Title: **Seasonal Pollen Exposure and Alcohol-Related Harm**

Authors: Joelle Abramowitz, University of Michigan
Olga Aristova, University of Michigan
Shooshan Danagoulian, Wayne State University
Owen Fleming, Wayne State University
Carina Gronlund, University of Michigan
Catherine Maclean, George Mason University
Allison Steiner, University of Michigan

Extended Abstract

Introduction

More than 100,000 Americans die annually from alcohol use disorders (AUDs), and up to 178,000 deaths per year can be attributed to excessive alcohol use more broadly. AUDs include alcohol abuse and dependence, as well as alcohol use resulting in intoxication, withdrawal, and alcohol-related mental disorders. While AUDs are well studied as chronic conditions, little is known about their interaction with other minor health conditions and environmental stressors, including seasonal pollen. This gap is particularly relevant given substantial geographic variation in alcohol- and drug-induced mortality across metropolitan, micropolitan, and rural counties in the United States. Between 2006 and 2020, alcohol- and drug-induced mortality rates were highest in micropolitan counties (14.04 per 100,000), exceeding metropolitan rates (13.48 per 100,000) by roughly four percent, while rural noncore counties had rates of 13.30 per 100,000.

Seasonal allergies, triggered by exposure to pollen released by trees and grasses, result in symptoms such as respiratory congestion, watery and itchy eyes, throat irritation, and cough, with severity ranging from mild to severe. Although these symptoms are commonly treated with widely available over-the-counter medications, both the symptoms and the treatments may interact with alcohol use and substance use disorders, potentially compounding their effects.

We investigate whether seasonal allergies triggered by pollen emissions are associated with alcohol- and substance-related emergency department (ED) visits and mortality. Combining national datasets on pollen emissions, mortality, and medical utilization, we exploit daily deviations in pollen levels from county and seasonal averages to examine whether short-run variation in environmental exposure is reflected in corresponding variation in alcohol- and substance-related outcomes. We place particular emphasis on differences across metropolitan, micropolitan, and rural counties.

Background

Alcohol poisoning occurs when large quantities of alcohol are consumed in a short period of time. Each year approximately 2,200 Americans die from alcohol poisoning (National Center for Drug Abuse Statistics), and there are roughly 8.6 million alcohol-related emergency department visits annually in the United States (SAMHSA 2024). The mean blood alcohol concentration (BAC) among individuals who die from alcohol poisoning is approximately 0.50

(Coughlin and Clary 2019), roughly five times the legal limit for operating a motor vehicle in the United States (0.08). Although tolerance varies across individuals based on biological factors, body composition, and food consumption, the average man (woman) can reach a BAC of 0.08 after consuming four (three) standard drinks within two hours.

Symptoms of alcohol poisoning include confusion, dizziness, slow or irregular breathing, reduced body temperature, dehydration, vomiting, and loss of consciousness. Individuals experiencing alcohol poisoning can often be effectively treated when medical care is obtained promptly. In emergency departments, treatment may include airway management or oxygen therapy, intravenous fluids, monitoring and treatment of seizures, removal of alcohol from the body, and prevention of aspiration associated with vomiting.

Seasonal allergies are also common and affect more than 80 million Americans (Grenier et al. 2011; Jauregui et al. 2009). Pollen allergies occur when the immune system reacts to pollen released by trees, grasses, and weeds during plant reproductive cycles. Seasonal Allergic Rhinitis (SAR), the collection of symptoms associated with pollen exposure, includes respiratory congestion, runny nose, itchy or watery eyes, throat irritation, and cough. While symptoms are mild for many individuals, they can be severe enough to require medical treatment or hospitalization for some.

