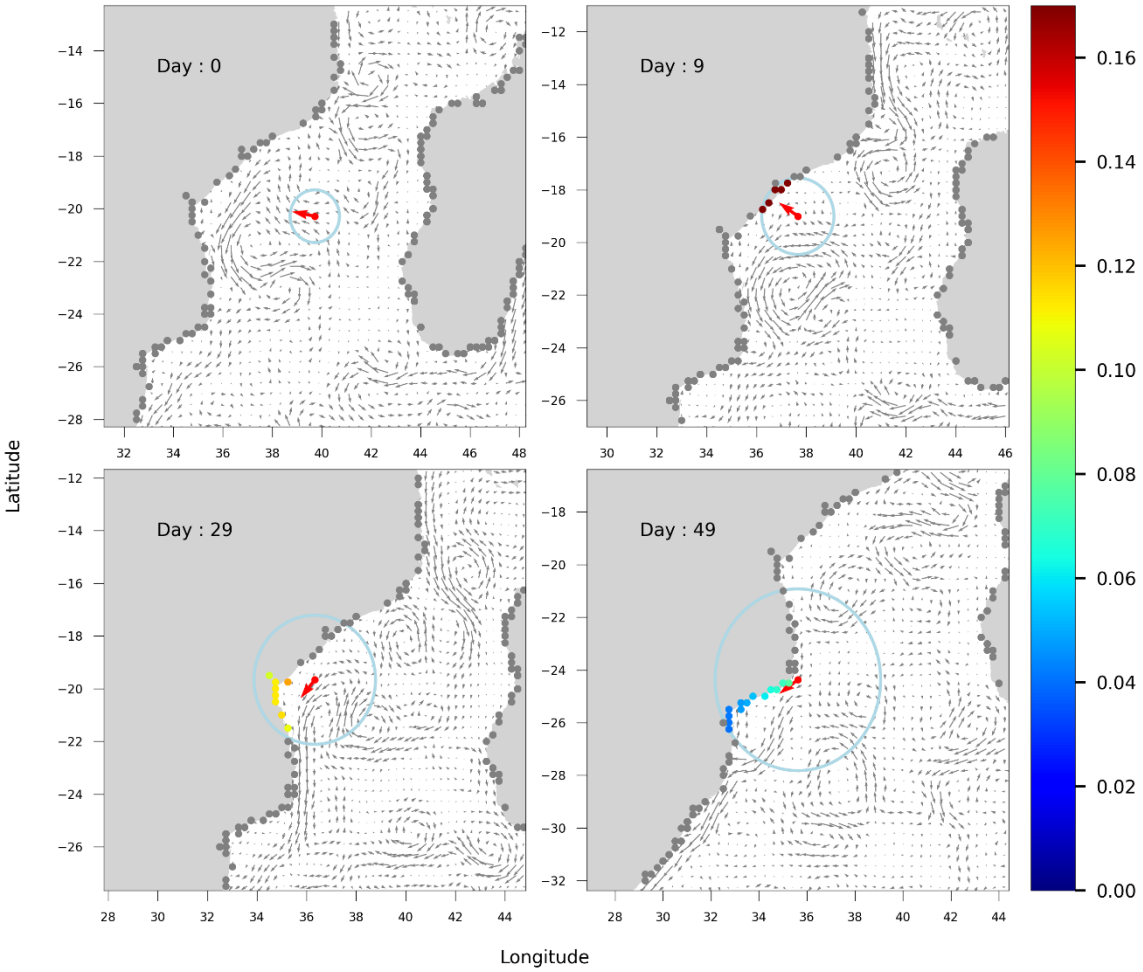
Notes: Panel (a) shows sources of plastic pollution emissions. Panel (b) shows categorization of plastic particles and potential marine animal exposure. Panel (c) shows a microscopic illustration of airborne microplastic particles.

Figure 2. Remote-Sensing Microplastic Measurement: Summary



Notes: Panel (a) shows geographic distribution of microplastic concentration from the remote-sensing measurement. Panel (b) reports average microplastic concentration trends in seven selected regions where our study samples concentrate.

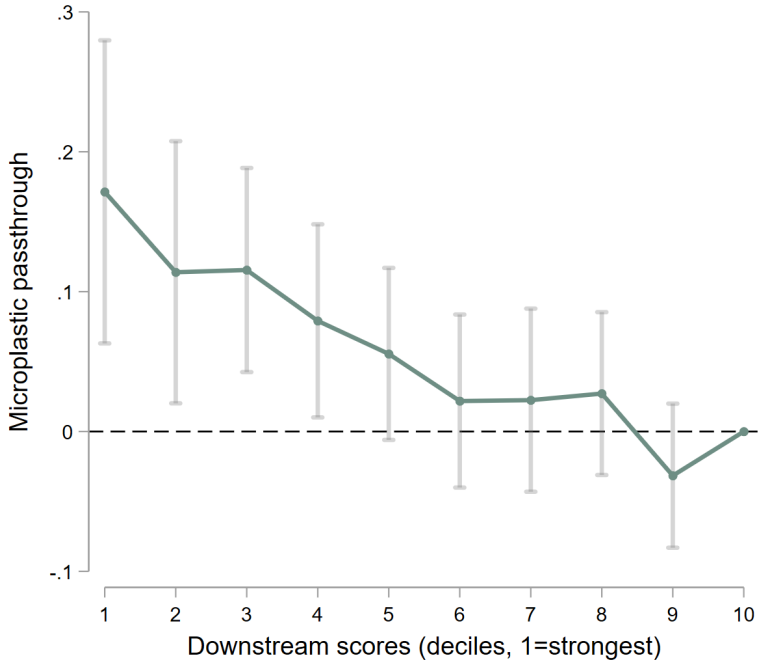
Figure 3. Transported Microplastic Modeling



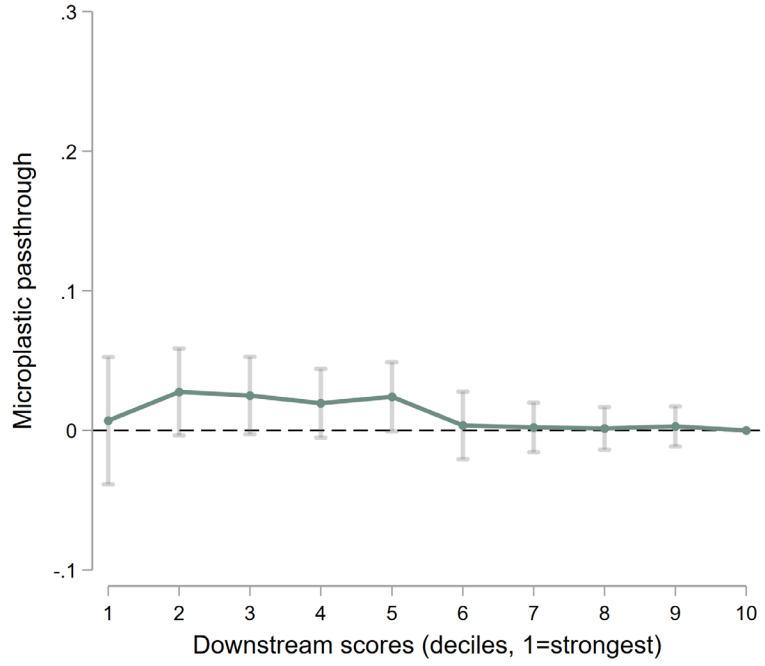
Notes: This figure provides an illustration of the transport modeling that tracks coastal variation in microplastic exposure due to microplastic presence from sea surface afar. Shaded gray areas represent land. Dots represent coastal locations. Panels track a source grid and show its location (red arrow), area of influence (blue circle), and coefficient-of-influences (colored dots) at four different time steps.

