Narrative Review Article

# Environmental Noise Exposure and Gastrointestinal Outcomes: Differential Impacts on M arginalized Communities

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## ABSTRACT

Environmental noise exposure is an increasingly recognized public health concern, linked to a variety of adverse outcomes, including gastrointestinal disorders. Chronic inflammation, in particular, has been associated with long-term exposure to elevated noise levels, yet the underlying mechanisms and contributing social factors remain poorly understood. This narrative review aims to investigate how noise exposure has been associated with chronic gastrointestinal inflammation and to examine the relationship between socioeconomic status and susceptibility to inflammatory conditions through a comprehensive literature review. Peer-reviewed articles, public health reports, and epidemiological studies were identified through targeted literature review and narratively synthesized to identify recurring patterns in noise exposure, gastrointestinal health outcomes, and socioeconomic disparities. The review revealed consistent evidence that higher noise exposure is associated with increased risk of gastrointestinal inflammation; additionally, socioeconomic factors such as income level and neighborhood composition may modulate vulnerability, with disadvantaged populations experiencing disproportionate impacts. In synthesizing findings from multiple studies, this review highlights the interplay between environmental and social determinants of gastrointestinal health and underscores the importance of considering both when developing public health interventions. These insights contribute to a deeper understanding of how environmental noise affects human health and provide evidence to inform policy and community-based strategies including urban planning, transportation noise regulation, and occupational exposure standards aimed at reducing health inequities.

**Keywords:** Noise pollution; stress hormones; gastrointestinal tract; inflammation; cortisol; socioeconomic disparities; chronic stress

## INTRODUCTION

Rapid urbanization and the resulting concentration of human populations in recent years has generated a significant number of environmental stressors that have considerable impacts on human health. Noise — or an unwanted sound — is one of the most commonly experienced stressors, with, in 2013, an estimated 104 million Americans having annual average noise levels above 70 decibels (dBA) and being at risk of noise-induced hearing loss (1). The numbers and risks have only increased since then, yet noise pollution continues to be overlooked as a significant health hazard and is rather framed as an annoyance or inconvenience.

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Similar to a number of other environmental pollutants, numerous studies found that noise pollution has disproportionate impacts on underprivileged communities. On average, research found that using geospatial lower-income neighborhoods were nearly 2 dBAs louder than affluent neighborhoods, and that communities with ~75% black residents had median nighttime noise levels of 46.3 dBAs — 4 dBAs louder than the 42.3 dBAs experienced by communities with no black residents (2). Considering that a 10 dBAs increase indicates that the volume of noise has doubled, 4 dBAs is a significant difference. Such disparities persist due to redlining — the discriminatory practice of denying or limiting financial services based on their race or ethnicity. This perpetuation of racial segregation concentrates minority homeowners within lower-income neighborhoods that are valued at lower price points due to their proximity to sources of noise such as major roads, industrial areas, and airports.

The intrinsic mechanism of noise-induced stress is strongly linked to the brain's amygdala, the brain structure responsible for identifying potential threats, retaining and managing memory, and, most importantly, regulating emotional responses — including stress. Once the lateral amygdala (LA) receives information from the auditory thalamus and auditory cortex, these signals sent to the central amygdala (CeA) activate the two core stress systems — the sympathetic-adrenal-medullary (SAM) axis and hypothalamic-pituitary-adrenal (HPA) axis — through the hypothalamus, thereby releasing stress hormones such as cortisol, adrenaline, and norepinephrine (3).

The volume of industrial/urban noise largely influences the intensity of the stress response along with the amount of stress hormones, as seen by a 2012 study observing the relationship between industrial noise with levels >80dBA and cortisol levels (nmol/L): participants who took a leisure day away from industrial noise (<80dBA) experienced typical evening cortisol levels of 4.2-_6.3 nmol/L, whereas participants on site at industrial workplaces experienced significantly higher levels of evening cortisol at 6.5-10.0 nmol/L (4). Such high levels of cortisol for those exposed to industrial noise are indicators of chronic stress, poor sleep quality, and various other health concerns.

Such elevated levels of stress hormones cause harm to many bodily systems, particularly the GI tract. The gut-brain axis, a bidirectional communication network linking the enteric and central nervous systems, plays a critical role in determining the impact and role of environmental stressors like noise pollution, as they extend to include multiple other routes of communication other than the anatomical, including the endocrine route. Chronic psychosocial stress disrupts the nervous system and triggers cascading effects. To begin, the activation of the sympathomedullary pathway triggers nerve activity in locus coeruleus (LC), subsequently stimulating the sympathetic ganglia to transmit excitatory signals to sympathetic nerves (5). This cascade not only causes the adrenal medulla to secrete adrenaline, but also stimulates the eosinophils to degranulate and release corticotrophin-releasing hormones (CRH). CRH then stimulates mast cells, which function as immune cells of myeloid lineage. The degranulation of mast cells releases inflammatory cytokines, disrupting the gut barrier and increasing intestinal permeability. Along the HPA pathway, central secretion of CRH from the paraventricular nucleus prompts the release of adrenocorticotropic hormone (ACTH) from the anterior pituitary gland. In turn, the adrenal cortex releases glucocorticoids (cortisol) which stimulates inflammation and compounds the negative effects and weakening of the intestinal barrier. This study aims to further investigate the ways in which noise exposure leads to such chronic gastrointestinal inflammation, and establish the relationship between socioeconomic status and inflammatory conditions.