Beyond these respiratory symptoms, pollen exposure has also been associated with cognitive and behavioral effects. Exposure to pollen has been linked to reduced cognitive performance, mood changes, fatigue, slower response times, and reduced working memory (Marshall et al. 2000; Kremer et al. 2002; Wilken et al. 2002; Marshall and Colon 1993; McAfoose and Baune 2009; Tashiro et al. 2002; Dowlati et al. 2010; Kronfol and Remick 2000). Because allergic inflammation can affect respiration and sleep quality, pollen exposure has also been associated with disrupted sleep and resulting daytime fatigue (Santos et al. 2006; Craig et al. 2004; Tashiro et al. 2002; McAfoose and Baune 2009). A growing literature links these symptoms to behavioral outcomes. Abramowitz et al. (2025) document increases in deaths by suicide as pollen levels rise, while Danagoulian and Deza (2025) show increases in fatal traffic accidents during periods of elevated pollen. Akesaka and Shigeoka (2023) document increases in accidents in Japan during high pollen periods, and Marcotte (2015, 2017) finds that elementary and middle school students perform worse on tests when pollen levels are elevated.

Taken together, this literature suggests that individuals sensitive to pollen may experience both physical and cognitive symptoms during periods of high pollen exposure. These symptoms may influence behaviors, including alcohol or substance use, either as a form of self-treatment or through impaired judgment.

While there is no direct medical evidence documenting a causal relationship between pollen exposure and alcohol poisoning, several biological mechanisms suggest that individuals may be more vulnerable to the intoxicating effects of alcohol on high pollen days. Histamines—the compounds responsible for allergic reactions—can produce symptoms such as dizziness and fatigue that resemble alcohol intoxication, potentially amplifying perceived impairment when alcohol is consumed. Both pollen exposure and alcohol consumption can also contribute to

dehydration through different physiological pathways, and dehydration itself can intensify perceived intoxication.

Medication use may further increase risks. Individuals managing seasonal allergies frequently use medications that interact with alcohol. For example, first-generation antihistamines (e.g., diphenhydramine/Benadryl) combined with alcohol can increase sedation and depress respiration, while decongestants elevate heart rate and blood pressure, increasing the risk of cardiovascular complications when alcohol is consumed concurrently.

Alcohol also interacts directly with the body's histamine system. Alcohol metabolism produces acetaldehyde and slows the breakdown of histamines (Kawano 2004; Shimoda 1996; Koivisto 1999; Zimatkin 1999; Maintz and Novak 2007). When histamine levels are already elevated—such as during allergic reactions—these processes may make the physiological effects of alcohol stronger at a given level of consumption. As a result, individuals may experience symptoms that prompt medical attention even at relatively moderate alcohol intake (Zawacki 2025). This mechanism suggests a shift toward nonfatal, medically attended alcohol-related events rather than an increase in fatal alcohol toxicity.

Alcohol consumption may also worsen allergy symptoms themselves. Many alcoholic beverages, including red wine and beer, contain histamines. Alcohol consumption also reduces the production of diamine oxidase (DAO), an enzyme that helps break down histamines in the body. As a result, alcohol consumption during periods of elevated pollen exposure may exacerbate allergic reactions. In addition, beer and wine often contain sulfites, preservatives that can trigger respiratory symptoms in individuals with allergies or other respiratory conditions. When consumed during periods of heightened allergic irritation, these substances may increase the risk of adverse respiratory outcomes.

Behavioral responses to pollen exposure may further influence alcohol consumption patterns. Individuals may reduce social activities and spend more time indoors on high pollen days, potentially lowering the likelihood of heavy drinking in social settings such as bars or restaurants. Conversely, spending more time at home may increase opportunities for alcohol consumption in private settings. These behavioral responses may also influence whether drinking occurs alone or in the presence of others who could recognize signs of intoxication or poisoning and seek medical assistance.

In addition, individuals with persistent alcohol use may have compromised immune systems or chronic health conditions that increase vulnerability to environmental stressors, including pollen exposure. These health conditions could further increase susceptibility to adverse outcomes during periods of elevated pollen.

In sum, while there is no direct evidence that pollen exposure causes alcohol misuse, several biological and behavioral mechanisms suggest a potential relationship between pollen levels and alcohol-related health outcomes. Our reduced-form empirical approach estimates the net effect of these potentially countervailing channels on alcohol-related outcomes. We then explore potential mechanisms in subsequent analyses.