Figure 4. Passthrough of Microplastic from Ocean to Coastal Locations by Downstream Score Deciles

(a) Open-sea microplastics to coastal microplastics passthrough

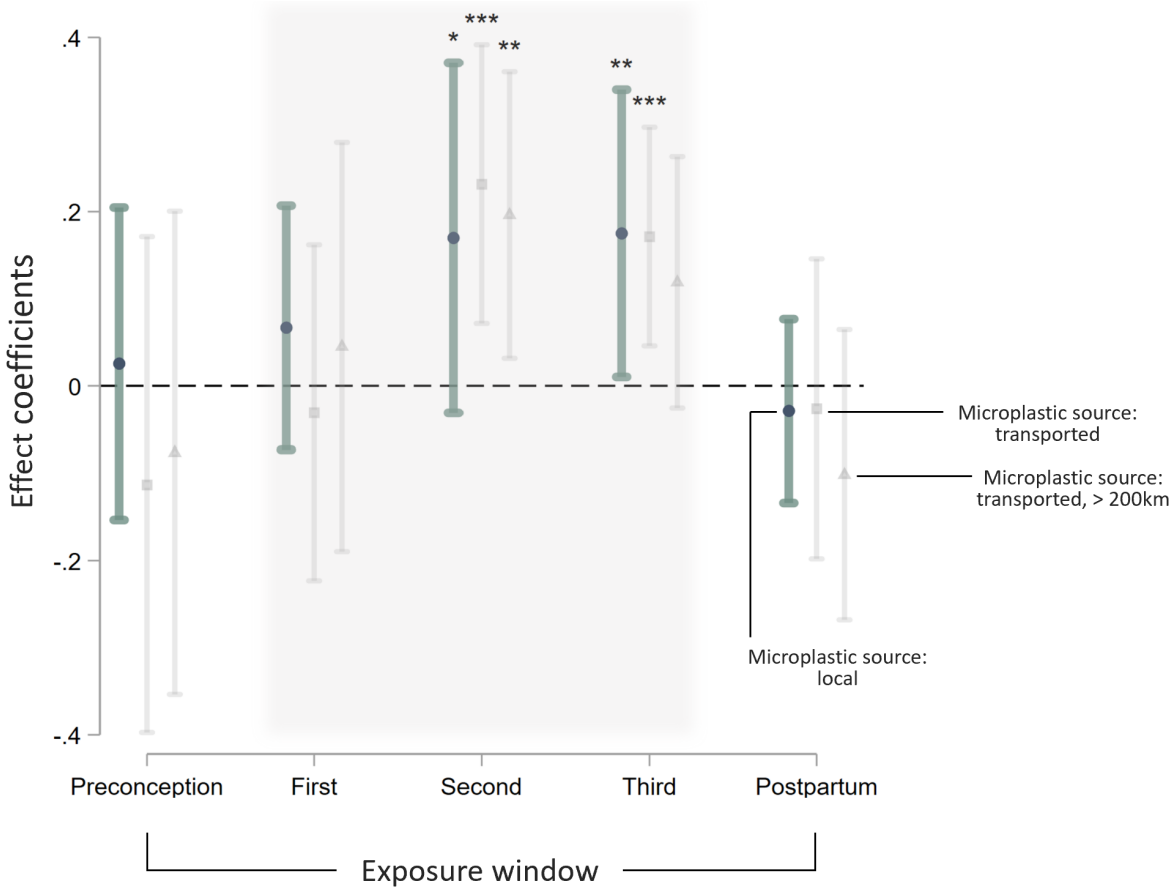


(a) Open-sea microplastics to coastal chlorophyll passthrough (placebo test)



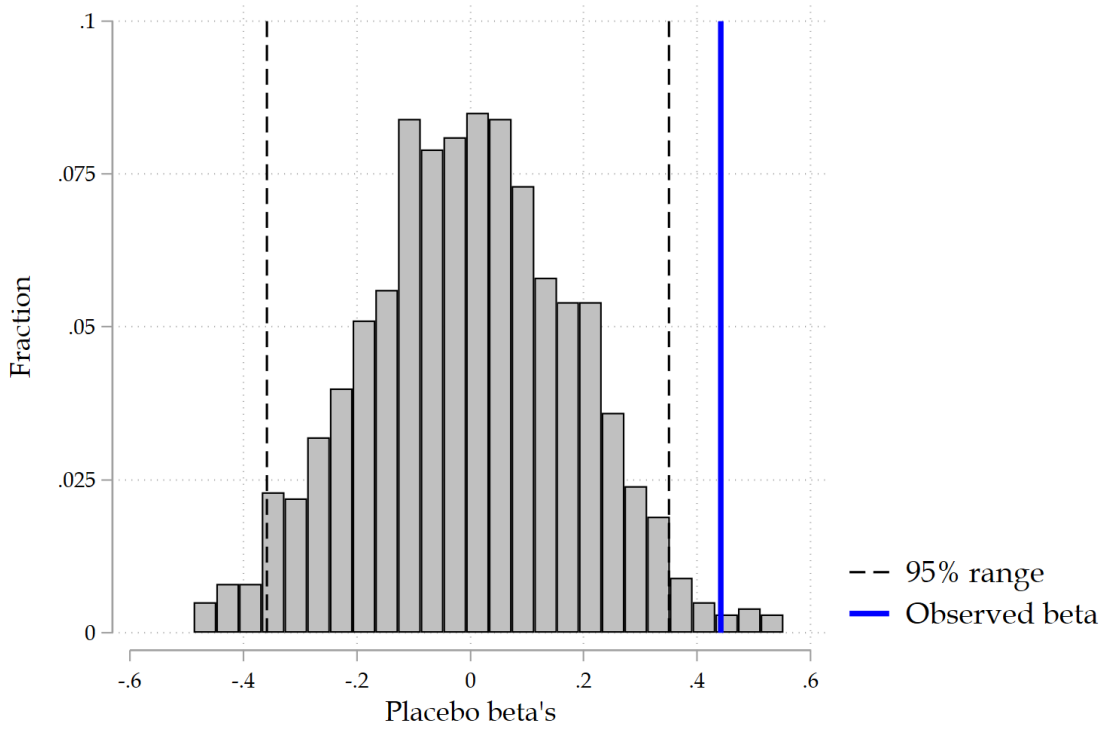
Notes: This figure displays coefficients from regressions estimating the passthrough of microplastics from open-sea locations to coastal locations, with the effect allowed to vary by downstream score decile based on our ocean current model (decile 1 = strongest downstream flow). Panel (a) shows the passthrough from upstream open-sea microplastic concentrations to coastal microplastic concentrations. Panel (b) provides a placebo test, where we replace the outcome with coastal chlorophyll levels. Vertical lines indicate 95% confidence intervals.

Figure 5. The Effect of In-Utero Microplastic Exposure on Low Birth Weight



Notes: This figure shows coefficient estimates from three separate regressions looking at the impact of in-utero microplastic exposure on incidence of low birth weight. The regressions use different microplastic measurement (circles for local microplastic source, squares for transported source, and triangles for transported source with a 200km buffer) but are otherwise with identical specification. "Preconception" corresponds to microplastic exposure during the 3-month period prior to conception, and "Postpartum" corresponds to exposure during the 3-month periods after birth. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Figure 6. Permutation Test on the Effect of In-Utero Microplastic Exposure on Low Birth Weight: Shuffling Open-Sea Microplastic Distribution while Holding Ocean Currents Fixed



Notes: This figure presents a permutation test on whether the observed effect of in-utero exposure to transported microplastics on low birth weight (LBW) could arise from generic ocean transport patterns – which can moving arbitrary environmental hazards in addition to true microplastic concentrations. We hold the ocean current system fixed but randomly reshuffle the spatial distribution of open-sea microplastic concentrations across grid cells. We then recompute transported coastal microplastic exposure using the reshuffled values and re-estimate the LBW effect using the same regression specification. The histogram shows the distribution of placebo coefficients across reshuffled samples. The blue vertical line marks the actual coefficient estimate using the true microplastic distribution.