## NOISE POLLUTION AND THE SOCIOECONOMIC GRADIENT

The following studies analyze noise pollution patterns across socioeconomic gradients measured by census-tract and -block level groups. Existing databases and computational models were used to conduct these cross-sectional investigations, which attempted to identify overlapping patterns between noise distribution across neighborhoods and racial/socioeconomic concentrations of specific demographics. All studies concluded that the data was indicative of a strong relationship between demographics and exposure to noise pollution, all coming to the conclusion that historically marginalized groups, such as Hispanic and Black communities, were exposed to net-greater levels of noise pollution. The ability of these studies to make such nationwide statistical analysis while identifying and emphasizing differences at the community level serves to contextualize studies on the health impacts of noise pollution in relevance to the entire nation.

### Scientific Measurement of Noise Complaint

One study by Casey *et al*. has two primary objectives:

to a) assess racial and socioeconomic inequalities in noise pollution in the contiguous United States; and b) consider the role of metropolitan-level racial residential segregation in the relationship between external variables and their corresponding outcome (2). This cross-sectional investigation utilized geospatial sound models to estimate median daytime and nighttime noise, as well as 90th percentile daytime noise during the summer, with neighborhood-level demographic and socioeconomic data (independent variables) drawn from the 2006–2010 American Community Survey (ACS) consisting of race/ethnicity, education, income, poverty, unemployment, homeownership, and linguistic isolation. Expected noise pollution was modeled using empirical acoustic data and geospatial features, which integrated over 1.5 million hours of long-term measurements from 492 urban and rural sites across the United States during 2000-2014 and underwent cross-validation processes using random forest, a tree-based machine-learning algorithm, before producing ambient sound maps at 270-m resolution that were averaged within blocks. Associations between demographic characteristics and noise levels per block group were analyzed in consideration of the present level of metropolitan area racial segregation (with higher values on the multigroup dissimilarity index indicating greater segregation). Statistical models which accounted for neighborhood clustering and differences in population size and density were used to observe how each socioeconomic factor related to noise. Results were organized into scatter plots and summarized using medians and interquartile ranges in order to simplify patterns. Within 214,105 block groups across 933 metropolitan areas (175,373 urban and 38,732 suburban/rural), data found that poverty, low education, and higher proportions of nonwhite residents were concentrated in the South and Southwest (2). Urban block groups had more racial/ethnic minorities (38% vs. 19.3%), more renters (34.5% vs. 26.9%), and slightly lower poverty (13.3% vs. 16.8%). In relation to noise, individuals, households, and families in urban areas with lower socioeconomic status (SES) and higher diversity had, on average, higher nighttime and daytime noise. These patterns stayed generally consistent across segregation levels.

This study identifies populations at highest risk for chronic noise exposure, and therefore risk for gastrointestinal health. It empirically shows that communities with lower socioeconomic status consistently face higher levels of noise pollution, establishing a solid ground for this study to build off of. Furthermore, showing that disparities persist across different segregation contexts highlights that noise inequity is a structural, nationwide problem, not just a feature of highly segregated urban neighborhoods. In demonstrating disproportionate exposure to and impacts from noise pollution for marginalized communities, this study provides critical social and environmental context.

Although geospatial modeling represents a major methodological advancement over earlier studies that relied primarily on population density as a proxy for noise exposure, this approach still carries important limitations. Because estimates are generated at the block-group level, the models capture patterns of noise distribution but cannot reveal the specific structural mechanisms—such as zoning decisions, transportation routing, industrial siting, or historical housing policies—that produce these disparities. As a result, the analyses illuminate where noise inequities occur but not why certain communities bear disproportionate burdens. Moreover, individual-level exposures, daily movement patterns, and co-exposures to other environmental stressors remain unmeasured, meaning the observed associations likely underestimate the complexity of lived noise experiences. Despite these constraints, the findings nonetheless provide a crucial foundation for understanding the socioeconomic and racial stratification of environmental noise across the United States.

Another study by Shekembi *et al*. has the primary objective of investigating whether racial and ethnic minority communities are disproportionately burdened by transportation- and workplace-related noise pollution, while assessing the impact of structural racism through historical redlining contributed to the hypothesized inequity in exposure (6). Estimates of census-tract level transportation noise pollutants were made using population-weighted estimates of the percentage of individuals exposed to 24-hour noise >55dBA (a level considered hazardous to human health regardless of the source. These estimates were based on transportation noise estimates from the 2018 Department of Transportation (DOT) National Transportation Noise Map (NTNM) using a Monte Carlo simulation approach (which uses repeated random sampling for estimates). Because the data estimates at the census block level, this study aggregated data up to the census tract level (n ~ 8,000,000 tracts) — excluding estimates with ≤20 residents — as the majority of demographic data is available only at the census tract level (6). Estimates of census-travel level workplace noise pollution were developed based on the percentage of workers exposed to

>85dBA (considered hazardous) for each census tract in the United States (US).

Census tract-level racial and ethnic composition variables were derived from the 2015-2019 American Community Survey as the proportion of non-Hispanic residents who are White, Black, Asian, American Indian or Alaskan, or Native Hawaiian or Pacific Islander, as well as the proportion of Hispanic Americans — racial and ethnic minority individuals were considered those who were not non-Hispanic White. The three indicators of socioeconomic status were from the same 2015-2019 American Community Survey: the proportion of residents who were low income; those without a high school diploma; and the proportion of unemployed individuals. Census tracts were also categorized either urban or non-urban using the US Department of Agriculture Rural-Urban Commuting Area Codes (levels 1-3 "urban"; levels 4-10 "non-urban").