Data and Methods

Empirical Strategy

Evaluating the relationship between seasonal allergies and AUDs presents two primary challenges. First, the relationship between health shocks and alcohol or substance use may be endogenous: unobserved individual characteristics may influence both allergy susceptibility and substance use behavior, and residential sorting may correlate environmental exposure with underlying risk factors. We address these concerns by exploiting high-frequency within-county variation in pollen exposure and outcomes. By focusing on daily deviations from local seasonal averages, we absorb time-invariant county characteristics and long-run behavioral patterns.

Second, population-level daily variation in health shocks is rarely observed directly, particularly when symptoms arise only after exposure crosses individual-specific thresholds. Allergic responses to pollen are heterogeneous: individuals differ in sensitivity, and symptoms are triggered when pollen concentrations exceed personal tolerance levels. As a result, increases in pollen may shift the share of the population experiencing symptoms without a discrete observable "health shock" in administrative data. We proxy daily allergy exposure using observed and simulated pollen concentrations, which are known triggers of Seasonal Allergic Rhinitis (SAR). While we do not observe individual-level allergy status, increases in pollen concentrations plausibly increase the number of individuals experiencing symptoms at the population level.

Our identifying variation comes from short-run deviations in pollen within county-season cells. Conditional on detailed weather controls and fixed effects, we treat these deviations as plausibly exogenous to contemporaneous human behavior. This design allows us to estimate associations without relying on cross-county comparisons or difference-in-differences assumptions such as parallel trends.

Given that outcomes are non-negative counts at the county-day level and frequently include zeros, we estimate Poisson models of the form:

$$\log\left(E\left(Y_{j,dmy}\middle|X_{j,dmy}\right)\right) = \alpha + \beta \ln\left(pollen_{j,dmy}\right) + W_{j,dmy}\Gamma + Wind_{j,dmy}\Xi + \nu_j + \kappa_{my} + \psi_{dm} + \varepsilon_{j,dmy}$$

where $Y_{j,dmy}$ are the outcomes of interest such as number of ED visits or number of deaths by type in county j, on day d, month m, and year y. Weather controls $W_{j,dmy}$ includ temperature (minimum, maximum, and mean), precipitation, and humidity; wind control, $Wind_{j,dmy}$, including speed and direction categorized into four 90-degree bins). County fixed effects absorb time-invariant local characteristics such as vegetation mix and regulatory environment. Granular time fixed effects—including month-by-day, month-by-day-of-week, and month-by-year—remove seasonal consumption patterns, holiday effects, and secular trends. Standard errors are clustered at the county level.

To investigate geographic heterogeneity, we estimate separate models by metropolitan, micropolitan, and rural county designation.

Data

Pollen Exposure

We use two sources of pollen data. First, we use daily pollen measurements from the National Allergy Bureau (NAB), collected by members of the American Academy of Allergy, Asthma, and Immunology between 2008 and 2020. Approximately 131 stations report near-daily pollen concentrations using standardized volumetric spore traps, expressed as particles per cubic meter (pcm). We aggregate total pollen across tree, grass, and weed species in the primary analysis and examine heterogeneity by pollen type and season in robustness checks.

NAB stations are primarily located in metropolitan counties and data collection is uneven across time and space. To extend coverage, particularly to micropolitan and rural counties, we use simulated pollen emissions from the Pollen Emissions for Climate Models (PECM) system (Wozniak and Steiner 2017). PECM combines vegetation type, land cover, phenology, and weather inputs to generate daily pollen emissions at 25-km resolution across the contiguous United States. These estimates are aggregated to counties and validated against observed NAB measurements in metropolitan counties before being used in national analyses.

Weather, Wind, and Pollution

Because pollen emissions and dispersion are shaped by meteorological conditions, and because pollen may interact with particulate matter, we control for weather and pollution variables.