Table 1. The Effect of In-Utero Exposure to Microplastics on Low Birth Weight

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Pooled data		DHS sample		USA sample		Brazil sample		Mexico sample	
Log microplastics (local)	0.287**	0.321***	5.813*	4.210	0.314*	0.582***	0.306	0.520**	0.084	0.215
	(0.111)	(0.111)	(3.418)	(2.769)	(0.156)	(0.122)	(0.215)	(0.216)	(0.350)	(0.480)
Log microplastics (transported)	0.442***	0.443***	5.511	0.334	0.382***	0.376***	0.588	0.503	0.462***	0.605***
	(0.078)	(0.072)	(8.401)	(6.975)	(0.078)	(0.064)	(0.352)	(0.395)	(0.113)	(0.098)
Log microplastics (transported from > 200km)	0.381***	0.373***	11.291	6.116	0.252***	0.230***	0.780***	0.732**	0.367***	0.520***
	(0.077)	(0.076)	(7.327)	(6.969)	(0.059)	(0.062)	(0.248)	(0.272)	(0.117)	(0.126)
Co-pollutants controls		✓		✓		✓		✓		✓
FEs: admin 1 region	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
FEs: country × month of sample	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Dep. var. mean	27.6	28.0	68.6	68.0	30.6	30.6	30.2	30.2	18.2	17.6
Observations	3,015,121	2,830,110	10,545	10,320	934,081	934,081	1,344,769	1,332,852	725,726	552,857

Notes: Each cell is a separate regression. In each regression, the dependent variable is a dummy for low birth weight multiplied by 1,000. Independent variable is a type of microplastic exposure measurement (local, transported, or transported from grids over 200km away). All regressions control for GADM admin level-1 subcountry region fixed effects and country by month-of-sample fixed effects. "Co-pollutants controls" include logged average temperature, precipitation, aerosol pollution, and coastal chlorophyll levels over the course of pregnancy. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Table 2. The Effect of In-Utero Exposure to Microplastics on Maternal Characteristics

	(1) Mother's age (years)	(2) Mother's education (< HS)	(3) Distance to coast (log)	(4) City temp (log)	(5) City precip (log)	(6) City GDP pc (log)	(7) City nightlight (index)
Log microplastics (local)	0.010	-0.000	-0.004	0.007	-0.034	-0.015	0.264
	(0.036)	(0.002)	(0.028)	(0.007)	(0.034)	(0.015)	(0.262)
Log microplastics (transported)	-0.003	-0.001	-0.008	0.001	0.003	0.002	0.071
	(0.031)	(0.001)	(0.015)	(0.004)	(0.023)	(0.022)	(0.149)
Log microplastics (transported from > 200km)	-0.015	-0.002**	0.003	0.000	-0.001	0.001	0.127
	(0.024)	(0.001)	(0.008)	(0.002)	(0.016)	(0.017)	(0.138)
Co-pollutants controls	✓	✓	✓	✓	✓	✓	✓
FEs: admin 1 region	✓	✓	✓	✓	✓	✓	✓
FEs: country × month of sample	✓	✓	✓	✓	✓	✓	✓
Dep. var. mean	27.4	0.62	3.73	3.00	-5.97	9.96	3.84
Observations	3,015,306	3,015,312	3,015,312	3,015,267	3,015,267	2,803,596	2,919,552

Notes: Each cell is a separate regression. Dependent variables are: mother's age (column 1); an indicator for mother's education attainment being less than high school (column 2); distance to coast (column 3); average of city's air temperature during pregnancy (column 4); average of city's precipitation during pregnancy (column 5); city's GDP per capita (column 6); city's nighlight index (column 7). Independent variable is a type of microplastic exposure measurement (local, transported, or transported from grids over 200km away). All regressions control for GADM admin level-1 subcountry region fixed effects and country by month-of-sample fixed effects. "Co-pollutants controls" include logged average temperature, precipitation, aerosol pollution, and coastal chlorophyll levels over the course of pregnancy. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Table 3. The Effect of In-Utero Exposure to Microplastics on Alternative Measures of Infant Health

	(1) Birthweight (grams)	(2) Low birthweight	(3) Very Low birthweight	(4) Gestation (weeks)	(5) Preterm birth	(6) Very preterm birth	(7) Summary index
Log microplastics (local)	-4.673***	0.321***	0.197***	-0.013*	-0.340	0.115**	0.017***
	(1.278)	(0.111)	(0.062)	(0.007)	(0.640)	(0.058)	(0.006)
Log microplastics (transported)	-0.712	0.443***	0.179***	-0.004	-0.015	0.204***	0.009*
	(0.921)	(0.072)	(0.055)	(0.005)	(0.390)	(0.052)	(0.005)
Log microplastics (transported from > 200km)	-0.213	0.373***	0.161***	-0.010	0.751	0.256***	0.012**
	(0.575)	(0.076)	(0.050)	(0.007)	(0.646)	(0.068)	(0.006)
Co-pollutants controls	✓	✓	✓	✓	✓	✓	✓
FEs: admin 1 region	✓	✓	✓	✓	✓	✓	✓
FEs: country × month of sample	✓	✓	✓	✓	✓	✓	✓
Dep. var. mean	3,209	28.0	12.7	38.6	103.2	14.7	0.01
Observations	2,830,110	2,830,110	2,830,110	2,716,398	2,716,398	2,716,398	2,830,301

Notes: Each cell is a separate regression. Dependent variables are: birth weight in grams (column 1); an indicator for low birth weight (<2,500 grams), multiplied by 1,000 (column 2); very low birth weight (<1,500 grams), multiplied by 1,000 (column 3); gestational length in weeks (column 4); an indicator for preterm birth (<37 weeks), multiplied by 1,000 (column 5); and an indicator for very preterm birth (<32 weeks), multiplied by 1,000 (column 6). Column 7 reports a summary index that aggregates columns 1–6. We multiply birth weight and gestational length by −1 (so that higher values indicate worse outcomes), standardize each variable to have mean 0 and standard deviation 1, and take their average. Independent variable is a type of microplastic exposure measurement (local, transported, or transported from grids over 200km away). All regressions control for GADM admin level-1 subcountry region fixed effects and country by month-of-sample fixed effects. "Co-pollutants controls" include logged average temperature, precipitation, aerosol pollution, and coastal chlorophyll levels over the course of pregnancy. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Table 4. Potential Channel 1: Access to Seafood

	(1)	(2)	(3)	(4)	(5)	(6)
Log microplastics	0.283	0.275**	0.389***	0.355***	0.273***	0.236*
	(0.189)	(0.111)	(0.090)	(0.116)	(0.071)	(0.135)
Log microplastics × Log seafood spending	-0.102		-0.025		0.100	
	(0.175)		(0.068)		(0.070)	
Log microplastics × Log fishing hours		-0.006		0.014		0.006
		(0.083)		(0.056)		(0.045)
Microplastics source	local	local	transported, all grids	transported, all grids	transported, >200km	transported, >200km
Observations	914,770	1,944,369	914,114	1,977,857	914,114	1,977,857

Notes: Each column is a separate regression. In each regression, the dependent variable is a dummy for low birth weight multiplied by 1,000. "Log seafood spending" is logged US county 2017-2018 average seafood consumption per capita measured from Nielsen scanner data (columns 1, 3, and 5). "Log fishing hours" is logged 2017-2018 average fishing hours measured from the Global Fishing Watch automatic identification system data (columns 2, 4, and 6). All regressions control for GADM admin level-1 subcountry region fixed effects and country by month-of-sample fixed effects. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Table 5. Potential Channel 2: Microplastic Aerosolization