The relationship between historical redlining and mortgage discrimination and present-day exposure to workplace and transportation noise pollution across US census tracts was assessed using area-weighted redlining scores for 2010 census tract boundaries, with 1 (best grade) reflecting the lowest proportion of historically redlined areas within its boundaries, and 4 (worst grade) reflecting the highest (6). Mortgage discrimination in the 1990s was assessed based on the Home Mortgage Disclosure Act Longitudinal Dataset (HLD) and the 2000 Census, calculating a discrimination index as the ratio of the share of mortgage loans to racial and ethnic minority borrowers to their share of households within each tract (6). All historical data was harmonized to the 2010 census tract boundaries via the OLongitudinal Tract Database (6).

Analysis of the data showed that neighborhoods with a higher proportion of racial and ethnic minority individuals faced disproportionately high cumulative exposure compared to nationwide levels. More specifically, tracts with the highest proportion of minority residents had an 8.59 times higher odds of experiencing high transportation and workplace noise compared to those with the lowest minority proportions (6). The greatest disparities were observed in tracts with high concentrations of Hispanic and non-Hispanic Black residents. Data found that structural racism contributed to higher exposure to noise pollution, with tracts with sustained mortgage discrimination showing a 1.83% higher average prevalence of noise (6). In studying workplace sources of noise, this study specifies how socioeconomic status mediates exposure to environmental noise. Research proves that in addition to living conditions and community environments, lower-income and marginalized workers are disproportionately represented in industries with less regulatory enforcement and high noise levels — such as manufacturing, construction, and transportation. This provides a structural explanation for the cumulative and cyclical stress burdens experienced by marginalized groups that heighten vulnerability to noise-related gut health issues.

One thing to note here is that most of the analysis was conducted at the ecological level, which introduces the risk of ecological fallacy. Furthermore, the focus on two distinct sources of noise pollution didn't allow the study to example the cumulative workplace and environmental exposures at the individual level, meaning that this approach cannot determine the degree of co-exposure to environmental and occupational noise (although some overlap is assumed). For transportation, the grouping of the three main sources of noise into a single measure of transportation-related noise pollution prevalence means the study does not provide information about geographical variation to different types of transportation noise. For workplace noise, the study cannot distinguish between ambient noise exposure and industrially-produced sources of noise.

To further support, Ramphal *et al*. used over 4 million noise complaints from the New York City (NYC) 311 database, this study quantifies socioeconomic disparities in noise complaints since 2010 (7). The NYC Noise Code sets limitations on noise: it circumscribes construction timing for 7AM to 6PM, sets dBA limits for nightlife, dissuades loud residential activities, and establishes fines for violations. Despite these buffers of noise pollution, over 4.3 million noise complaints were downloaded (98.7% of which included latitude and longitude) using the tigris package in R (7). Statistical analysis of this data primarily aims to examine socioeconomic disparities in census tract-level noise complaints prior to COVID-19 (2010-2020). Using a linear mixed-effects model, the study modeled the monthly number of complaints as the dependent variable, with time, month, and the proportion of low-income residents as primary predictors (7). Covariates included tract-level population and population density. The data was best represented through a linear approach. A post-hoc seasonal analysis — using January as the reference month — examined seasonal differences in noise complaint disparity growth. Results found primarily that tract-level population density was associated with noise complaints, with overall complaints since 2010 having increased in a socioeconomically disparate manner (7). Warmer months displayed greatest socioeconomic disparities in complaints, with the overall number of complaints across the socioeconomic gradient increasing exponentially between 2010 and 2020. In quantifying and observing the number of noise complaints in a given neighborhood, this study describes the environments in which lower-class, marginalized people live, contextualizing the disproportionate exposure to noise seen in numerous noise pollution studies (7). A product of redlining and discriminatory housing policies, racial minorities are frequently situated near high-noise environments like those observed in the study.

Nonetheless, acoustic street recordings in specific locations have potentials for errors as it may not capture the full spectrum of noise sources and resultant annoyance and associated disparities possibly obfuscating potential public health ramifications. Disparities may also be underestimated in this study given that socioeconomically disadvantaged communities are less likely to make complaints to their local municipalities about quality-of-life issues due to the economically and racially discriminatory nature of policing and the attenuating effects of invasive policing on noise complaints.

This diagram summarizes the biological pathways through which environmental noise from transportation, residential, and industrial sources activates the sympathetic–adrenal–medullary (SAM) system and the hypothalamic–pituitary–adrenal (HPA) axis. Activation of the adrenal glands increases secretion of cortisol and adrenaline, which subsequently reduce tight junction protein expression, alter cytokine production, and decrease microbial diversity in the gut. These downstream effects collectively increase gastrointestinal permeability, promote chronic inflammation, and contribute to clinical outcomes such as functional dyspepsia, inflammatory bowel disease, intestinal perforation, and potentially elevated cancer risk. As illustrated in Figure 1, environmental noise activates neuroendocrine stress pathways that compromise gut barrier integrity and promote inflammatory responses.

## ENDOCRINE STRESS RESPONSES TO INDUSTRIAL NOISE

In hopes of better understanding this abstract relationship, these studies observe the correlation between noise pollution and stress hormone levels. Given that saliva is a widely available source of measurable cortisol, both studies on cortisol used the methodology of swabbing the mouth for samples at designated times of day before being frozen until ready for analysis. Stress is also largely psychological, however, so all studies on hormones, including the studies on cortisol and the study on adrenaline, required the study's human participants to fill out reflective surveys as methods of subjective data collection. Recognizing that stress can manifest in multiple forms, these studies play critical roles in identifying pathways to focus on in this comprehensive analysis; namely, cortisol and adrenaline.