Daily weather data are drawn from PRISM, including temperature, humidity, and precipitation. Wind speed and direction are constructed from North American Regional Reanalysis (NARR) u- and v-components. Pollution data, including Air Quality Index (AQI) and PM2.5, are obtained from the Environmental Protection Agency. These controls reduce potential confounding between pollen, weather conditions, and respiratory or behavioral outcomes.

Mortality Data

Mortality outcomes are constructed from National Vital Statistics System Detailed Multiple Cause of Death (NVSS-MCOD) files, 2007–2017. These administrative records include the universe of U.S. deaths, with county and date of death and ICD-coded causes. We restrict analysis to deaths attributed to alcohol or substance use disorders using underlying cause-of-death codes. Deaths are aggregated to the county-day level and further stratified by demographic characteristics.

Medical Utilization Data

We analyze emergency department utilization using Centers for Medicare and Medicaid Services claims files: 20% Medicare claims (2008–2020) and 100% Medicaid claims (2008–2019). Analyses are conducted separately by program.

ED visits are identified by place of service and primary diagnostic codes corresponding to alcohol use disorder and substance use disorder. For robustness, we examine secondary diagnostic codes. Visits are aggregated to the county-day level and stratified by demographic characteristics.

Population denominators are obtained from the American Community Survey 5-year files to construct rates. Counties are categorized as metropolitan, micropolitan, or rural using Census designations. Metropolitan counties include large central, large fringe, medium, and small metropolitan areas (1,186 counties, 86.2% of the population). Micropolitan counties (658 counties, 8.4% of the population) contain urban cores of 10,000–49,999 residents. Non-core counties (1,300 counties, 5.4% of the population) are categorized as rural.
Observed and simulated pollen measures are merged with mortality and claims data at the county-day level. We include zero-count observations and exclude days lacking pollen measurements to avoid imputing zero exposure.

Preliminary Results

Alcohol-Related Emergency Department Visits (AUD)

In our preferred specification, including weather controls and granular county and time fixed effects, a 100 percent increase in pollen is associated with 0.01 additional alcohol-related emergency department visits per county-day among Medicaid beneficiaries, corresponding to a 0.6 percent increase relative to baseline. Though inclusion of space and time fixed effects makes a material difference to the magnitude of the estimated effect, the inclusion of weather controls does not affect the estimation significantly.

Estimates for Medicare beneficiaries are negative but imprecisely estimated and not statistically distinguishable from zero once weather and fixed effects are included.

The estimates are robust to changes in specification, choice of fixed effects, and definition of the treatment variable. The estimates of the alcohol effect in Medicaid claims remains stable across alternative fixed-effect structures, including saturated specifications with county-by-time interactions. We estimate the effects using ordinary least squares specifications which produce slightly larger and more precise estimates but does not alter qualitative conclusions. Normalizing visits by county population similarly yields comparable results. Including a 7-day moving average of pollen does not materially change the magnitude of the contemporaneous effect.

Nonlinear specifications suggest stronger responses at higher exposure levels. Though the logarithmic transformation of pollen is in part motivated by approximate nonlinearity at high levels of pollen, we also use quartile bins to estimate Poisson and OLS specifications. These confirm that while Medicaid visits increase with each quartile, when pollen is in the highest quartile visits increase by approximately 0.5 visits in Poisson models and 0.10 visits in OLS specifications.

Dynamic specifications indicate that alcohol-related responses are largely contemporaneous. Including one- and two-day lags of pollen does not materially alter the contemporaneous coefficient, and lagged effects are small and statistically insignificant.

As alcohol consumption is more frequent on the weekends, we estimate the preferred specification by weekday and weekend. Among Medicaid beneficiaries, alcohol-related visits increase by 0.02 visits (0.7 percent) on weekends, compared to 0.003 on weekdays.

Geographic heterogeneity is pronounced. Using simulated pollen to obtain full national coverage, we find that the Medicaid alcohol effect is concentrated in micropolitan counties. In these counties, a 100 percent increase in pollen is associated with 0.009 additional visits (10.3 percent relative to baseline), while metropolitan and rural counties show smaller and statistically insignificant effects. Among Medicare beneficiaries, alcohol-related visits increase in metropolitan counties (0.01 visits, 25.6 percent), with no significant effects in micropolitan or rural counties.