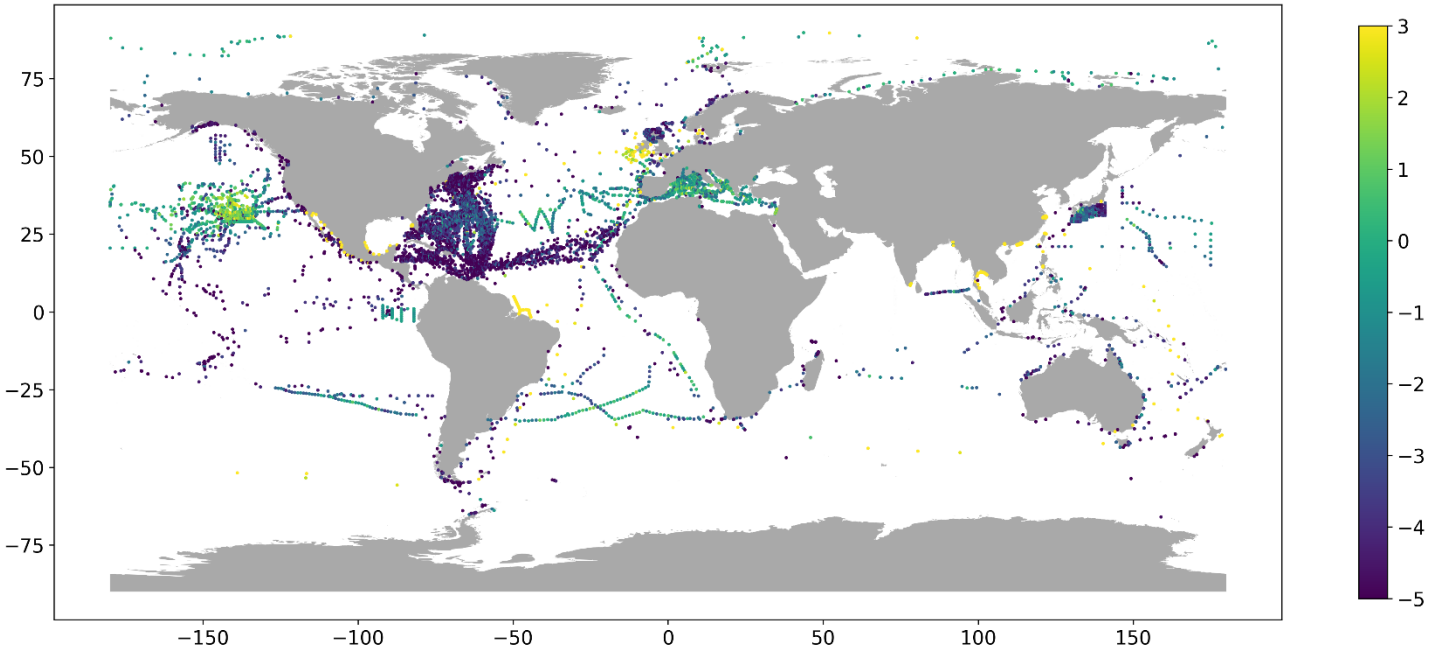
	(1)	(2)	(3)	(4)	(5)	(6)
Log microplastics	0.0217***	0.0143***	0.0451***	0.0371***	0.0435***	0.0358***
	(0.0006)	(0.0007)	(0.0010)	(0.0011)	(0.0009)	(0.0011)
Log microplastics × Log evaportation		0.0136***		0.0113***		0.0071***
		(0.0009)		(0.0016)		(0.0014)
Microplastics source	local	local	transported, all grids	transported, all grids	transported, >200km	transported, >200km
Observations	143,042	83,808	142,608	83,650	141,773	83,297

Notes: Each column is a separate regression. In each regression, the dependent variable is logged aerosol optical death measured at the coastal grid. "Log evaporation" is logged rate of evaporation measured at the coastal grid. All regressions control for grid fixed effects and country by month-of-sample fixed effects. Standard errors are clustered at the grid level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

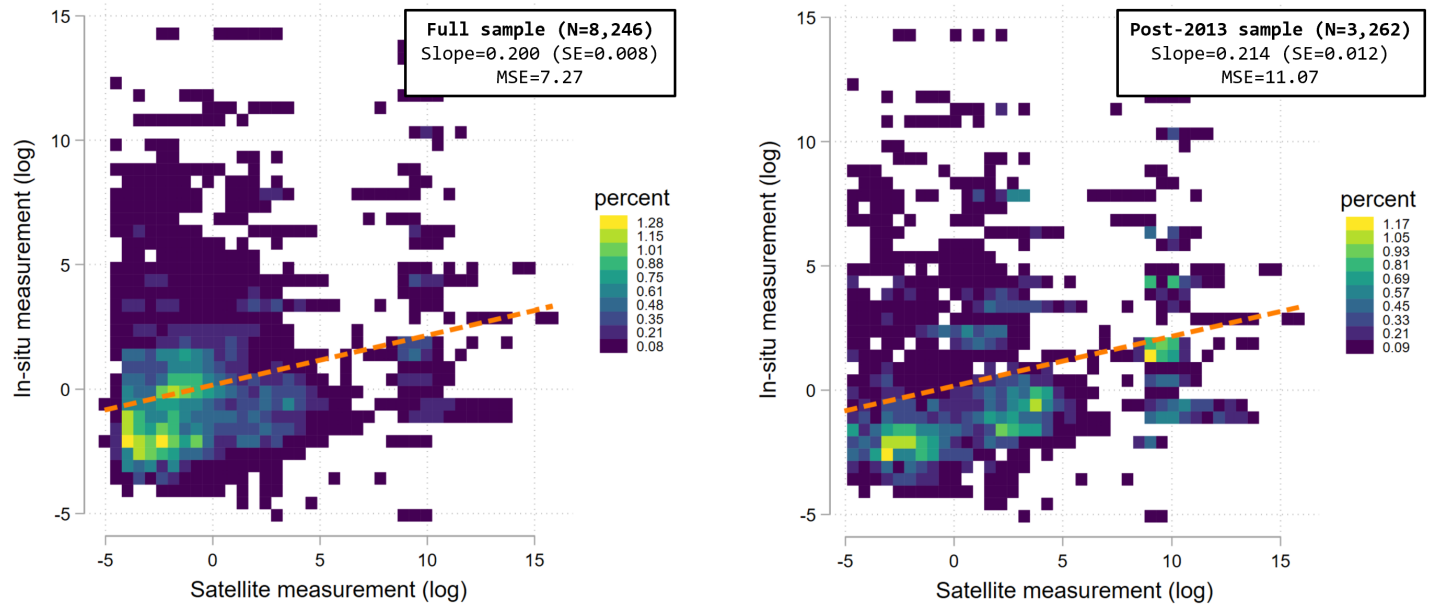
Appendix A. Supplementary Figures and Tables

Appendix Figure 1. Remote-Sensing Microplastic Measurement: Validation

(a) In-situ microplastic sampling locations



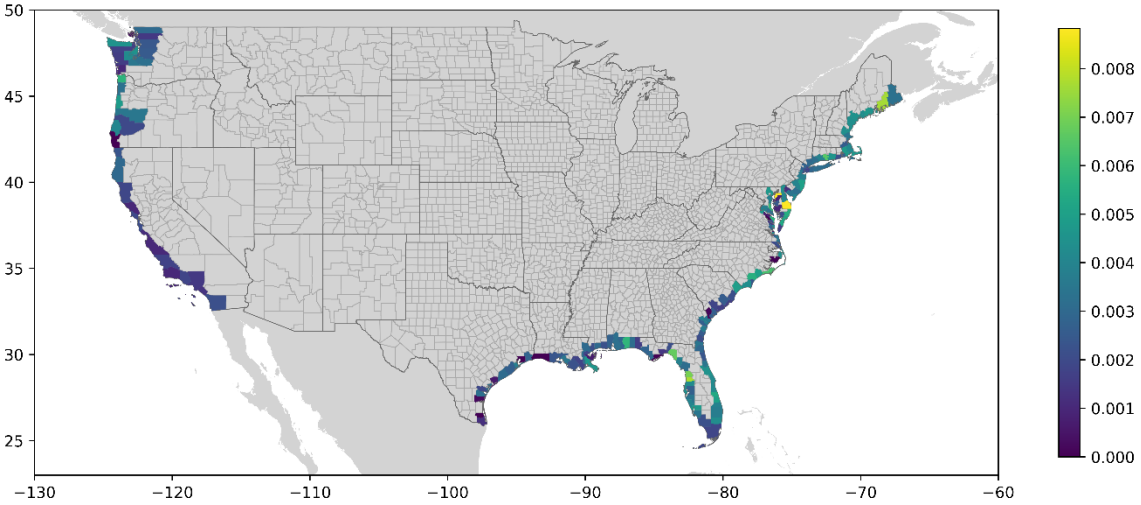
(b) Correlation between in-situ and remote-sensing measurements