### Cortisol Response to Occupational Noise Exposure

Fazli *et al.*'s quasi-experimental study observes the impact of occupational noise exposure on stress levels, and measured by salivary cortisol, in workers in the textile industry (8). The studied population, selected

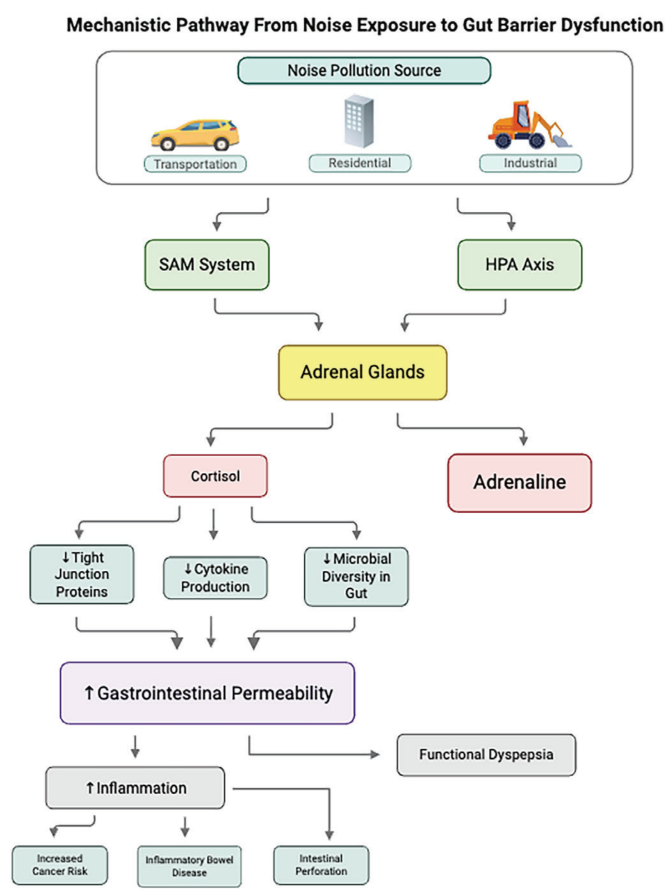


*Figure 1. Mechanistic pathway linking noise exposure to gut barrier dysfunction and gastrointestinal inflammation. Created in Biorender.*

from similar exposure groups in one textile hall from a weaving factory in southeast Iran, consisted of 72 workers, 53 of which were excluded from the actual study due to falling in the exclusion criteria (which included hearing impairment, headaches, cardiovascular disease, work experience of <1 year, etc.) (8). All workers gave written informed consent before completing a structured checklist containing individual demographic information, such as gender, age, weight, height, and work experience. Stress levels throughout the study were observed both subjectively and objectively. The Osipow occupational stress questionnaire (OJSQ), a widely used tool to assess occupational stress and its impact on individuals, was used to measure stress subjectively. 60 questions relating to job stress are divided into six subgroups (workload, incompetence, duality, role range, role responsibility, and physical environment), each containing 10 questions. The 60 questions generated scores divided into five categories: stress below level (60 to 109), normal level (110 to 159), mild level (160 to 209), moderate level (210 to 259), and severe level (260 to 300) (8). Objective stress levels were observed through salivary cortisol samples, which were collected on the morning and afternoon of two consecutive days: a working day exposed to noise in the textile hall, and a rest day exposed to normal noise in the boarding house. Saliva samples of 2mL were collected in a sterile vial immediately after waking up (between 6:00AM and 7:00AM, at the start of their shift), and at 2:00PM (end of working time). These samples were stored in a freezer at -18°C until analyzed for salivary cortisol using the electrochemiluminescence immunoassay, which uses an Elecsys ™ immunoassay analyzer and the cobas ™ salivary kit (8).

All statistical analysis was performed using SPSS software, version 16. Data analysis confirmed that salivary cortisol levels were higher on working days than during rest for both men and women — 5.596±1.74 compared to 3.99±1.41 for men in the morning, and 6.552±1.85 compared to 3.68±1.91 for women in the morning (8). Results also showed that all participants were more exposed to noise on working days than the permissible occupational limits. These findings underscore the role of noise as a nonspecific stressor that triggers the autonomic nervous system and the endocrine pathway. This quasi-experiment provides both subjective and objective evidence linking occupational noise exposure to increased stressed levels in industrial workers. This study is especially critical in understanding the relationship between cortisol levels and occupational stress, as it captures multifaceted impacts of noise exposure as a stressor. As a result, it highlights the importance of noise regulation and the implementation of workplace interventions to ensure the physical and psychological wellbeing of workers. The only limitation of this study was transporting saliva samples to the laboratory promptly after collection — hence why samples were frozen until analysis.