Substance Use Disorder (SUD)–Related Emergency Department Visits

In contrast to alcohol-related visits, estimates for SUD-related visits are smaller and less consistent. Among Medicaid beneficiaries, we detect no statistically significant change in SUD-related visits in response to pollen increases in preferred specifications. Among Medicare beneficiaries, in the preferred specification, the estimated change is −0.006 visits (2.7 percent), marginally significant.

Dynamic models suggest short-run temporal adjustment for SUD-related visits. With one lag included, contemporaneous pollen is associated with 0.004 additional Medicaid visits (0.3 percent) and 0.01 fewer Medicare visits (27 percent). With two lags, the contemporaneous effect dissipates, and lagged coefficients alternate in sign. These patterns suggest delayed adjustment rather than immediate response.

Mortality

Main Results

[Results embargoed until release by NCHS]

Robustness of Estimates

[Results embargoed until release by NCHS]

Heterogeneity Over Time and Space

[Results embargoed until release by NCHS]

Discussion and Conclusion

Though pollen is a naturally occurring plant emission, growing evidence documents its adverse effects on human health and behavior. Seasonal allergies triggered by pollen have been linked to reduced cognitive functioning (Marshall et al. 2000; Kremer et al. 2002; Wilken et al. 2002), worse mental health (Abramowitz et al. 2025), and reduced alertness (Danagoulian and Deza 2024; Craig et al. 2004; Santos et al. 2006). Our results contribute to this literature in several ways.

First, we show that rising pollen levels are associated with more alcohol-related emergency department (ED) visits. We estimate 0.011 additional visits per county per day, which corresponds to a 0.6% increase relative to the mean. Back-of-the-envelope calculations suggest this translates to roughly 1.2 additional alcohol-related ED visits per county per year. Although

modest in magnitude, these visits represent episodes of alcohol consumption severe enough to require medical intervention. The estimates therefore suggest that instances of alcohol incapacitation in the community rise as pollen levels increase.

Second, we examine alcohol-related mortality. While the point estimates indicate a change in alcohol-related deaths as pollen levels rise, the magnitude of the effect corresponds to approximately X.XX deaths per county per day, or about X.X% of the mean. Back-of-the-envelope calculations imply a change of approximately X.X deaths per county per year associated with the interaction of seasonal allergies and alcohol consumption.

Third, the mortality results suggest that seasonal allergies may alter the physiological risks associated with alcohol consumption. Although pollen exposure increases alcohol-related ED visits, we observe a different pattern for alcohol-related mortality. This divergence implies that pollen exposure increases alcohol-related incidents while altering the likelihood that those incidents become fatal. Prior work has largely emphasized behavioral pathways—such as fatigue, sleep disruption, and reduced cognitive functioning—through which allergies impair judgment. The pattern we observe is difficult to reconcile with purely behavioral mechanisms and instead suggests that allergies may also influence how alcohol is metabolized or tolerated, modifying the risk of severe outcomes. Clinical studies indicate that allergic inflammation and antihistamine treatment may interact with alcohol metabolism and physiological responses, potentially altering the severity of alcohol-related outcomes.

Fourth, we find no statistically significant changes in ED visits or mortality related to other substance use disorders (SUDs) as pollen levels increase. If alcohol or drug use were primarily serving as a coping mechanism for allergy symptoms, we might expect increases across substances. The absence of such effects suggests that the relationship between pollen exposure and alcohol outcomes is unlikely to be driven solely by substitution toward substance use as a behavioral response to allergy symptoms.

These findings have implications for how alcohol impairment is understood in policy contexts. Policies such as drunk-driving laws rely on blood alcohol concentration thresholds as proxies for impairment. However, prior research suggests that interactions between histamines and alcohol may increase impairment at lower levels of blood alcohol concentration. If allergic responses alter the physiological effects of alcohol, then environmental factors such as pollen exposure may influence the relationship between alcohol consumption, impairment, and health outcomes.