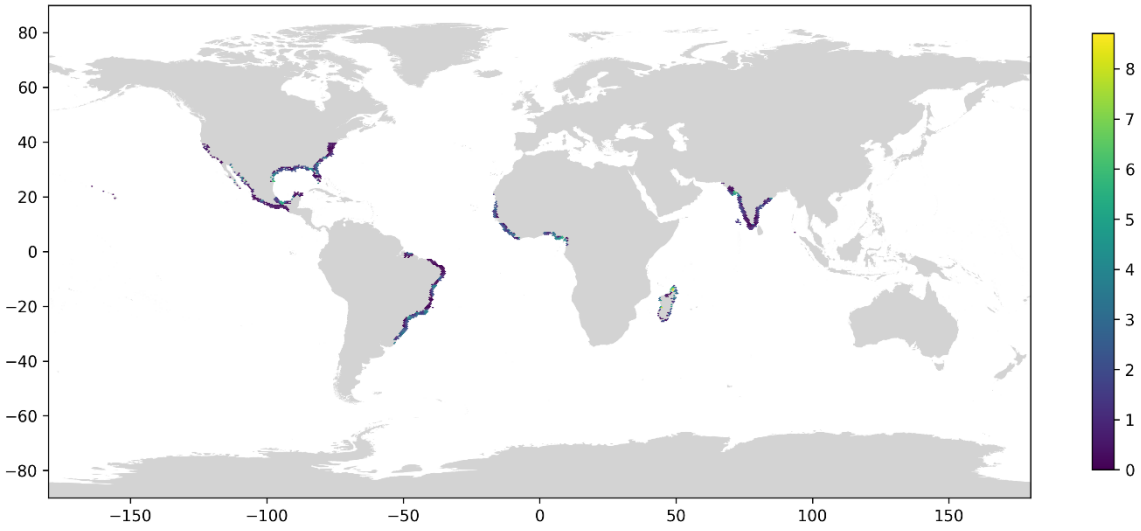
Notes: Panel (a) shows geographic distribution of in-situ microplastic sampling locations and recorded concentrations. Panel (b) reports correlation between the in-situ and remote-sensing measurements at collocated areas. Left panel shows full-sample results. Right panel restricts to post-2013 periods where the in-situ measurements are not used in training the remote-sensing measurement.

Appendix Figure 2. Seafood Access Measurements

(a) Per capita seafood spending, 2017-2018

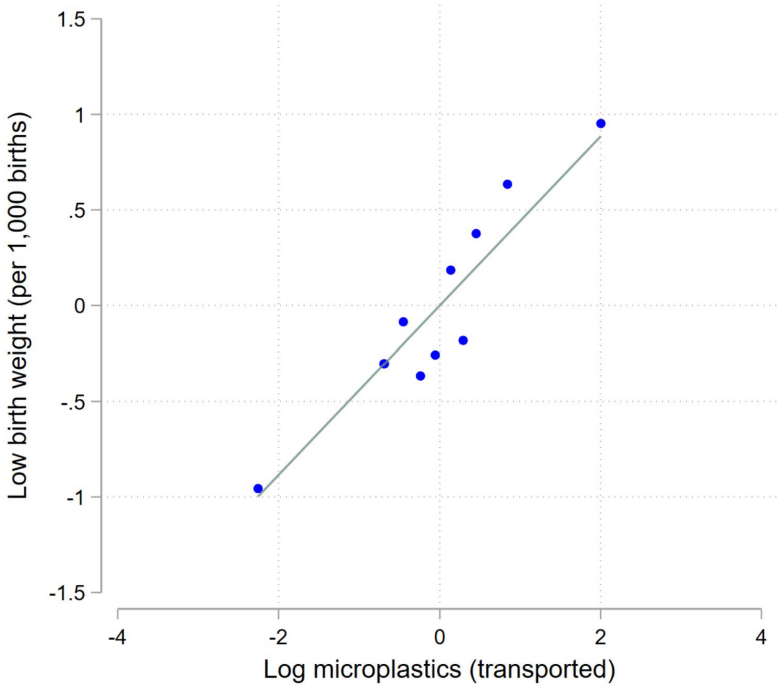


(b) Average fishing hours within 100 km of the coastal location, 2017-2018



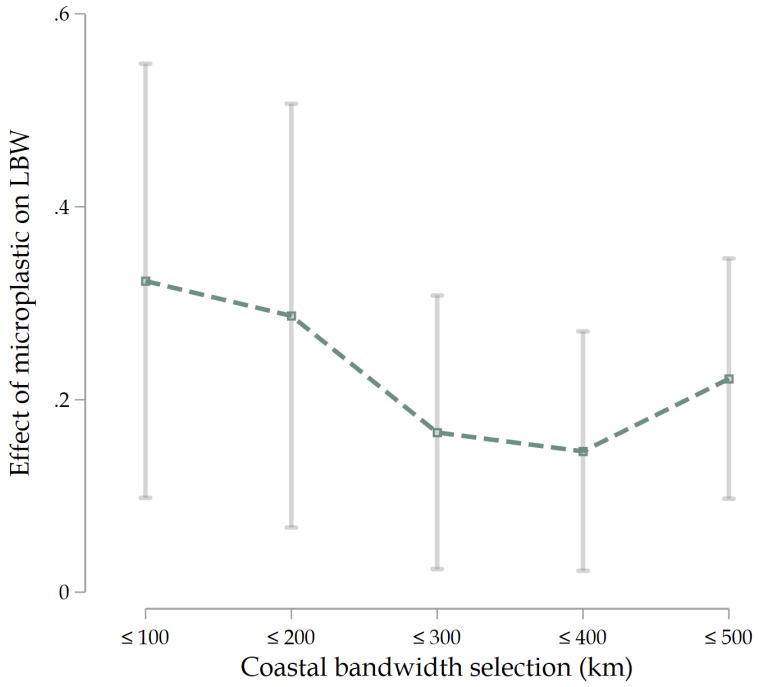
Notes: Panel (a) shows county level 2017-2018 average per capita seafood spending from Nielsen scanner data. Panel (b) shows 2017-2018 average fishing hours from Global Fishing Watch Automatic Identification Systems (AIS) data.

Appendix Figure 3. The Effect of In-Utero Exposure to Microplastics on Low Birth Weight: Dose-Response Function



Notes: This figure shows the dose-response relationship between low birth weight and in-utero exposure to microplastics. We take estimation equation (1), residualize both the LBW variable and the in-utero microplastics variable using the full set of fixed effects, and then plot the residualized variables in a decile bin scatterplot. The slope of the superimposed linear fit corresponds to the point estimates in Table 1, column 1.

Appendix Figure 4. The Effect of In-Utero Exposure to Microplastics on Low Birth Weight: Alternative Coastal Bandwidth Selection



Notes: This figure shows the estimated effect of in-utero exposure to microplastics on the likelihood of low birth weight (LBW), using alternative definitions of the coastal sample based on distance to the shoreline. Each point represents the coefficient from a separate regression restricting the sample to mothers living within the indicated distance (≤100 km, ≤200 km, etc.) from the nearest coastline. The vertical lines indicate 95% confidence intervals.