Another study by Fouladi *et al*. adds further insight connecting noise pollution's impact on stress hormones (9). Fouladi's study has three primary objectives: 1) assess and compare saliva cortisol concentrations in the morning and evening in normal workday and leisure day in industrial workers, 2) assess the relationship between industrial noise exposure and salivary cortisol concentrations, and 3) assess the possibility of using salivary cortisol as a possible marker of noise-induced stress. The 80 male participants working in 4 different parts (painting, assembling lines, casting, and packaging) of a household manufacturing company (with 170 male workers total) received the information regarding the aim and scope of the study and enrolled as volunteers, meaning they were free to leave the experiment whenever demanded. The study group had a mean age of 37, mean height of 173.9 centimeters, mean weight of 74.5 kilograms, and mean work experience of 17.1 years (9). General health status was self-reported through a questionnaire (9). Morning and evening saliva samples were collected at 7.00 am and 4.00 pm, respectively, and were refrigerated soon afterwards to be carried to the laboratory for further analysis (9).

Depending heavily on the participants' contributions, the study's first round of noise measurement (during a leisure day) instructed workers to collect their own saliva at the given times and turn the noise dosimeter on shortly after awakening and to turn it off at 4:00PM after collecting the second sample. The second round of noise measurement followed the same procedure, this time with the additional request that participants do not use ear protection devices. In addition to personal noise dosimeters, a Brüel and Kjær type 2236 sound level meter was used to determine ambient noise levels in the workplace at the 80 workstations where the participants worked, excluding work stations with peak sounds. Noise levels ranged from 80 to 88dBA (9).

Results showed that all 80 participants on the leisure day had morning salivary cortisol levels that were significantly higher and more concentrated than evening levels. There was no significant difference between morning levels between leisure day and working day samples, but as for evening cortisol concentrations, there was a significant difference: the concentration difference between morning and day salivary cortisol was significantly higher than that of working day saliva samples — meaning cortisol levels and resulting stress remain high rather than settling down throughout the day. Exposure to high levels of industrial noise (>80 dBA) strongly and significantly increased evening saliva cortisol levels, although noise levels lower than 80dBA had no significant impact on levels (9). This quasi-experiment provides objective evidence linking occupational noise exposure to increased stressed levels in industrial workers. Depending on the self-reporting of participants, this study is especially critical in understanding the relationship between cortisol levels and occupational stress, as it utilizes multiple methods and develops numerous conditions under which participants will experience stress. As a result, it highlights the importance of noise regulation and the implementation of workplace interventions to ensure the physical and psychological wellbeing of workers.

**Adrenaline Response to Lab Induced Stress**

In investigating adrenaline response in less formalized experimental settings, this study by Schmidt *et al*. investigates how acute nighttime aircraft noise affects cardiovascular function and stress hormone release in healthy adults, with the primary goal of testing for whether or not noise exposure during sleep would 1) impair endothelial (vascular) function and 2) trigger an increase in adrenaline release (10). The method of investigation was a blinded field study in 75 healthy volunteers (mean age 26 years) who were randomly exposed at home to two different noise scenarios (30 or 60 aircraft noise events per night) for one night each while polygraphies were performed during each study night (10). Other than refraining from consumption of coffee, tea, alcohol, sleep altering medications, and nicotine on the day prior to the study night, no further instructions were given, and participants continued their usual diets and daily routines.

Participants were scheduled to return to the laboratory for three visits, where during the night preceding each visit, subjects were exposed in a randomized order to one of three noise patterns: normal background noise (control night), Noise30 (30 aircraft noise events), and Noise60 (playback of 60 aircraft noise events) (10). At the study center, flow-mediated dilation (FMD) of the brachial artery was measured at the same time — in the early morning and before 10AM — using a linear ultrasound probe at rest and after a 5 minute occlusion period with a pressure cuff. Changes in diameter were given in percentages and reflect the endothelial release of vasodilatory substances such as nitric oxide; in other words, lower FMD values is an indicator of endothelial dysfunction (ED) (10). After FMD was measured, blood samples were drawn and frozen for later testing and the Dortmund Noise Sensitivity Questionnaire/Horne-Ostberg Morningness-Eveningness Questionnaire were filled out. These samples were evaluated for pulse transit time (PTT, time between the heartbeat and peak oxygen saturation at the fingertip). Adrenaline was measured in NH4-heparin anticoagulated tubes 30 minutes after puncture and after the blood cooled during transport (10).

Increased noise decreased sleep quality. Noise from Noise60 blunted endothelial responses, and exposure to more severe noise caused more severe ED. There was a marked increase in plasma adrenaline concentrations between control and Noise 60 and Noise60 exposure nights, up from 28.3 ± 10.9 ng/L during the control night to 34.1 ± 19.3 ng/L for Noise60 (10). This increase remained unique to adrenaline, as morning plasma levels of cortisol did not increase with noise exposure.

This study investigates aircraft noise, an environmental stressor, and its impact on healthy adult participants' adrenaline levels in a semi-formal experimental setting. Controlled experimental design using multiple audio recordings that represent different levels of noise demonstrate that noise intensity and frequency significantly impact the magnitude of hormone responses. Furthermore, in observing adrenaline levels immediately before and after sleep, this study uniquely isolates the direct impact of noise from other lifestyles or environmental factors to provide causal evidence of stress activation. One limitation of this study was that the majority of participants were healthy young adults (mostly female), which is not an accurate representation of the actual population.