Appendix Table 1. The Effect of In-Utero Exposure to Microplastics on Low Birth Weight: Robustness Specifications

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Log microplastics (local)	0.287**	0.321***	0.260**	0.295**	0.263	0.246	0.114	0.060
	(0.111)	(0.111)	(0.105)	(0.116)	(0.191)	(0.225)	(0.197)	(0.226)
Log microplastics (transported)	0.442***	0.443***	0.473***	0.483***	0.357***	0.328***	0.400***	0.394***
	(0.078)	(0.072)	(0.084)	(0.078)	(0.083)	(0.080)	(0.112)	(0.118)
Log microplastics (transported from > 200km)	0.381***	0.373***	0.401***	0.395***	0.382***	0.355***	0.415***	0.416***
	(0.077)	(0.076)	(0.081)	(0.078)	(0.084)	(0.086)	(0.104)	(0.120)
Co-pollutants controls	N	Y	N	Y	N	Y	N	Y
FEs: admin 1 region	Y	Y	Y	Y	N	N	N	N
FEs: admin 2 region	N	N	N	N	Y	Y	Y	Y
FEs: month of sample	N	N	Y	Y	N	N	Y	Y
FEs: country × month of sample	Y	Y	N	N	Y	Y	N	N
Dep. var. mean	27.6	28.0	27.6	28.0	27.6	28.0	27.6	28.0
Observations	3,015,121	2,830,110	3,015,121	2,830,110	3,014,832	2,829,876	3,014,832	2,829,876

Notes: Each cell is a separate regression. In each regression, the dependent variable is a dummy for low birth weight multiplied by 1,000. Independent variable is a type of microplastic exposure measurement (local, transported, or transported from grids over 200km away). "Co-pollutants controls" include logged average temperature, precipitation, aerosol pollution, and coastal chlorophyll levels over the course of pregnancy. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Appendix Table 2. Impacts of Local versus Seafood Exporter Countries' Microplastics on Low Birth Weight (USA Sample)

	(1)	(2)	(3)
Log self area microplastics	0.646***	0.407***	0.266***
	(0.058)	(0.086)	(0.072)
Log seafood exporters' microplastics	-0.048	0.063	0.022
	(0.117)	(0.125)	(0.133)
Self-area microplastics source	local	transported, all grids	transported, >200km
Observations	552,706	552,050	552,050

Notes: Each column is a separate regression. In each regression, the dependent variable is a dummy for low birth weight. Independent variable "Log self area microplastics" is a type of microplastic exposure measurement (local, transported, or transported from grids over 200km away). "Log seafood exporters' microplastics" is the weighted average of the coastal microplastics concentrations in the seafood exporting countries that the birth state imports from. The weights are determined by the country-year-month seafood importing value. All regressions control for GADM admin level-1 subcountry region fixed effects and country by month-of-sample fixed effects. Standard errors are clustered at the admin level-1 level. *: $p < 0.10$; **: $p < 0.05$; ***: $p < 0.01$.

Appendix B. Scientific and Clinical Literature on Microplastics

Emission Sources. Since the 1950s, when synthetic organic polymers (plastics) became widely used, global plastic production has surged to 322 million metric tons annually by 2015. About half of this plastic is used for disposables and packaging, with 40 percent of plastic waste not being properly managed through recycling or landfill facilities. Annually, an estimated 4.8 to 12.7 million metric tons of this waste enters the oceans as both plastic litter and microplastic (MP) particles (Worm et al., 2017).

Microplastics are classified into two types: primary, which are intentionally small-sized plastics used in products like cosmetics, cleaning agents, and medical supplies; and secondary, which result from the breakdown of larger plastic items due to sunlight, physical wear, and biological degradation (Cole et al., 2011). The primary sources of microplastic pollution in the oceans stem from both land-based and ocean-based activities. Land-based sources account for approximately 80 percent of the microplastics found in the marine environment (Li et al., 2016). These plastics mainly originate from populated or industrial areas characterized by significant littering, usage of plastic bags, and improper waste disposal. For example, coastal recreational activities frequently lead to both floating and beached plastic debris, especially in regions like the northern South China Sea (Lee et al., 2013). Additionally, manufacturing mishaps can also lead to plastic debris being spilled and carried to beaches (Redford et al., 1997). Moreover, plastics reach the oceans via rivers and wastewater systems that transport them from cities and towns (Browne et al., 2010; Cole et al., 2011). Heavy rainfall and extreme weather events, such as hurricanes, can further increase the movement of these plastics from land to sea (Barnes et al., 2009). Ocean-based activities contribute the remaining 20 percent of marine plastic debris (Li et al., 2016). The most significant contributor in this category is commercial fishing, which loses about 640,000 tons of fishing gear each year. This includes items like nets and lines that continue to trap and harm marine life, a phenomenon known as "ghost fishing" (Good et al., 2010). Figure 1(a) provides a summary of various microplastic sources.

Several studies have aimed to quantify the total number and weight of microplastic particles in marine environments using various methodologies. For example, Jambeck et al. (2015) estimated that populations living within 50 km of coastlines worldwide generate over 8 million tonnes of mismanaged plastic waste annually, much of which enters the oceans. Van Sebille et al. (2015) introduced a statistical framework to standardize a global dataset of plastic marine debris collected via surface-trawling plankton nets. By integrating this data with three different ocean circulation models, they spatially interpolated observations to estimate that between 15 and 51 trillion plastic particles, weighing as much as 236,000 tons, have accumulated in marine systems by 2014. Similarly, Eriksen et al. (2014) used methods including surface net tows and visual surveys in various global marine regions, and reported finding a minimum of 5.25 trillion plastic particles, totaling 268,940 tons, in the world's oceans. These findings emphasize the significant extent of marine microplastics pollution. It is worth

noting that most of these studies that describe global microplastic patterns uses cross-sectional measurements. As we will detail further below, for the purpose of our research, it is crucial to consider not only the spatial variability of microplastic exposure but also the temporal variation – where within the same area, births that occur at different times have different in-utero microplastic exposure. This motivates us to use the new spaceborne global measurements of microplastic distribution developed by Evans and Ruf (2021).

Ambient Presence. Microplastics have become a prevalent environmental concern, with their presence detected in various ambient environments, including water bodies, soil, and the atmosphere. These tiny particles, generally defined as being less than 1 mm in size, enter the marine environment through various pathways, including coastal tourism, fishing activities, marine vessels, industries, and the breakdown of larger plastic debris (Cole et al., 2011). Notably, high concentrations of microplastics have been reported floating in remote ocean areas, particularly in convergence zones of subtropical gyres (Cozar et al., 2014). Furthermore, microplastics have been discovered in deep-sea sediments, marking their presence in even the most remote marine environments (Van Cauwenberghe et al., 2013).

The distribution of microplastics is not confined to marine settings. Research shows that these particles are also pervasive in freshwater systems and soil. For example, Lake Hovsgol in Mongolia, a large mountain lake, has shown contamination (Free et al., 2014). Rodrigues et al. (2018) discovered microplastics distributed in both water and sediments of the freshwater system in Antua River, Portugal. Studies have also reported the presence of plastic in soil (Rillig 2012; Lwanga et al., 2016). Rillig et al. (2012) discovered that microplastic particles can be transported from the soil surface down through the soil profile by earthworms. This movement has implications for the exposure of other soil organisms, the duration of microplastics residence at greater depths, and the potential eventual arrival of microplastics in groundwater.

Microplastics have been found even in atmosphere, indicating that their small size allows them to be airborne and inhaled, potentially causing respiratory issues (Prata 2018; Liu et al., 2019). This airborne nature of microplastics highlights their ability to infiltrate even remote areas, affecting both urban and rural settings. Recent studies have proposed the mechanisms by which microplastics can transfer from aquatic environments to the atmosphere. For example, Lehmann et al. (2021) demonstrates that microplastics can be ejected into the atmosphere by raindrops. This aerosolization process is influenced by the concentration in water and their particle size. Additionally, atmospheric studies have shown that microplastics can be transported over significant distances by air currents, with sea spray and atmospheric movements playing a crucial role in this global distribution (Allen et al., 2020; Caracci et al., 2023). See Figure 1(c) for an illustration.

These findings collectively underscore the pervasive nature of microplastics across various compartments of the ambient environment, emphasizing their potential to be ingested by marine life and subsequently enter human food chains and life.

Environmental Exposure. Microplastics pose a significant environmental threat to organisms in both terrestrial and marine ecosystems. In terrestrial ecosystems, microplastics are found to impact the biophysical properties of soil, suggesting that its widespread contamination may have negative consequences for plant performance and, consequently, for agroecosystems and terrestrial biodiversity (de Souza Machado et al., 2019).

In marine ecosystems, microplastics can accumulate and move around in the bodies of invertebrates such as corals and shellfish and negatively impact the health and feeding behavior of zooplankton, which serve as intermediary species that transfer energy in the ecological food chain (Cole et al., 2011). Microplastics also serve as vectors for chemical pollutants such a pyrene, which get absorbed by mussels and concentrated in their tissues (Avio et al., 2015). The ingestion of plastics has also been observed in marine animals such as seabirds, turtles, crustaceans, and fish, leading to adverse effects such as gastrointestinal blockages and disruption of normal feeding and reproductive behaviors (Ryan et al., 2009; Cole et al., 2011). See Figure 2(b) for an illustration. However, the comprehensive impact of microplastics, including their role in contaminant transfer within the marine food web and potential health implications for human consumption of affected seafood, requires further exploration (Carbery et al., 2018).

Health Effects. Existing evidence showing the direct health consequences of microplastics exposure is very limited (Lim, 2021). To the best of our knowledge, only three papers demonstrate a correlation with health outcomes. All of them use small sample sizes with limited spatial coverage, short study periods, and sampling schemes that may not fully represent the general population.

Specifically, Yan et al. (2022) investigated the correlation between microplastics exposure and inflammatory bowel diseases (IBD). The authors recruited two groups of participants in Nanjing, China: 50 healthy participants and 52 IBD patients, with other non-IBD characteristics similar between groups. Fecal samples revealed microplastics in both groups, with higher concentrations, wider distribution of sizes, and more polyethylene terephthalate microplastics in the IBD group. Microplastics concentrations are positively correlated with HBI score and Mayo score, both capturing IBD activity. The authors also used questionnaires to collect basic information and found that participants who drank bottled water, consumed takeaway food, or had higher dust exposure at work exhibited higher microplastics concentrations in fecal samples, suggesting sources of microplastics exposure through digestion.

The second study by Baeza and Martinez (2022) detected airborne microplastics in human respiratory systems. The authors recruited 44 patients in Spain and found microplastics in the lower airways of 30 patients.

Higher microplastics levels were observed in older age groups, active smokers, and those with high-risk occupations. Combining microplastics samples with X-ray diagnoses, higher microplastics levels are correlated with radiological abnormalities, increased pathological microbial growth, and reduced forced vital capacity, indicating potential respiratory health risks associated with microplastics exposure.

The third paper by Amereh et al. (2022) studied 43 pregnant women in Iran, focusing on how microplastics are associated with birth outcomes. Placenta samples collected within 10 minutes after delivery showed microplastics in all 13 pregnancies with intrauterine growth restriction, compared to only 3 out of 30 normal pregnancies, suggesting a positive correlation between abnormal pregnancies and higher microplastics levels. Additionally, there was a negative correlation between microplastics concentration and birth weight, length, head circumference, and 1-minute APGAR score.

In contrast, a larger number of papers have documented the risks of microplastics exposure by identifying microplastics in food, water, and the environment without directly measuring health outcomes. Microplastics have been found in various marine species and seafood across different regions, including mainland China (Fang et al., 2019; Pan et al., 2022), Hong Kong (Lam et al., 2023), Taiwan (Lu et al., 2021), Thailand (Akkajit et al., 2022), India (Chen et al., 2021; Selvam et al., 2021), Indonesia (Luqman et al., 2021), Iran (Akhbarizadeh et al., 2019), Nigeria (Mahu et al., 2023), Mexico (Martinez-Tavera et al., 2021), Italy (Squillante et al., 2023), Montenegro (Boskovic et al., 2023), Portugal, and Atlantic Ocean (Barboza et al., 2020). Affected species include and are not limited to clams, shellfish, latus, and punctatus, and cover both wild and commercially caught species. These findings suggest potential human health risks due to contaminated seafood consumption. Among them, Luqman et al. (2021) also detected microplastics in coastal residents' stool samples, although they did not analyze other health consequences. Motivated by the seafood contamination, our study tests whether seafood consumption is a potential mechanism through which marine microplastics could affect human health.

Moreover, microplastics have also been detected in water bodies in the US, China, Austria, Saudi Arabia (Liao et al., 2020), and India (Khaleel et al., 2023). They potentially expose humans through direct contact or contaminated drinking water sources. Microplastics also exist in the air as components of particle pollution affecting human health. Studies have assessed airborne microplastics in the UK (Wright et al., 2020) and Iran (Dehghani et al., 2017). Additionally, microplastics have been found in human lung tissue by Amato-Lourenco at al. (2021) and sputum samples by Huang et al. (2022), although direct effects on human health or respiratory malfunctions were not confirmed.

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Appendix C: Ocean Current Modeling Details

To construct a matrix that summarizes monthly long-distance ocean current flow intensities from open-ocean grids to coastal locations, we developed a model that evaluates the intensity of currents between a "sender" grid cell and a "receiver" coastal city for each date. Beginning from a given day and ocean grid, the model traces out a streamline by following the ocean current's direction and speed sequentially on a daily basis. This procedure yields trajectories of current flow that approximate how microplastics could be carried from far-sea locations toward the coast.

To define the set of far-sea sender grids, we first determined a grid spacing (denoted *res*) in polar coordinates such that one grid cell corresponds to approximately 250 km at the equator. We then took all intersection points of this grid (of size *res*) that lie within the latitude range covered by our microplastic concentration data to serve as sender locations. The input current data (from OSCAR) provide daily current vectors (direction and speed) at a 0.25° spatial resolution. These data are used as the basis for the modeled streamlines.

For a given sender grid i and a start date d, we simulate the downstream current trajectory over time. We begin at $t = 0$ with the current position p_0 set to the coordinates of grid i. At each daily time step t, the algorithm first obtains the ocean current vector at the current position p_t on day $d + t$. Because p_t typically does not fall exactly on one of the 0.25° grid nodes, we use linear interpolation of the surrounding OSCAR data to approximate the current at p_t, denoting this interpolated vector by $c_t = (u_t, v_t)$. Next, we identify all coastal receiver cities that lie within a radius rad_t of the position p_t. For each such receiver r, we then compute a raw downstream intensity score:

$$\text{Current}_{i\to r,d,t} = \exp\{-\alpha \cdot \text{rad}_t - \beta \cdot |\theta|_{i\to r,d,t} - \gamma \cdot \text{dist}_{i\to r,d,t}\}$$

where α, β, γ are positive parameters.

The exponential formulation above has three components corresponding to the three terms in the exponent. The first term rad_t is the search radius at step t. This radius represents a general decay in downstream influence as the current travels multiple steps. We increase rad_t by 0.05 each day (approximately 6 km at equatorial latitudes) to capture the growing uncertainty in the exact streamline path and the dispersion of microplastics in the ocean over time. The initial search radius is $rad_0 = 1$, which corresponds to about 111 km at the equator.