Furthermore, Gesi *et al*'s paper conducted in 2002 measures the effects of single or repeated bouts of exposure to loud noise (a measurable stressful stimulus) on the catecholamine content and ultrastructure of the rat adrenal medulla (11). Eighty male Wistar rats weighing 200-250g were housed in the animal facility, red ad libitum, and kept under controlled environments (11). The sound stimulus was produced by two loudspeakers installed at a distance of 40 cm on two opposite sides of the cage and driven by a white-noise generator, with a precision sound level meter being used to adjust the intensity of sound to 100dBA uniformly in the cage (11). Rats were randomly assigned to eight groups (each of 10 rats), where groups A and B were exposed to noise for 6 hours; groups C and D were exposed 6 hours daily for 21 days (repeated exposure) (11). Immediately after noise, exposed and controlled animals were killed by decapitation before the adrenal glands from each animal were removed and dissected to isolate the medulla. Tissue samples were homogenized in ice-cold 0.1 M perchloric acid to stabilize catecholamines, where catecholamines and metabolites were then quantified (11). Adrenaline (A) cells were distinguished from Nonadrenaline (NA) cells based on their greater size and electron density of chromaffin granules.

Results found that noise exposure induces rapid and differential changes in adrenal medullary catecholamine dynamics (11). Levels of medullary A were increased after brief noise exposure, but were reduced after repeated noise exposure compared with controls — this suggests habituation or early secretory dysfunction in A-storing cells. After brief exposure, A- granules showed uniform distribution despite increased content, unlike the polarization of NA granules (11). Furthermore, repeated exposure induces structural changes in A cells, undergoing modifications consisting of the appearance of giant pale vesicles, suggesting a potential dysfunction of this component of the gland. In analyzing the adrenals glands of rats exposed to different levels and frequencies of noise, this study provides evidence that both acute and repeated exposures to high noise impair the gut indirectly via the stress response. This study is especially important, as its experiment design rooted in studying a rat species in a microcosmic environment keeps the experiment incredibly controlled. Direct observation of structural and function changes in the adrenal medulla provides a mechanistic explanation for the relationship between noise exposure, stress, and adrenaline.

## STRESS HORMONE IMPACT ON INTESTINAL PERMEABILITY

Stress hormones play critical roles in regulating system functions in many parts of the body, one being the gastrointestinal tract. Studies attributed such impacts, notably negative, to multiple pathways: decrease in tight junction proteins, lower levels of cytokines, and decreased microbial diversity in the gut. Considering that humans have many physiological and genetic similarities with rats, both studies on tight junction proteins and gut microbiota carried out experiments using rats. The study on cytokines gathered data from healthy human workers. Furthermore, all of these studies either contain noise-induced stress as independent variables or simulate similar conditions, allowing for data collection to most accurately reflect the conditions faced by general populations and communities affected by noise. Breaking down the most probable causes for increased intestinal permeability serves to establish precise frameworks for which researchers can evaluate consequent disease pathogenesis and potential therapeutic targets. A consolidated overview of biomarkers discussed across studies is provided in Supplementary Table 1.

***Supplementary Table 1.*** *Comprehensive overview of biomarkers discussed throughout the review, including their classification and method of identification and measurement.*

| Biomarker | Type | How It Was Measured |
|---|---|---|
| Cortisol | Hormone | Salivary sample |
| Adrenaline | Hormone | Blood testing |
| IL-1β | Cytokine | Gene expression evaluated in isolated PBMCs |
| IL-6 | Cytokine | Multiplex immunoassay using flow cytometer |
| TNFα | Cytokine | Multiplex immunoassay using flow cytometer |
| IL-10 | Cytokine | Multiplex immunoassay using flow cytometer |
| CLDN-1 | Tight junction protein | Observed in a 21-day Caco-2/BBe cell model |
| HES-1 | Transcriptional repressor | Observed in a 21-day Caco-2/BBe cell model |
| Bacteroides | Bacterial genus | DNA sequencing using cecal content samples |
| IFN-γ | Cytokine | Multiplex immunoassay using flow cytometer |
| IL-12p70 | Cytokine | Multiplex immunoassay using flow cytometer |
| MCP-1 (CCL2) | Cytokine | Multiplex immunoassay using flow cytometer |
| iNOS | Enzyme | Real-time PCR |
| Coprococcus | Bacterial genus | DNA sequencing using cecal content samples |
| Pseudobutyrivibrio | Bacterial genus | DNA sequencing using cecal content samples |
| Dorea | Bacterial genus | DNA sequencing using cecal content samples |

## Tight Junctions Proteins

By regulating paracellular permeability, intestinal epithelial tight junction proteins contribute significantly to intestinal barrier function. However, as Zheng *et al.* finds, elevated glucocorticoids (including cortisol) reduce the intestinal epithelial tight junction protein claudin-1 (CLDN1) by decreasing HES1 (Hairy and Enhancer of Split 1), a Notch signaling repressor that is high at the crypt base, and glucocorticoid receptor NR3C1 (low at the crypt base) (12). This high-low pattern reverses at the crypt apex. Research finds through observing CLDN-1 expression in rats that chronic stress (artificially and naturally induced) decreased colon lumen CLDN1 protein expression in the rat, which was prevented by the NR3C1 antagonist RU486. Rat epithelial cells harvested by scraping were used to observe that CLDN1 mRNA was downregulated by around 50%, meaning there was 50% less production of CLDN1 mRNA to begin with (12).

Furthermore, another experiment in the same study observing HES1 and NR3C1 levels in a 21-day Caco-2/BBe cell model showed that early in cell growth, HES1 was high and NR3C1 and CLDN1 were low, whereas later in cell growth, NR3C1 and CLDN1 levels increased and HES1 levels decreased (12). This experiment replicated how real intestinal cells mature while migrating from the bottom to the top of a crypt. Early in this maturation process, siRNA was used to block the HES1 protein. Consequently, HES1 levels dropped, and CLDN1 levels went up, signifying that HES1 typically suppresses CLDN1 activation, and its absence enables cortisol to increase CLDN1 levels (12).