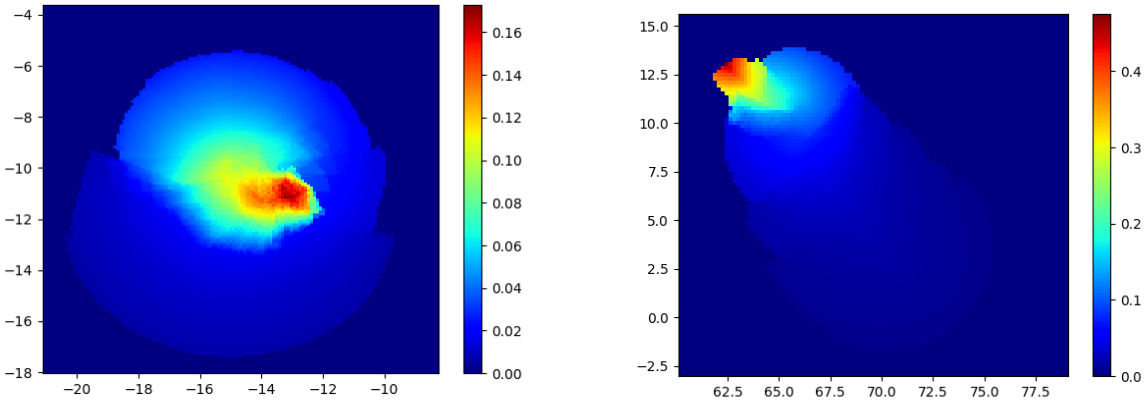
The second term $|\theta|_{i\to r,d,t}$ captures the alignment of the receiver city relative to the current's direction at step t. More precisely, let's note $l_{i\to r}$ the vector between sender grid i and receiver city r. The sense of the vector is not important. Let's note v_t the vector defined as $v_t = (v_t, -u_t)/||c_t||$. Thus, it is a normal vector to the current

vector c_t that has a norm of 1. Then, we define the absolute scalar product $|\theta|_{i\to r,d,t} = v_t \cdot l_{i\to r}$. The higher this term, the closer $l_{i\to r}$ is to be perpendicular to c_t. Since the higher $|\theta|_{i\to r,d,t}$, the lower the score $\text{Current}_{i\to r,d,t}$, and thus the aim of this term is to penalize cities that are less impacted by the current streamlines because they are not in the exactly-downstream direction from the current streamline at that step. It is also important to note that v_t is normalized to avoid seeing lower scores when the speed of the current is higher. However, $l_{i\to r}$ is not normalized to penalize cities that are further from the sender grid.

The third term $\text{dist}_{i\to r,d,t}$ represents the distance (in kilometers) from the original sender grid i to the current position p_t at step t. This distance-based component captures geographic decay: even a perfectly aligned downstream current will have diminishing influence over very long distances. This formulation is inspired by Phillips et al. (2021), which also employs an exponential distance-decay when modeling the dispersion of air pollution. By including a separate distance term $\text{dist}_{i\to r,d,t}$ (in addition to the angular term θ), we allow the decay in intensity with distance to be handled independently of the directional alignment penalty.

In implementing the above formula, we impose two cutoff conditions to ensure that only meaningful downstream influences are counted. We set $\text{Current}_{i\to r,d,t} = 0$ if either (1) $d_{i\to r,d,t} > \text{rad}_t$ (meaning that receiver r lies outside the search radius at step t or (2) $\theta_{i\to r,d,t} > 0.4$ radian radians (meaning that receiver r is more than 0.4 radians out of the downstream direction at that step). We calibrated the parameters to $\{\alpha, \beta, \gamma\} = \{0.8, 0.49, 0.23\}$. These values were chosen empirically to ensure that the accumulated current influence over a 90-day period changes smoothly over time (i.e., the function assigning current scores is approximately continuous rather than erratic). To verify this, we generated heatmap visualizations of $\text{Current}_{i\to r,d,t}$ for various sender grids and days. The final parameter values produce a smoothly varying intensity field; for illustration, the figures below show example current-influence heatmaps for two representative sender locations (one in a circular current pattern, and another in a predominantly unidirectional current).

Examples of current indexes heat maps for two senders with a circular current on the left and a unidirectional current on the right



After computing the contributions for step t, the model advances to the next day (incrementing to $t+1$) and repeats the procedure, up to a maximum of 90 days. As long as $t < 89$, we update the search radius and the current position for the next step. Specifically, we increase the radius by 0.05 degree and move the position $p_t = (x_t, y_t)$ in the direction of the local current vector c_t.

$$x_{t+1} = x_t + 24 * 3600 * u_t / \text{dist}_m((x_t, y_t), (x_t + 1, y_t))$$

$$y_{t+1} = y_t + 24 * 3600 * v_t / \text{dist}_m((x_t, y_t), (x_t + 1, y_t))$$

$$p_{t+1} = (x_{t+1}, y_{t+1})$$

To understand those expressions, we must consider that x and y coordinates are in degrees while the vectors' coordinates u and v are in m/s. The distance (positive or negative) in meters crossed by the current in 24 hours is of $d_{m,x} = 24 * 3600 * u_t$ along the x-axis and $d_{m,y} = 24 * 3600 * v_t$ along the y-axis. To obtain an approximation of the distance d_p crossed in polar coordinates corresponding to a distance d_m in meters, we use a cross product : if a delta of 1 degree in longitude at the latitude y_t represents $\text{dist}_m((x_t, y_t), (x_t + 1, y_t))$ meters, then, an approximation of d_p is $d_p \approx 1 * d_m / \text{dist}_m((x_t, y_t), (x_t + 1, y_t))$. Thus,

$$d_{p,x} \approx 1 * d_{m,x} / \text{dist}_m\big((x_t, y_t), (x_t + 1, y_t)\big)$$

$$d_{p,y} \approx 1 * d_{m,y} / \text{dist}_m((x_t, y_t), (x_t + 1, y_t))$$

and hence the expression of $x_{t+1} = x_t + d_{p,x}$ and $y_{t+1} = y_t + d_{p,y}$.

If point p_{t+1} lies outside of the convex hull of points that represent the position of local current vectors, we stop the algorithm. Indeed, that convex hull represents the ocean area around p_t. Thus, if p_{t+1} lies outside of that polygon, it means that it is inland and the streamline must be stopped at p_t. Otherwise, we can proceed to step $t+1$. Starting from each particular sender grid and day of the period April 2017 to September 2018, we iterate the procedure for 90 steps (i.e., approximately three months) so for $t = 0$ to $t = 89$.

After this computation, we have a set of current scores $\text{Current}_{i \to r,d,t}$ that need to be aggregated at a day level, which means that we want to have a single coefficient for a given tuple (sender grid i, receiver city r, date of arrival d'). For a given index $\text{Current}_{i \to r,d,t}$, the date of arrival d' is the sum of the delay in days from the emission at the sender grid i.e. step t and of the date of emission $d : d' = t + d$. The downstream intensity score aggregated at a day-level for tuple (sender grid i, receiver city r, date of arrival d') is:

$$\text{Current}_{i \to r,d'} = \sum_{d+t=d'} \text{Current}_{i \to r,d,t}$$

Next, we aggregate these daily downstream intensity measures to the month level, which is the time scale used in our econometric analysis. For each sender–receiver pair (i, r) and each month m, we compute the average intensity score in that month:

$$\text{Current}_{i \to r,m} = \text{Average}_{d' \in m}\, \text{Current}_{i \to r,d'}$$

so that $\text{Current}_{i \to r,m}$ represents the mean influence (per day) of currents from grid i on receiver r during month m. This monthly aggregation yields a matrix of intensity scores indexed by sender, receiver, and month of arrival.

Finally, we assemble the full sender–receiver matrix for the study period. We include every pair (i, r) for which at least one nonzero intensity score is recorded over the course of the simulation. For each such pair, if no intensity is recorded in a given month m (within our period of interest, 2017–2021), we assign $\text{Current}_{i \to r,m} = 0$ for that month. The resulting matrix contains up to $n = 15$ rows for each (i, r) pair, where 15 is the number of fully observed months between July 2017 and September 2018. (Because a current emitted in April 2017 can take up to 90 days to reach a coast, only months beginning at least 90 days after the start of the simulation period—i.e. July 2017 onward—are complete. Moreover, if currents originating from a particular grid i never reach receiver r within 90 days for any start date, then that pair (i, r) will have no entries in the final matrix.