In investigating how HES1 and NRC31 regulate the CLDN1 promoter in colon cells, this study demonstrates how stress affects gut barrier function at the molecular level. Chronic stress (simulated in this study by cortisol) is shown to change the balance of HES1 and NR3C1, which in turn alters CLDN1; this study therefore proves how molecular imbalances due to stress downregulate CLD1, therefore weakening tight junctions in the gut and allow the gastrointestinal barrier to become leaky (more susceptible to inflammation and gastrointestinal issues). This establishment of a mechanistic link between noise pollution and gastrointestinal health is a critical step to understanding what disproportionate health effects marginalized groups face.

The molecular regulatory interactions described above are summarized in Figure 2. This schematic illustrates the regulatory interaction between HES-1, NR3C1, and CLDN-1 in intestinal epithelial cells under stress conditions. *Left panel:* Under normal or stress-enhanced conditions, HES-1 represses the expression of CLDN-1, limiting tight junction formation, while cortisol-bound NR3C1 provides only partial activation. *Right panel:* Knockdown of HES-1 via siRNA removes this inhibitory signal, allowing NR3C1-mediated activation of CLDN-1 to proceed, resulting in increased CLDN-1 expression. This mechanism demonstrates how chronic stress–related changes in HES-1 and glucocorticoid signaling contribute to altered tight junction integrity and increased intestinal permeability.

## Cytokines

In evaluating workplace stress, Marcella *et al*, measured the levels of salivary cortisol and mononuclear cell production of cytokines, along with perceptions of workplace stress and plasma levels, were measured and examined in 80 healthy workers recruited among a population of operators on gas and oilfields (13).

Salivary cortisol concentration is evaluated as a marker for the activation of the HPA-axis and as a biomarker of stress. Standard cortisol shows a diurnal rhythm, with highest levels in the morning before undergoing steady decline with lowest levels at night; compared to "normal" levels of 2.2 – 4.1ng/ml in saliva on average, data indicated that 61.25% of workers recruited for this study had cortisol levels greater than 10ng/ml (13).

As for cytokine gene expression, this was evaluated in peripheral blood mononuclear cells (PBMCs) isolated from the enrolled workers. These PBMCs were stimulated with the mitogenic lectin Phytohaemagglutinin (PHA) to account for low amounts of cytokines produced by resting immune cells and differences related to complex cell-cell interactions (13). Statistics showed that between

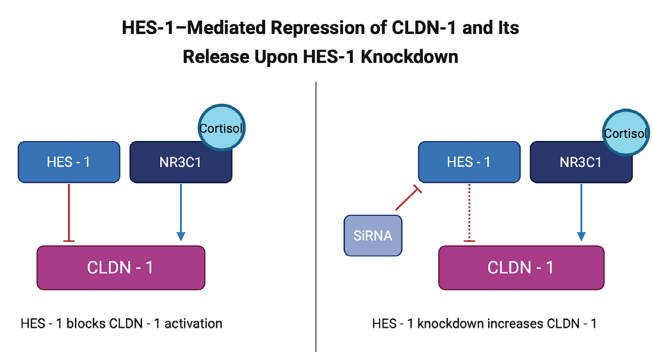


***Figure 2.*** *HES-1–mediated repression of CLDN-1 and its release upon HES-1 knockdown. Created in Biorender.*

the two groups of workers with cortisol levels greater than and less than 10ng/ml, cytokine levels in PHA-stimulated PBMCs from workers with cortisol >10ng/ml were significantly higher, specifically for IL-1β, IL-6, TNFα, and IL-10 (13). The expression of these cytokines was evaluated in the context of production by analyzing their levels in the supernatants of PBMCs inoculated with PHA. Results indicated that the levels of released cytokines were in accord with gene expression levels.

The lipopolysaccharide (LPS), a component of the outer wall of bacteria and a well-known inducer of inflammatory cytokines production, was used to assess if cortisol and subsequent cytokine levels influenced immune activation and inflammation. The results of the study show that LPS did, indeed, induce an increase of cytokine expression, leading to on-average lower levels of cytokines and therefore reduced immune response. This study represents a specific source of stress that proves to have counterintuitive results — the gas and oil industry, where workers have some of the highest national average salaries, is dominated by white men, and push people of color out of the space. Yet, it is this population that experiences high levels of occupational stress. Nonetheless, the results of this study provides evidence critical to understanding how occupational stress overall leads to increased gut permeability.

**Gut Microbiota**

Studies show that exposure to stressors impacts the stability of the microbiota, leading to bacterial translocation (14). To understand whether the microbiome contributes to stressor-induced immunoenhancement, Bailey *et al.*'s study exposed mice to the social stressor social disruption (SDR) to increase the circulation of cytokines and prime the immune system for enhanced reactivity.

Male CD-1 mice were purchased for this study. After acclimation to lab environments, the mice were housed in groups of 2-5 per cage and kept on a 12-hour light: dark schedule with lights on at 6:00PM (14). No limitations on food and water were set. The SDR stressor occurred over 16:30-18:30PM (the transition period from the end of the light cycle to the beginning of the dark) and was initiated by placing an aggressive male mouse into the home case of the resident mice (14). After ensuring that the aggressor attacked and defeated all resident mice within the first 20 minutes of agonistic interaction, the aggressors were left in cages for 2 hours, removed, and left the residents undisturbed before repeating the cycle the next day (14). After exposure to a total of 6, two-hour cycles of SDR, the subjects of the experiment were euthanized at identical points in time. Cecal contents were then harvested using the aseptic technique before being stored and shipped for analysis. Samples were centrifuged and resuspended in 500 µl RLT buffer before adding one sterile 5 mm steel bead and 500 µl of 0.1 mm glass beads for complete bacterial lyses (14). The sample underwent one more brief centrifugation before the supernatant was extracted and mixed 1:1 with 100% ethanol. The mixture then was added to a DNA spin column, where QIAamp DNA Mini Kit instructions guided the experiment to elute, dilute, and quantify bacterial DNA samples.

After sequencing and removing all failed reads, low-quality ends, and tag sequences, remaining sequences filtered out non-bacterial ribosomal reads and chimeras. Then, the cleaned sequences were queried using a distributed BLASTn algorithm against a curated database of high-quality 16S rRNA sequences. Through this method, each bacterial sequence was assigned a species, genus, family or order based on its match with known sequences, with the number of sequences for each taxon describing the abundance of each bacterial group.

Concurrently, blood was collected from the mice via cardiac puncture and was measured for serum cytokines using the mouse inflammation kit cytometric bead array per manufacturers instructions, which allowed for the simultaneous assessment of IL-6, IFN-γ, IL-12p70, TNF-α, MCP-1 (CCL2), IL-10 (14). Inducible nitric oxide synthase (iNOS) mRNA in the spleen was also assessed using real-time polymerase chain reaction (PCR) (14). iNOS is an enzyme that produces nitric oxide, a marker of immune activation in the spleen. Finally, the mice were given oral antibiotic cocktails of 1mg/ml ampicillin, 0.5 mg/ml vancomycin, 1mg/ml neomycin sulfate, and 1 mg/ml meronidazole. These antibiotics were administered twice daily by orgal gavage, starting three days prior to stress and continuing during stress.

Using all of the gathered data, results found that exposure to the SDR stressor did, indeed, significantly decrease microbial diversity and richness in the gut (genus *Bacteroides*, in particular) — however, this effect was delayed, suggesting that change occurs over time. Concurrent with these microbial changes, exposure to SDR also resulted in a significant increase in circulating cytokine levels, with the largest increase found in IL-6 and MCP-1 levels, which was significantly correlated with stressor-induced changes to the three bacterial genera *Coprococcus, Pseudobutyrivibrio,* and *Dorea*, and is known to prime the innate immune system by increasing iNOS gene expression (14). Both of these stress-induced immune effects are dependent on the presence of gut microbiota. Administration of the antibiotic cocktail, which reduced intestinal bacterial density by approximately 100-fold, blocked the increase in circulating IL-6 and attenuated iNOS expression. The results of this study support a casual role for gut bacteria in mediating stress-driven immune activation, specifically underscoring the gut microbiome as a critical mediator of the physiological consequences of chronic stress. This study implies that chronic environmental stress may contribute to heightened gastrointestinal inflammation and immune dysregulation due to changes in the gut microbiome, highlighting the role of cyclical oppression in the health of marginalized communities.

## CONCLUSION

These studies conclude that increased exposure to intense noise has been consistently associated with elevated stress hormone levels with higher stress levels, demonstrated and measured in levels of stress hormones cortisol and adrenaline. Increased levels of adrenaline were directly indicative of the body's response to stress; increased cortisol, along with indicating higher stress, impacted internal systems beyond that of the HPA axis. Specifically, higher cortisol levels are associated with decreased tight junction proteins, cytokines, and gut microbiota diversity, all of which lead to increased gut permeability and consequent inflammation and dysregulation.

These numerous pathways for gastrointestinal inflammation have negative consequences for human health: inflammation results in an increased cancer risk, as such impaired function complicates the removal of mutant and senescent cells and thereby enables tumor outgrowth. Gut dysbiosis, intestinal barrier disruption, and immune system imbalances stemming from chronic inflammation are just a few of the factors that contribute to the development of inflammatory bowel disease (IBD). Finally, chronic inflammation may lead to gastrointestinal perforation as it weakens the intestinal wall tissue and causes a rupture.

It is critical to recognize that the relationship between these many conditions — e.g. cortisol and tight junction proteins or inflammation and IBD — are often bidirectional, without defined linear progression or causality. Rather, the relationship is built on a complex interplay of correlation, significantly complicating the seemingly-straightforward relationship between stress, inflammation, and consequent health concerns.

Considering that socioeconomic status plays an immense role in the communities one is involved in, it can be reasonably concluded that there is a strong correlation between increased risk of gastrointestinal inflammation and race and economic class. There are a number of factors that explain this correlation, the most prevalent being the proximity to high-volume areas such as industrial sites and highways — areas which are often priced at lower values due to the noise disrupting quality of life.

A major limitation in this paper is the lack of consistency in the size of the observed population — for instance, a limited number of studies focus specifically on the city of New York and rather address the United States as a whole, which fails to account for regional factors and differences that may shape data. Overall, there are many inconsistencies in the observed population, meaning the connections drawn across the numerous studies may not accurately reflect the true relationship between stress and human health in a given region. Furthermore, future studies may benefit from incorporating longitudinal designs, given that the major impact gastrointestinal inflammation has on human health can only be observed upon identifying chronic inflammation.

## CONFLICT OF INTEREST

The authors declare that there are no conflicts of interest related to this work.

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